Claude Franceschi

VENOUS INSUFFICIENCY OF THE PELVIS AND LOWER EXTREMITIES

Hemodynamic rationale of pathophysiology, diagnosis and treatment

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Hemodynamic rationale for pathophysiology, diagnosis and treatment

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‘I tremble lest I have mankind for my enemies, so much has wont

and custom become second nature. Doctrine once sown strikes

deep its root, and respect for antiquity influences all men’.

De Motu Cordis (1628): William Harvey.

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Research and studies on hemodynamic venous insufficiency and its therapeutic consequences, such as CHIVA, would not have been possible without the collaboration of vascular surgeons and angiologists from all over the world who have verified, validated, disseminated, and generously taught their knowledge. They also had the professional courage to promote an intellectually demanding method against the simplism of destructive methods supported by powerful sponsors without whom all the congresses could not take place. They would not have been excluded from most congresses if their knowledge did not jeopardize the profitability of ethically and legally questionable destructive procedures.

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FOREWORD 1

When Claude asked me to write the foreword for his new book, VENOUS INSUFFICIENCY OF THE PELVIS AND LOWER EXTREMITIES, I felt honoured but also humbled, since I am not a bona fide hemodynamics expert with enough knowledge to interpret CHIVA (Cure Conservatrice et Hémodynamique de l'Insuffisance Veineuse en Ambulatoire) principles properly to compare them with traditional principles. At the beginning, I hesitated because I am a plain clinician who's dedicated an entire career to (kidney and liver) transplant and vascular surgery, mostly to arterial surgery, with fairly limited knowledge in venous surgery/hemodynamics—barely enough to handle venous malformation as my recent specialty.

But I also felt like I could give an unbiased and fair commentary on Claude's lifetime work on CHIVA. That is because I am known to be one of the only non-practitioners of CHIVA among the vascular surgeons on both sides of the Atlantic but have led its thorough review through the IUP Consensus on venous hemodynamics of the lower extremity.

Indeed, when Claude introduced CHIVA in 1988, such a fresh interpretation of venous hemodynamics was regarded as a heretical view challenging the traditional venous hemodynamic concept. It has received an unfairly hostile reaction from many colleagues all over the world based on their prejudice, despite evidence validating its rationale in 120 publications, including observational, randomized studies and Cochrane Reviews.

Hence, when the IUP executive committee offered us the opportunity to organize this long-overdue consensus on venous hemodynamics of the lower extremity, we were determined to include the controversial CHIVA concept.

Despite it taking four years for us to complete the consensus, clarifying every item of controversy among the CHIVA principles, we were finally able to prove its long-neglected value. We gave credit to CHIVA as one of the fully established concepts of hemodynamic rationale for pathophysiology, diagnosis, and treatment doctrine, like William Harvey's *De Motu Cordis,* which Claude has quoted in his book.

So, I greatly enjoyed every chapter of VENOUS INSUFFICIENCY OF THE PELVIS AND LOWER EXTREMITIES, especially in how it clarifies fluid mechanics for vascular specialists so they can achieve a proper understanding of hemodynamics—fundamental to vascular functions—and improve their clinical care. The historical review piece, in particular, is phenomenal as the highlight of this manuscript. Various laws and equations of fluid mechanics were also well articulated in easy-to-understand language, together with their venous hemodynamic effects, so I appreciate the ability of this book to explain venous hemodynamics as a whole.

I also share Claude's appeal to save innocent saphenous veins from unnecessary sacrifice during contemporary varicose vein care through the proper application of CHIVA principles such as the saphenous vein salvage campaign.

Most physicians vaguely recognize the value of the saphenous vein as one of the graft materials used for coronary bypass, but not many know this autologous vein has been the free (vascular) graft of choice and still remains essential for all bypass surgeries. In the old days, when open surgery was still the first choice for handling vascular cases in particular, all surgical trainees who encountered invasive trauma were taught to include both groin-down upper legs/thighs for scrubbing, just in case the saphenous veins could be used as vascular graft material to repair damaged vessels.

Naturally, we, both general or vascular surgeons, all keenly appreciated the saphenous vein's unique value, as well as its role as the gold standard; no other man-made materials could compete for vascular repair for decades. But this unshakable fact was slowly eroded through the last three decades and abandoned by methodical brainwashing, often facilitated by the industry, that claimed "man-made material/graft is as good as, if not better than, natural vein material," which is utterly wrong.

Besides, since the era of endovascular surgery began three decades ago, taking over the traditional leading role of open surgery, many lost interests in this saphenous vein, no longer considering it as the benchmark of vascular graft. Also, unfortunately, during the same era, a new health agenda emerged. The increased knowledge and interest in venous hemodynamics identified "reflux by venous valvular insufficiency" as the culprit of varicose veins, which hampered the quality of life.
This newly established hemodynamic concept accusing the reflux, along with the simultaneous introduction of the endovascular ablation device by the industry, facilitated an overly casual approach to varicose veins, with no second thought for its priceless value as a vascular graft. Even symptomless varicose veins were removed by some clinicians with no hesitation.

Should you look back through history, such a supposedly heretical hemodynamic interpretation is quite a timely blessing, warning against the current abuse of valuable saphenous veins under the rationale of supposedly improving quality of life hampered by varicose veins.

And I can't help but agree with the lament of William Harvey (1578–1657) in *De Motu Cordis* (1628), which Claude kindly shared in his book: "Doctrine once sown strikes deep its root, and respect for antiquity influences all men."

All the best,

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FOREWORD 2

This book was conceived as a comprehensive overview on the clinical management of chronic venous disease of the lower limbs and the pelvis, designed to fill what Claude Franceschi believed to be a void in the current medical consideration about this very widespread disease. Perhaps it is true that the prefaces are not read, however I wrote with honour and pleasure because the world expert and international authority, Claude Franceschi, compiled a wide-ranging yet easy to read textbook with ten chapters and plenty of line drawings and colour illustrations to guide us through a not easy new concepts and a different procedure in this field compared to the past. In few words, this is a recommendable *educational path*.

Strange story that of the conceptual innovation brought to venous pathology: brilliantly published by Claude Franceschi with a completely captivating originality in 1988, it found an immediate positive response through many media, particularly in Italy, and equally among patients who saw a simplification in the treatment of their "varicose veins". However, the time was not ready to undertake a change in the clinical and instrumental paradigm so deeply rooted in a hemodynamic field difficult to study as that of the venous circulation of the lower extremities. All the more so for a pathology – the varicose veins, in reality only a part of a much more complex chronic venous disease – considered between clinically benign or simply aesthetic for many others. It should not be forgotten that most surgical interventions are still performed today mainly by general surgeons anchored to the lesson of the past, namely stripping in its different propositions; or by doctors of various specialties such as dermatologists, angioradiologists, aesthetic doctors, and not always by vascular surgeons. This book, or better *educational path*, as I personally consider it, does not want to be directed only to those who already master these hemodynamic concepts, but above all to those who have training, personal experience and valid results with their own "traditional" vision of venous circulation based on concepts that imposed themselves starting from the years 1904-1907 for over a century. But «*learning is like rowing upstream: the moment you stop, you go backwards*», so goes an old Chinese saying. It is probably in the lack of study of physics that doctors in general and even the vascular doctor still today can have gray areas of interpretation of the physiological and pathological phenomena underlying the whole tree of circulation, starting from the misunderstanding of a venous circulation much more complex than the arterial and lymphatic one. Welcome to Claude Franceschi's lecture on the importance of fluid mechanics studies the behavior of fluids and the internal forces associated with them.

FOREWORD 3

Primary varicose veins of the lower extremities are the most frequent disease of venous and vascular pathology. Their prevalence is around 20% of the population. Primary varicose veins are usually a benign disease, although complications may occur. It is curious, however, that this process has been predominantly considered from a morphological, if not merely aesthetic, point of view.

Based on this morphological conception, priority has been given to treatments based on a destructive strategy. Various techniques have been used: surgery (stripping, phlebectomies), physical techniques based on heat (laser, radiofrequency), based on cold (cryosclerosis), chemical techniques (sclerosis with or without foams), etc. In other words, almost everything that can be used for the direct elimination of varicose veins has been used. Industry has contributed to this situation by creating more and more glamorous and sophisticated devices that have been advertised to doctors and patients. It has mattered little to them that the massive destruction of the superficial venous system, including the saphenous veins, has deprived patients of important elements for the venous drainage, or of precious autogenous material for eventual vital coronary and vascular surgery.

In other words, the pathophysiological and haemodynamic aspects of varicose veins have hardly been considered in the therapeutic basis of this pathology. And this might not have been the case:

At the end of the 19th century Trendelenburg with his manoeuvre suggested the existence of a private recirculation between the deep and superficial venous system. He hypothesized by more than a century what we know today as veno-venous shunts thanks to Duplex US hemodynamic data.

In the first half of the 20th century Perthes demonstrated, with his manoeuvre, the retrograde emptying of varicose veins by the suction into the deep venous system by the valvo-muscular pump. This is the principle used by the CHIVA method in most cases.

The Trendelenburg and Perthes manoeuvres have been classically used in the clinical examination of varicose syndrome. However, they have had little or no impact on the strategy for treating varicose syndrome. In other words, the basic strategy has been the destruction of the superficial venous system. This attitude can be justified by the lack of non-invasive morphological and haemodynamic information. Phlebography as the only topographical examination was invasive and provided images often difficult to interpret due to too little or absent haemodynamic information.

In 1955 Satomura and Kaneko first used the continuous wave Doppler in vascular examination. Despite the lack of spatial resolution of this technique, it represented a breakthrough in non-invasive arterial and venous exploration.

In the early 1960s Strandness developed with members of the Department of Bioengineering at the University of Washington Rushmer, Franklin and Baker the first prototype devices using CW Doppler in vascular scanning. In 1967 Strandness using CW Doppler published the first paper on the differences between normal and pathological flow velocity profiles in peripheral vessels.

In 1977, Franceschi publishes "Investigation vasculaire par ultrasonographie doppler "the world's first book on the usefulness of CW Doppler in arterial and venous examination.

In 1975, the first prototypes were introduced simultaneously in the United States (Strandness) and in France (Pourcelot) showing the association of B-mode ultrasound with pulsed Doppler. This procedure, known as duplex in the USA and echo-Doppler in Europe, gave a colossal boost to non-invasive vascular diagnosis.

In 1981 Franceschi introduced for the first time a device in the form of a strand-off bag of water which, when incorporated into the echo-Doppler probe, provided adequate imaging of the superficial vessels. This allowed for the first time, the US imaging of the supra-aortic trunks as well as the arteries and veins of the lower extremities. Thanks to this device, Franceschi published the world's first atlas of arterial and venous ultrasound in 1986.

The constant technical improvement (multifrequency transducers, Doppler spectral analysis, colour Doppler, power Doppler, CVI, echo-flow, etc.) has led to a significant improvement in the quality of these devices. Echo Doppler began to be used basically in the diagnosis of arterial pathology, particularly in the exploration of the supra-aortic trunks, where it contributed great advances in non-invasive diagnosis of carotid stenosis. Its use was also extended to arterial mapping of the lower extremities, making it possible to avoid numerous diagnostic arteriographies.

In relation to venous pathology, echo-Doppler began to be used for the diagnosis of deep vein thrombosis, especially when exploring venous compressibility.

In 1988 Franceschi, after observing the "in vivo" behaviour of the deep and superficial venous circulation of the lower extremities using echo-Doppler, described a new procedure for the treatment of varicose veins of the lower extremities which he called the CHIVA Cure (acronym for cure Conservatrice et Hémodynamique de l'Insufffisance veineuse en Ambulatoire). This treatment could be summarised as the application of the Tendelenburg and Perthes manoeuvres in a permanent and selective way through minimally invasive surgery performed under local anaesthesia.

Since its introduction, the CHIVA Cure has been the subject of great controversies, as it was introduced by a non-surgeon and above all for presenting a "non-destructive" strategy for varicose veins based on the haemodynamic control of this syndrome.

The European CHIVA Association, which I have had the honour of chairing since 1994, was created in 1988. Since its creation, the Association has held biennial meetings in various countries in Europe and America. This has undoubtedly contributed to the improvement and dissemination of the haemodynamic strategy in the treatment of varicose syndrome.

The procedure has been enriched by the enthusiastic collaboration of people such as Bailly, Dadon, Cappelli, Ermini, Delfrate, Zamboni, Mendoza, Escribano, Parés, etc, directed and encouraged by Franceschi himself. For me it has been a privilege to learn and work with them. The CHIVA cure has been complemented and optimised.

With the critical evaluation of the accumulated experience, we can say that today the CHIVA Cure is a highly effective and minimally invasive method in the treatment of varicose veins of the lower limbs. Its results have been demonstrated in publications in scientific journals of recognised prestige.

What no one have doubted, even the worst enemies of this procedure, is that thanks to the research that led to the introduction of the CHIVA strategy, we have learned relevant aspects regarding normal and pathological venous haemodynamics that we did not know before. Doppler ultrasound has proved to be the fundamental tool in the study of the pathophysiology of venous insufficiency of the lower extremities. This has laid to the foundations for a rational treatment that addresses the previously ignored haemodynamic aspects of this syndrome.

But a further step was needed, and this is what Franceschi tackles in this extensive book: to unite physics with haemodynamics. In other words, a scientific approach linking the complex physics of fluid dynamics with the relevant aspects of venous physiopathology.

This book aims to fill the gaps that exist between the clinic as we perceive it and the physical phenomena that determine it. And it does so in language accessible to the physician, for whom the study of the basic sciences is a long way off. This is a book that should be read slowly. Only in this way the concepts can be properly understood and related to the pathophysiology and clinic of varicose disease.

Thanks are due to Dr Claude Franceschi for the enormous amount of work he has put into this book. It is essential to explain the complex world of venous haemodynamics in an intelligible way. Only in this way we will be able to understand the basis for the rational treatment of venous insufficiency syndrome.

Jordi JUAN SAMSÓ

President of European CHIVA Association

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7373235--Systolic Paranà and reflux N1>N2 at the saphenopopliteal junction SPJ.

73733236--Tibio-safenous junction

737323237-Pulsed saphenous outflow

73732371-Pulsatile retrograde flow due to tricuspid heart valve reflux.

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73732373-Anterograde pulsatile flow due to resistance to flow:

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7373242-The hemodynamic function of the lesser saphenous vein is particular.

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7379-Ecodoppler by pathology

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737911-Nutcraker's syndrome NTS or aorto-mesenteric clamp:

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737915--Common femoral vein occlusion

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737917--Popliteal vein occlusion

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Chapter 9

CHIVA Literature

By Massimo Cappelli and Paolo Zamboni

91- ARTICLES FOCUSED ON THE PRESENTATION OF CHIVA THERAPY.

Chapter 10

Sclerotherapy and CHIVA

Chapter author: Massimo Cappelli Florence Italy

1073- 3) TREATMENT OF COLLATERALS CONNECTED TO THE SAPHENOUS TRUNK

108- VASCULAR FILLING OF THE FOAM AND INJECTION TECHNIQUE

109- MATTING

1091-EARLY MATTING

1092-LATE MATTING

INTRODUCTION

Why this book?

The hemodynamic approach to the venous physiopathology led me to revisit the classical concepts. It has led to a new model, proposing new concepts which have led to a more refined semiology and a therapeutic strategy called CHIVA which is diametrically opposed.

The CHIVA cure would have no practical value if it had not significantly improved the treatment of venous insufficiency and allowed the preservation of the saphenous trunk. As a matter of fact, this vein is still destroyed by the classical approach although it represents a vital potential arterial bypass.

Note that, most often, ***the patients whose saphenous vein was destroyed for a benign disease, were not informed of this loss of chance. This raises questions about human rights, ethics and legality.***

The conceptual and therapeutic evidence of the ***CHIVA cure, a French acronym for Cure Conservatrice et Hémodynamique de l'Insuffisance Veineuse en Ambulatoire (Conservative and Hemodynamic Venous Insufficiency in Out Patients) was published in 1988.*** ***Ref***: Théorie et pratique de la cure conservatrice et hémodynamique de l'insuffisance veineuse en ambulatoire [CHIVA] Editions de l' Armancon 1988 ISBN-10: 2906594067 ISBN-13: 978-2906594067. ***It has been reported and validated by 120 publications including observational, randomized studies and 2 Cochrane reviews****.(Chapter 9)*

The purpose of this present book is to explain and, above all, to help those who want to improve their practice, both for their patients and for their professional and intellectual satisfaction, to understand venous hemodynamics.

The lack of consideration given to hemodynamics in the classic management of venous insufficiency, particularly varicose veins, can be explained by the daunting aspect of fluid mechanics*. Indeed, theoretical hemodynamics is feared by the non-physicist, who is not accustomed to manipule laws and equations. All the more so as they are often counterintuitive, especially when they are isolated from their practical context. This is why I have tried to make the physical bases of fluid mechanics understandable both in the theoretical context of pathophysiology and in their applications to diagnosis and treatmentI redefine old words in order to avoid semantic confusions (notably the term reflux which is polysemous) and I use new words to designate new concepts (notably shunts, re-entry point).*

Fluid mechanics studies the behavior of fluids and the internal forces associated with them*.*

Static studies fluids at rest. Dynamics studies fluids in motion.

Venous hemodynamics is the mechanics of fluids applied to the venous system*. Its study is essential, because it should be to the vascular specialist what optics is to the ophthalmologist and hydraulics to the dam builder. It is more complex than arterial hemodynamics because its pathophysiology depends on a subtler interaction of the physical variables. As I have already pointed out, it often repels physicians by its often-counterintuitive aspects. However, the understanding of physiopathological concepts, such as veno-venous shunts, radically changes the approach to diagnosis and treatment. I will try to explain them as clearly as possible. They will be better understood if the reader has more curiosity and an open mind. Indeed, the most frequent obstacle to understanding is not the lack of intelligence, but the conviction that one could not understand.* ***Thus, I sometimes tell listeners who say they feel smarter at the end of my courses that they are not smarter...but that they understand better when their intelligence is called upon****.*

The laws and related equations of fluid mechanics and their venous hemodynamic effects are explained in simple language. They are explained in the context of their application to diagnosis and treatment

Unlike most books, this one takes the risk of being redundant. Indeed, I recall and repeat these laws in each paragraph or chapter, throughout this book in their pathophysiological, diagnostic and clinical context. The purpose of these repetitions is to accustom the reader to the reasoning and pathophysiological explanations without necessarily having to refer to the preceding or following chapters. This should allow to read of each chapter almost independently of from the rest of the book.

*The effort required by the reader will be rewarded by the pleasure of better understanding leading to better diagnose and treatment the various aspects of venous insufficiency****.***

A brief historical review does not only pay tribute to the precursors. It helps to better understand the current problem. *Venous physiopathology has progressed step by step with the discoveries of anatomy, biology, physiology and fluid mechanics****.***

Fluid mechanics progressed mainly with Archimedes (212 BC), Simon Stevin (1548-1620), Blaise Pascal (1623-1662), Evangelista Torricelli (1608-1647), Isaac Newton (1643-1727), Daniel Bernoulli (1700-1782), Jean-Léonard Marie Poiseuille (1797- 1869), Louis-Marie-Henri Navier (1785-1836), George Gabriel Stokes (1819-1903) and many others.

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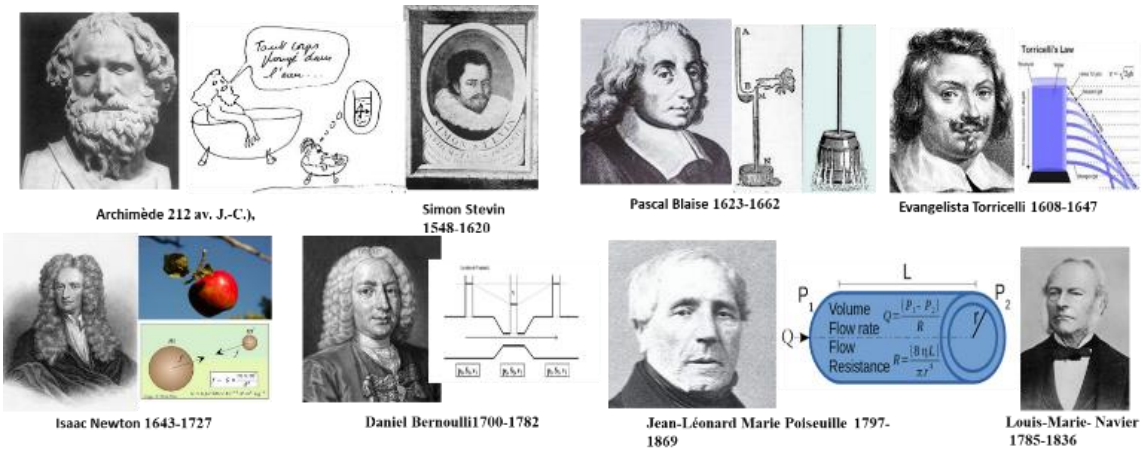


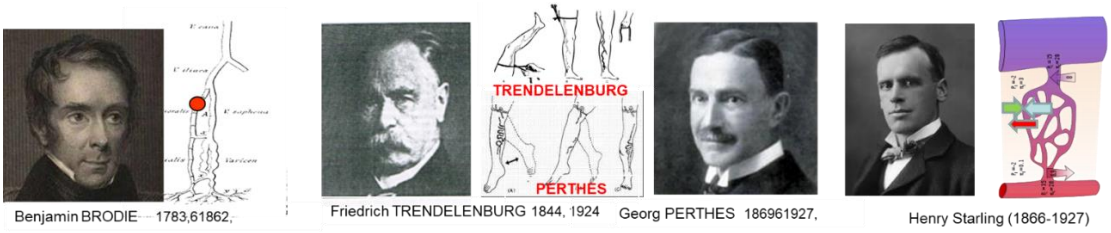
Anatomy and physiology of blood vessels

Physicians gradually established links between the anatomy and physiology of vessels. Ibn Al-Nafis Damishqui (1210-1288) and Giovanni Battista Canano (1515-1579) described venous valves. William Harvey (1578-1657), a student of Hieronymus Fabricius, published in 1628 "Exercitatio Anatomica de Motu Cordis et Sanguinis in Animalibus" in which he demonstrated venous circulation by compressing the superficial veins of the arm upstream and then downstream. He met with fierce opponents such as Primerose, Reid and Plemp. Guy Patin called him a "circulator" (a charlatan in Latin) and Jean Riolan condemned his discovery as "paradoxical, useless for medicine, false, impossible, unintelligible, absurd and harmful to human life". Marcello Malpighi discovered capillaries in 1661, i.e. the communications between arteries and veins. In 1670 Richard Lower described the vis a tergo (cardiopetal flow from the capillaries to the heart) and the venarum tonus (venous tone). In 1710 Antonio Valsalva described the vis a fronte (cardiac aspiration). In 1803 Justin von Loder discovered the perforators. In 1817 Chevalier de Richer explains the muscular pump. Ernest Henry Starling (1866-1927) described the principle that bears his name, according to which the net flow (direction and quantity of flow) in each section of the capillary wall is due to the equilibrium between the hydrostatic pressure difference and the oncotic pressure difference. This principle was discussed by Levick in 2010 without changing the practical value of Starling's model.

Hemodynamics and venous insufficiency.

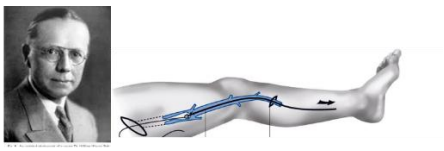
Other physicians have established the links between fluid mechanics and venous pathology. Sir Benjamin Brodie (1783-1862) wrote in 1846 "Lecture VIII: Varicose veins and leg ulcers" in his book "Lectures illustrative of various subjects in pathology and surgery". He attributes the

cause of the ulcers to the excessive weight of the blood column due to the incompetence of the valves and proposes to treat them by ligation of the great saphenous vein (GVS) or by a tight bandage of natural rubber when the ligation operation was too risky. Later, Friederich Trendelenburg (1844-1924) performed the great saphenous vein ligation and described his hemodynamic test. He compressed the varicose great saphenous vein at the root of the thigh with his finger in the patient who was lying down. He maintained the compression while the patient got up and remained standing. The great saphenous vein and its varicose tributaries remained empty longer than when no compression was applied. When he withdrew his finger, they expanded instantly because of the weight of the blood column transmitted, he said, by the valvular incompetence. He also hypothesized a "private circulation" which he described as follows: "During walking, blood from the varicose veins is drawn through communications when the deep blood is violently pumped upward. Then, presumably, some of this deep blood flows back down from the iliac and femoral veins, filling the varicose veins again." His assistant, Georg Clemens Perthes (1869 -1927), described the "Perthes test" which confirmed this intuition. He showed that when the patient walks with a tight tourniquet around the thigh, the varicose vein empties or not depending on the permeability of the deep veins. In spite of the accuracy of the hemodynamic diagnosis and the consequent healing of venous ulcers, the ligation of the GSV performed by Trendelenburg was not generalized. Indeed, as Benjamin Brodie had already pointed out, at that time it presented too many risks of infection and sometimes fatal haemorrhage in less expert hands than those of Trendelenburg.



Advances in antisepsis and anesthesia would have made surgical ligation safer. The ligation was replaced by radical removal of the GSV (stripping by Keller in1905, Mayo in 1906 and Babcock in 1907). *Thus, advances in anesthesia and antisepsis have set back those in hemodynamics. This* ***radical stripping*** *reduced venous insufficiency to a* ***simplistic and erroneous hemodynamic concept****. Indeed, varicose veins were no longer considered as the effect, but as the cause of excess venous pressure. Consequently, recurrence was attributed to incomplete venous eradication. Thus, "the more varicose veins are removed, the better the results and the less recurrence" was and still is for many the dogma. These* ***concepts based on stripping led to alternative ablative procedures that consisted of intravenous injections*** *of various occlusive products. Jean Sicard (1920), Karl Linser, Raymond Tournay (1893-1984)*

used less dangerous products than those previously injected by Valette, Petrequin, Desgranges (1853) and Weinlechner (1884).



Willian Babcock 1907

For more than a century, the ineffectiveness and recurrence of varicose vein treatments have been attributed to the non-radicality of venous destruction. *The apogee of this concept was the recommendation by some to destroy as many veins as possible, both varicose and normal (Poilleux in particular).*

The technological improvement of the destruction is not a scientific progress but the perseverance for a century of erroneous physiopathological concepts. It reflects a lack of knowledge of the hemodynamic bases of venous functions, particularly those of drainage.

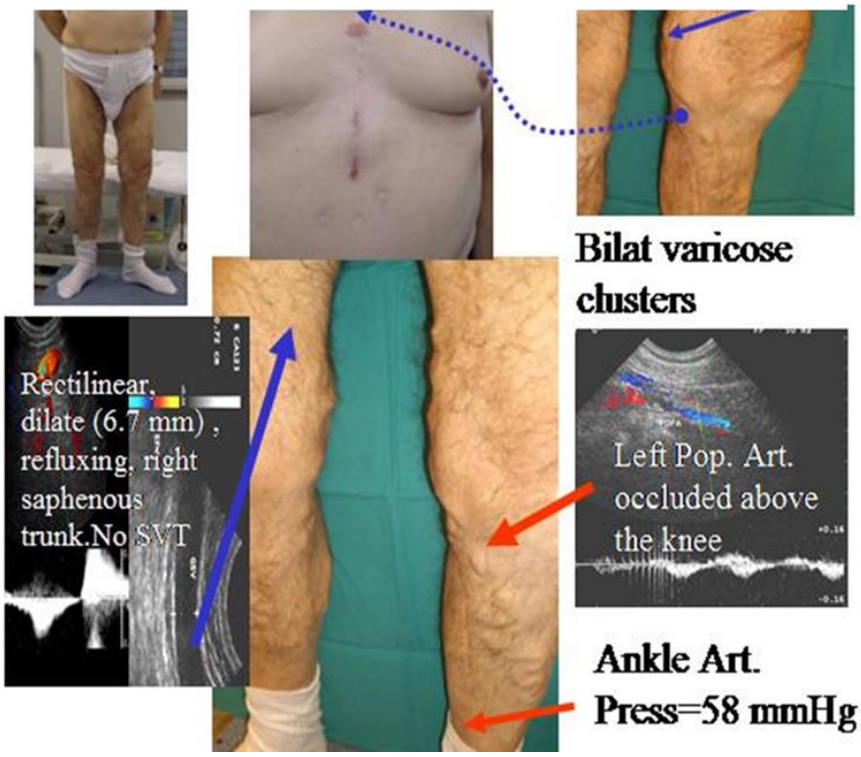
Theoretical and practical venous hemodynamics.

In 1988, motivated by patients who could not have vital arterial venous bypass surgery due to the lack of saphenous veins previously "treated" for varicose veins, I sought and proposed a conservative therapeutic approach. So, thanks to a better understanding of hemodynamics provided by the echodoppler, CHIVA was born, as a conservative and hemodynamics treatment of venous insufficiency in the outpatient setting. Since then, although "counter-intuitive", the conservation of varicose veins leads to fewer complications and recurrences than destruction.

The Saphenous vein can save life, even in varicose people. Why destroy it without warning the patient? Especially since it can be treated effectively without destroying the saphenous vein by hemodynamic methods

Male 78 y

To-day:
-left leg limp
-Bilat varicose clusters
10 years ago
-5 coronary by-passes (3 left GSV) +
-Right GSV crossectomy for SVT

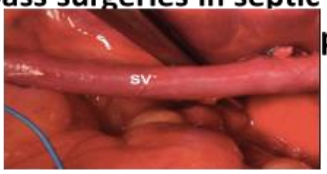


Bilat varicose clusters

Ankle Art. Press=58 mmHg

Bioprotec (Lyon France)
collects, freezes and sells stripped great saphenous veins as allografts

The great saphenous vein, continuous or incompetent
-Aorto-coronary bypasses
- Peripheral bypasses
Patch, vascular access
Bypass surgeries in septic environments, especially

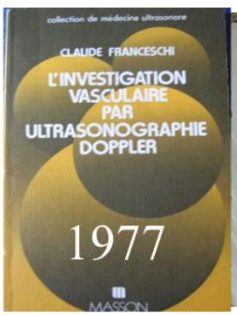


pro

- **No touch" harvesting**
- **Less spasm**
- **No dilation (less endothelial trauma)**
- **Conservation of vasa vasorum,**
- **less parietal ischemia**
- **Conservation of NO synthesis (less intimal hyperplasia**

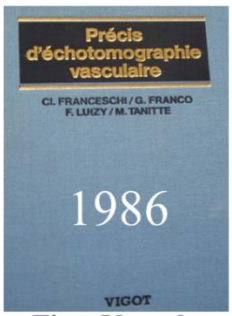
The path of my studies and research could help the reader to understand them better. They progressed with my intensive practice of the echodoppler and in the light of my references to fluid mechanics, which I applied to the arterial and venous systems. In 1977, I published the Doppler semiology that I had derived from it in the first book in the world published on the subject: Ref:"Claude Franceschi L'investigation vasculaire par ultrasonogrpahie Doppler" Masson Editeur France*, in French and then in Italian and Spanish. It concerned arterial and venous flows, normal and pathological (in particular carotid and peripheral arterial stenoses). In 1986, I published the first book in the world on ultrasound imaging of the vessels of the neck and extremities Ref:* "Précis d'échotomographie vasculaire" Claude Franceschi et al. Vigot, 1986 ISBN IISBN:2-7114-0989-9 (rel.): EAN: 9782711409891*, in French and Italian. This was the first book thanks to a water bag device that I had designed and patented previously. Réf:* 1981 Un dispositif permettant la visualisation des vaisseaux et organes superficiels: Brevets d'invention :Dispositif pour la transmission d'ultrasons pour une sonde d'échotomographie INPI N° d'Enregistrement National : 81 22294.

Indeed, this standoff adapted to the ultrasound devices of the American company ATL allowed for the first time to see in a non-invasive and indefinitely reproducible way, the supra-aortic trunks, the peripheral arteries, and veins. ***Doppler combined with ultrasonic imaging has thus revolutionized the structural and especially the hemodynamic diagnosis of symptomatic and asymptomatic arterial and carotid stenoses, aneurysms and thrombophlebitis.***

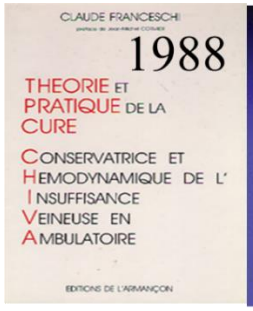


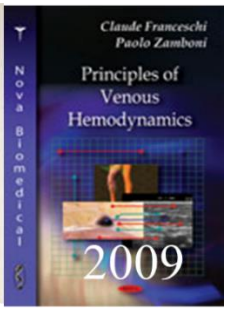
First Doppler semiology

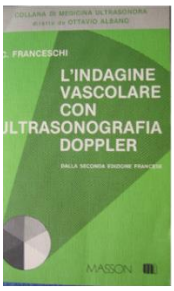
First carotide Echography

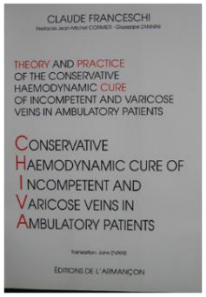


First Vascular Echo Imaging







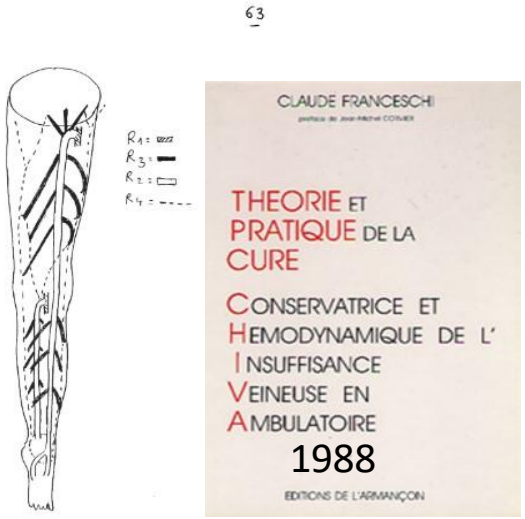


This experience in ultrasound physics and fluid mechanics applied to vascular pathology opened the doors to complex hemodynamics. Indeed, the hemodynamics of venous pathology is more complex than that of arteries because it depends on more variables. This is probably the reason why the majority of vasular specialists, still impregnated with classical approaches, have difficulty assimilating these theoretical and practical advances. Moreover, they do not practice sufficiently the echodoppler themselves. Indeed, this technique is performed in many countries by ultrasonographers subject to a standard protocol too poor to provide the necessary information. Therefore, this book aims to fill the gaps in the classical teaching of theoretical and practical venous hemodynamics.

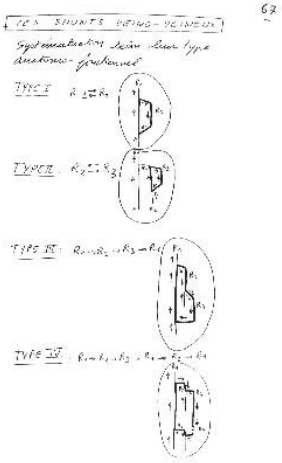
*The history of anatomical, functional, and hemodynamic concepts can help the reader to better understand them. The anatomy of the venous system, particularly the superficial system, is highly variable and, contrary to the still frequent opinion, does not prejudge its pathology****. Pathology is a disorder of function, regardless of anatomy! Anatomists have been inspired by without referring to the classification of veins into anatomical, functional, topographical and hemodynamic networks that I proposed in 1988,*** ***Ref:*** Caggiati A. Novelties in saphenous anatomy. Relationships of the saphenous veins with the fasciae: the saphenous compartment. Phlebology, 2003, 56, 1, 19-25***.*** *They confirmed on the cadaver what was evident in ultrasound imaging and* ***translated R1, R2, R3 networks (R for Réseaux in French) into English N1, N2, N3 networks****.* ***Others have shown on the cadaver constitutional venous obstacles to the Hunter that I had already described hemodynamically*** *(Open Vicarious and mixed shunts with systolic escape point of the saphenopopliteal junction)* ***Ref***: Principles of

Venous Hemodynamics C. Franceschi, Zamboni Nova Science Publishers 2009-01 ISBN Nr 1606924850 / 9781606924853.

The hemodynamic classification into N1, N2, N3, N4 networks and the Open vicarious shunts OVS, Open deviated Shunts ODS and Closed Shunts CS, defines the veins by their physiopathological function whatever their anatomy.



Réseaux



Shunts

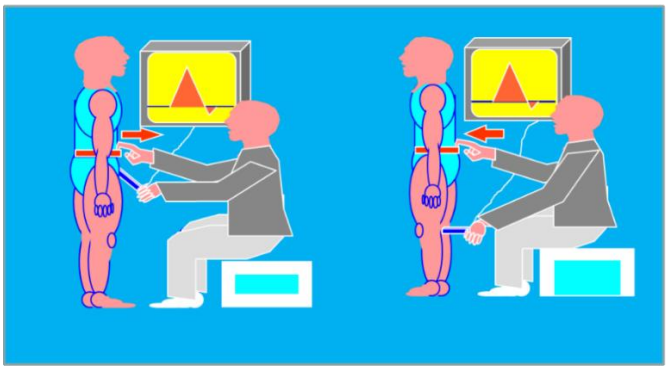
We can say that the veins are not always where we look for them, but they are always where we find them thanks to the echodoppler, which also allows us to focus our attention on hemodynamic abnormalities and the search for their causes (escape points, pathways and re-entries). *This is how I found the pelvic escape points* ***Ref:1-*** Franceschi, C, Bahnini A. Points de Fuite Pelviens Viscéraux et Varices des Membres Inférieurs. Phlébologie 2004, 57 n.1, 37-42. 2-C.Franceschi, C, Bahnini A. Treatment of lower extremity venous insufficiency due to pelvic escape points in women Ann Vasc Surg 2005; 19:284-8

Indeed, by scanning the descending flow (normal direction) but Valsalva + (pathological) of the descending tributaries of the saphenofemoral junction that I found and defined anatomically and functionally these escape points. By observing reflux from the saphenopopliteal junction during systole of the calf pump, often associated with a reduction in the calibre of the superficial femoral vein, I described the hemodynamic obstacle of the superficial femoral vein, without seeing it.

The venous system must be assessed not only in supine, but also and necessarily in standing*. This was demonstrated by Trendelenburg and Perthes more than a century ago.*

The echodoppler allowed me to follow the normal and abnormal flows in standing patients according to the activity of the cardiac, thoraco-abdominal and valvulo-muscular pumps and the related dynamic tests as Valsalva and Paranà. I replaced the calf compression-relaxation with the Paranà manoeuvre, which is more physiological because it provokes a reflex proprioceptive isometric contraction. Paranà is the name of the Argentine city on the banks

of the Paranà River where I first taught the manoeuvre. ***Ref:*** Franceschi C. Mesures et interprétation des flux veineux lors des manœuvres de stimulation. Compressions manuelles et manœuvre de Paranà`. Indice dynamique de reflux (IDR) et indice de Psatakis. J Mal Vasc 1997;22:91–5



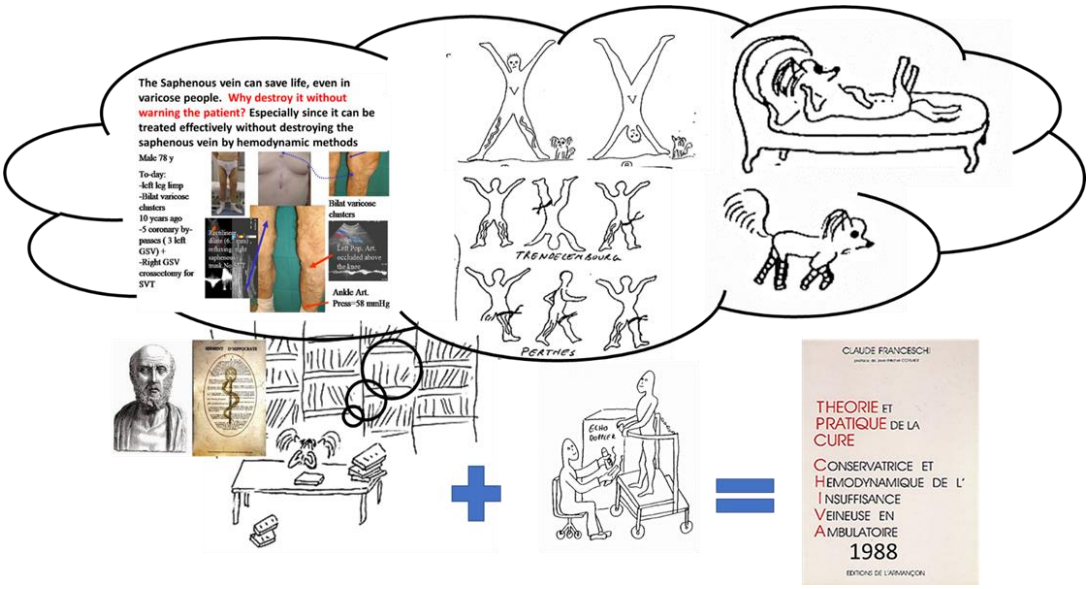
Paranà Manoeuvre

Paranà River

These simple postural and dynamic data helped me better understand venous disease. The reason why even severe valve incompetence is asymptomatic and does not interfere with venous return in the supine position but becomes pathogenic as soon as standing and even more so when walking. This is why valvular incompetence is asymptomatic in paraplegics and bedridden patients!

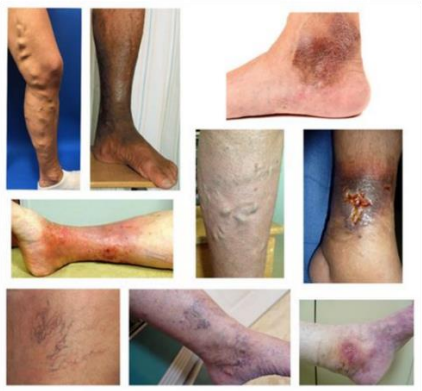
The origin, the pathway, the destination of the flows, according to the postures and the activity of the pumps, led me to understand that the ***varicose veins and other signs and symptoms are not the cause but the result of a hemodynamic disorder*** *due to valvular incompetence and/or resistance to the flows (venous obstacles, cardiac or thoracoabdominal failure).*

All these hemodynamic disorders, whatever the cause, have a common effect which is an excess of Transmural pressure (TMP). This is what dilates the veins and, by hampering drainage, causes oedema, hypodermitis and ulcers.



Whatever the clinical manifestation, the diagnosis and treatment must seek out the cause of the excess transmural pressure TMP and treat it.

Whatever the etiology, signs and/or symptoms, venous insufficiency is always due to an excess of Transmural Pressure (TMP)



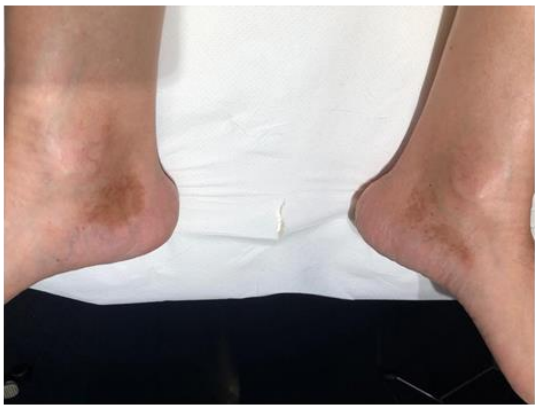
Here is an example of clinical, echodoppler and therapeutic discordance due to different pathophysiological and hemodynamic models

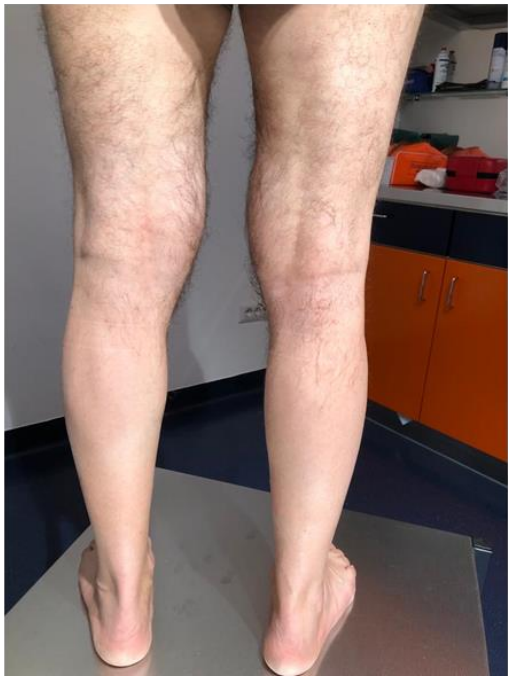
53 year male, architect.
Symptoms:

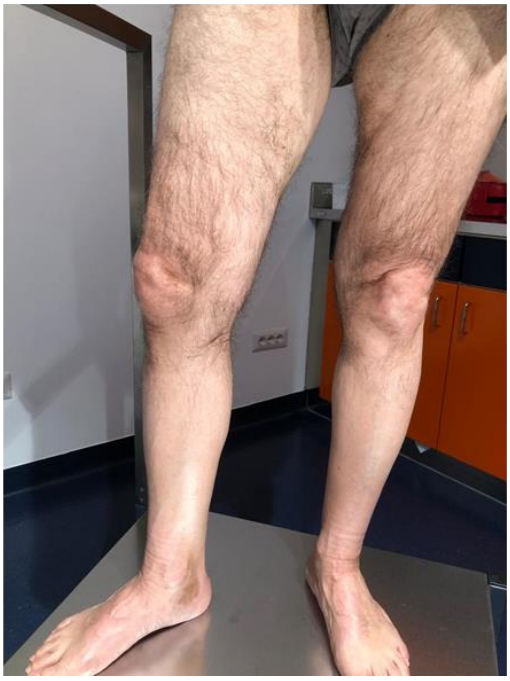
Pain in both soles as soon as standing then progressively relieves with walking

Posterior paresthesia of both thighs when seated.

Signs : Light pigmentation of the medial face of the heels. CEAP: C4. No visible varicose nor spider veins. No edema. No hypodermitis.







[redacted]

Lower Extremity Venous Report

Location: [redacted] MRN: [redacted] Date of Service: 5/12/2021
Patient Name: [redacted] DOB: 2/13/1968
Referring Physician: [redacted], MD, FACC, FCCP Gender: M
Interpreting Physician: [redacted], MD, FACC, FCCP Age: 53
Sonographer: [redacted]

CPT: **93970**

Study Quality: **Technically adequate**

Lower extremity venous imaging was performed utilizing B-mode, color flow, pulsed Doppler and spectral analysis.

Physician Review

Conclusions: 1. Severe deep system reflux noted in the right lower extremity. No DVT bilaterally.

Findings:

Right: Normal compressibility of the deep veins in the right lower extremity.
Severe reflux seen in the femoral vein posterior tibial vein.

Left: Normal compressibility of the deep veins in the left lower extremity.

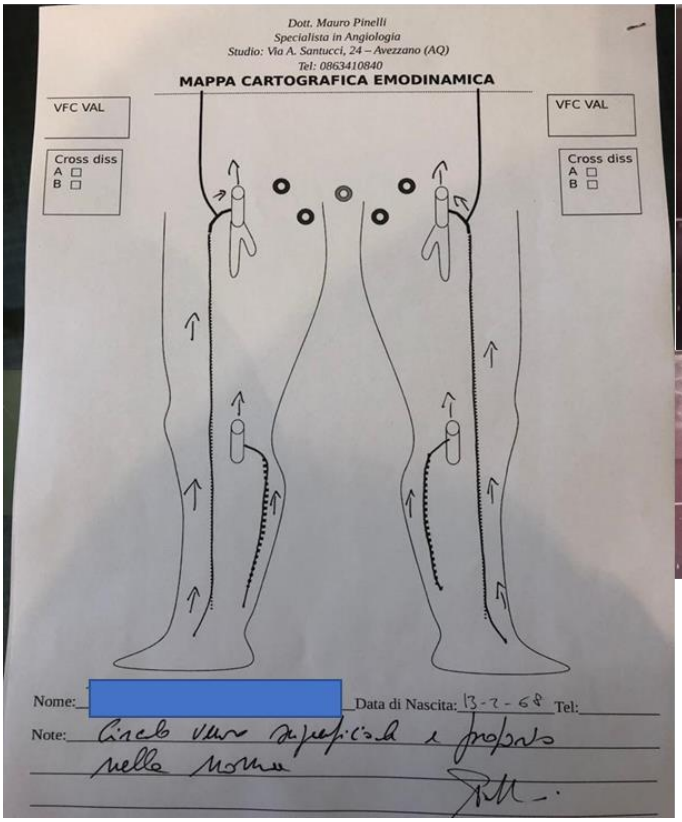
Electronically signed [redacted], MD, FACC, FCCP 5/16/21 12:35 PM

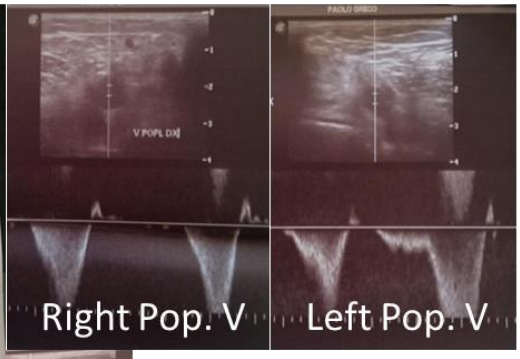
Exam Data

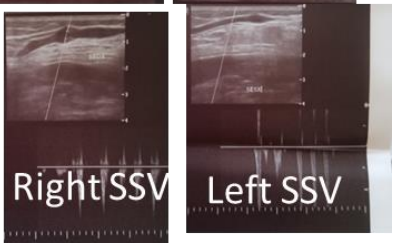
	EIV		CFV		FV		POP		PTV		GSV at SFJ		GSV		PER	
	R	L	R	L	R	L	R	L	R	L	R	L	R	L	R	L
Spontaneous:	☐	☐	☒	☒	☒	☒	☒	☒	☒	☒	☐	☐	☐	☐	☐	☐
Phasic:	☐	☐	☒	☒	☒	☒	☒	☒	☒	☒	☐	☐	☐	☐	☐	☐
Patency:	☐	☐	☒	☒	☒	☒	☒	☒	☒	☒	☐	☐	☐	☐	☐	☐
Augmentation:	☐	☐	☒	☒	☒	☒	☒	☒	☒	☒	☐	☐	☐	☐	☐	☐
Compressible:	☐	☐	☒	☒	☒	☒	☒	☒	☒	☒	☐	☐	☐	☐	☐	☐
Valsalva:	☐	☐	☒	☒	☒	☒	☒	☒	☒	☒	☐	☐	☐	☐	☐	☐
Competent:	☐	☐	☒	☒	☒	☒	☒	☒	☒	☒	☐	☐	☐	☐	☐	☐

Report for [redacted] 5/12/21

According to signs, symptoms and different reports from 2 different health centres, EVLA is suggested without info about possible future need GSV nor conservative therapies.
Patient finds info on the web about possible conservative and travels to Europe to have a CHIVA cure.
New Ecodoppler is performed personally by an experimented angiologist who concluded: Normal deep and superficial venous network of both lower extremities and suggests lumbarspine MRI.







26.10.2021

ECO COLOR DOPPLER VENOSO DEGLI ARTI INFERIORI E CONSULENZA ANGIOLOGICA

COGNOME: [redacted]

DATA DI NASCITA: 13.02.1968

REFERTO:

Segni ecografici ed emodinamici di pervietà, completa compressiblità e continenza valvolare si rilevano lungo il decorso delle vene femorali comuni e superficiali, poplitee,tibiali, peroneali, gemellari, soleali, safene esterne e safene interne.
Le vene plantari sono compressibili.
Circolo arterioso pervio e indenne da lesioni ostruttive o stenosanti.

CONCLUSIONI

Circolo arterioso e venoso degli arti inferiori nella norma.
La sintomatologia dolorosa e le parestesie lamentate hanno una verosimile genesi neuroradicolare .
Possibile fascite plantare .

Dott.Mauro Pinelli
Specialista in Angiologia

[redacted]

Centro Diagnostico [redacted]

Via degli Stadi, 2 - Piazza Europa - 87100 Cosen
Radiologia Medica - Laboratorio Analisi Cliniche, Ambula[illegible] Polispecialistico, Ecografia, Ecocolordoppler,
Radiologia Digitale, Radiologia Pediatrica, Radiologia Dentale, MOC, Mammografia, Risonanza Magnetica.
E-mail: laboratoriosanfrancesco@gmail.com Web Site: www.laboratoriosanfrancesco.com

[redacted]

Data: 29/10/2021

Data di nascita: 13/02/1968

RM RACHIDE LOMBOSACRALE

Esame eseguito con sequenze TSE pesate in T1, FSE pesate in T2 e STIR, ottenute su piani sagittali, e con sequenze FSE pesate in T2, ottenute su piani assiali, condotti a livello degli spazi intersomatici L1-L2, L2-L3, L3-L4 ed L4-L5, L5-S1.

Iniziali alterazioni spondilo-artrosiche a cui si associano segni di artrosi interapofisaria con ipertrofia delle faccette articolari e dei legamenti gialli.
Fenomeni degenerativi di disidratazione a carico dei dischi intersomatici del tratto esaminato, che presentano riduzione della propria intensità di segnale nelle sequenze pesate in T2 e appaiono di altezza ridotta da L3 a S1.
Si rileva la presenza di protrusioni discali ad ampio raggio. In particolare esse si localizzano:
-A livello di L3-L4, prevalentemente estesa in sede paramediana-laterale destra, ove determina restringimento del canale radicolare corrispondente e fenomeni compressivi nei confronti della corrispondente emergenza radicolare;
-A livello L4-L5 ed L5-S1, che si estrinsecano prevalentemente in sede paramediana/laterale destra ove contattano le emergenze di L4 e L5 omolaterali.
Non aree di alterata intensità di segnale a carico del cono midollare.

L3-L4 : reduced radicular channel
L4-L5, L5-S1 : contact emerging nervous roots

EVLA was as suggested without informing of the potential necessity of his GSV for a vital arterial by-pass after the 2d report. The patient traveled to Italy to be operated by a CHIVA expert in order to save his GSV. The Angiologist partner of the vascular surgeon performed an ecodoppler that resulted in normal deep and superficial veins

Conclusion: Veins and arteries of the lower extremities are normal, evidenced by Ecodoppler data.

C4 CAP in this case is not reliable for venous insufficiency grading.

Pain in both as soon as standing and posterior paresthesia of both thighs when sitting are not symptoms of venous insufficiency. MRI confirms related lumbar spine damages.

No clinical signs of venous insufficiency (see photos) except a light pigmentation (CAP C4 grade of venous insufficiency of the medial face of the heels not exceptional in an individual who works seated along his lifetime (architect). C4 CAP in this case is not reliable for venous insufficiency grading.

2 discrepant Ecodoppler performed in 2 different places by different sonographers and interpreted as deep severe venous insufficiency of one limb by a referent MD and as severe superficial insufficiency of both GSV and both SSV by an other MD, then interpreted as normal by an experimented angiologist who performs personally the ecodoppler. This underlines the difference of ecodoppler interpretation according to the hemodynamic and pathophysiological and clinical background of practitioners.

In any case, why a destructive “treatment” of the saphenous veins in a venous asymptomatic patient, whatever the ecodoppler findings, has been proposed. For the patient’s sake?

:

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Chapter 1

Each chapter includes some of the elements of the previous chapters and anticipates those of the following chapters.

Chapter 1

1- Definitions of Venous Function, Venous System, Venous Insufficiency and Transmural Pressure.

11- Venous function has three main objectives.

12- The venous system is the set of organs that provides the movements and flow pressures necessary to perform its functions.

13- Venous insufficiency.

14- Venous pressures.

141-Transmural pressure (TMP) is the key hemodynamic parameter of venous functions.

1411- Intravenous lateral pressure IVLP should be as low as possible.

14111-Residual RP pressure

141112- The reservoir effect

141114- Cardiac, Thoracic and Abdominal Pumps

141115- Valvulo-muscular pumps

14112- Gravitational hydrostatic pressure

14113- Pressure gradient

1412- Extravenous pressure

142- Oncotic pressure

143- Thinking about TMP and knowing its parameters lifts the veil on the main "mysteries" of venous insufficiency.

144- Waterfall et Starling Resistor

1- *Definitions of Venous Function, Venous System, Venous Insufficiency and Transmural pressure.*

The definition of venous insufficiency and the hemodynamic parameters and concepts are essential to the understanding of venous physiopathology. This theoretical understanding is not always emphasized. However, it is essential for good practice. So, the clinical, diagnostic and therapeutic context is recalled with each of these definitions and explanations. The reader will also find analogies and comparisons in order to understand certain counterintuitive concepts with which he was not familiar during his medical studies or at conferences.

Moreover, these definitions are necessary for the understanding of this book. *Indeed, they are intended to avoid the frequent misunderstandings related to terms and concepts whose definitions are imprecise or contradictory.*

Venous function, venous insufficiency and the venous system are not limited to venous return, veins and varicose veins .

11-*The venous function is triple: Drainage of tissues, Reservoir effect and Thermoregulation.*

Venous Insufficiency is the inability of the venous system to provide all or part of these three functions.

To maintain a low Venous Transmural pressure (TMP) in order to:

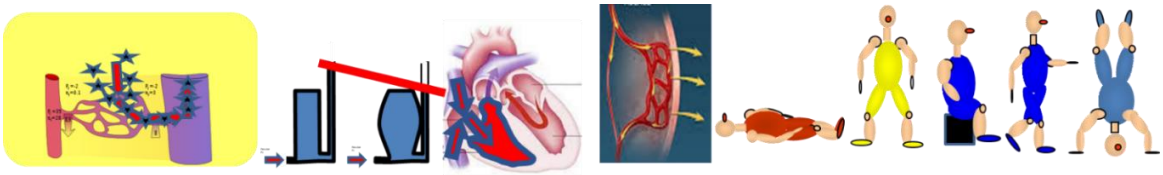
-Drain the tissues and

-Provide the appropriate preload of the right heart through the reservoir effect

Regardless of posture and muscular activity.

To participate in thermoregulation.

The venous system is made up of veins and pumps whose characteristics and actions determine the Transmural pressure (TMP), a central hemodynamic parameter of venous physiopathology.



Drainage | Reservoir effect | Thermorégulation | Postures et activities

12- *The venous system is the set of organs that provides the movements and pressures of the flows necessary to carry out its functions.*

***Five organs** are necessary for venous function.*

-The microcirculation that receives liquids, waste and catabolites from the tissues,

-The veins and venules in which they flow and

-The 3 serial pumps: cardiac, thoraco-abdominal, and valvulo-muscular pumps that push them towards the right atrium.

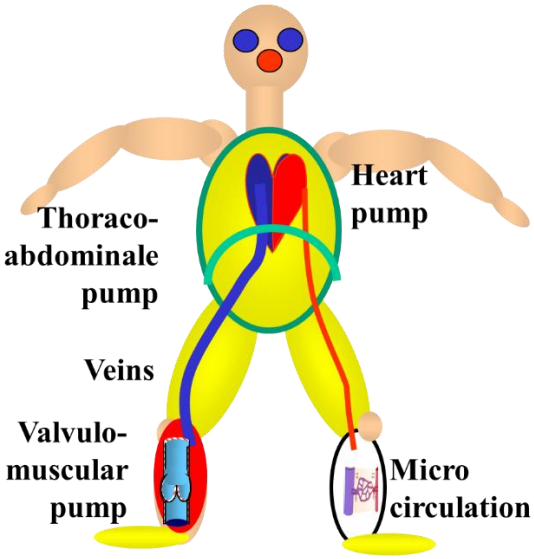
It permanently adapts

-the direction,

- the flow and

-the pressure of the venous blood

to the needs of the venous function



5 Organs of the venous system

13- *Venous Insufficiency.*

I call venous insufficiency any inability of the venous system to

*-Reduce the excess of **Transmural pressure (TMP)***

*That **impairs its functions** of*

-Drainage of tissues,

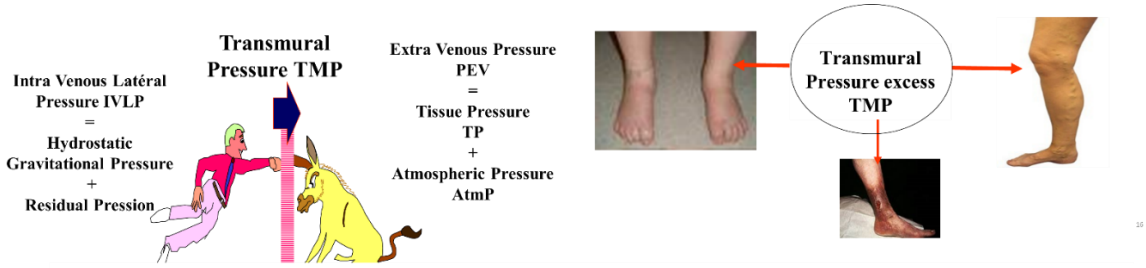
- Thermoregulation, and

- Preloading of the right heart

whatever the posture and muscular activity.

It is due to the <u>failure of one or more organs</u> of the venous system.

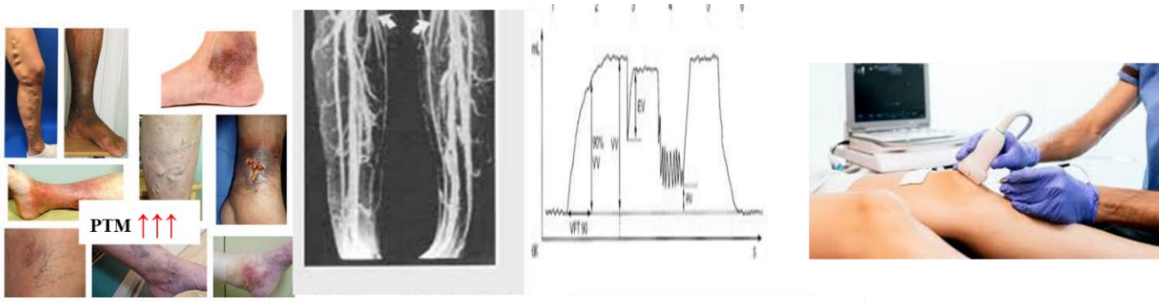
<u>The clinical presentations are not pathognomonic</u> of a specific venous impairment and may be <u>confused with non-venous aetiologies</u>.



Whatever the etiology, signs and or symptoms,

Venous insufficiency is always due to an excess of Transmural Pressure (TMP)

Therefore, <u>paraclinical investigations</u>, foremost among which is the Echodoppler, <u>are necessary for pathophysiological diagnosis and appropriate therapeutic strategies.</u>



CAP Venographie ARM
Non Hemodynamic
Non topographic Non structural

Plethysmography
Hemodynamic
Non topographic
Non structural

Echodoppler
Hemodynamic
Topographic
Structural

Diagnosis of the causes of excess Transmural Pressure (TMP)

It is a mistake to reduce the physiology and pathology of the venous system to the veins alone.

Varicose veins and trophic disorders are not the cause, but the signs of the lack of control of Transmural pressure TMP by all or part of the five organs that constitute the venous system.

Recognition of the true causes of excess TMP allows for rational and effective diagnosis and treatment.

Approaching venous insufficiency through clinical aspects (CEAP), limited and poorly interpreted instrumental hemodynamic data (reflux or non-reflux) and various recipes for destroying the veins, demonstrates a lack of knowledge of the physiopathology and leads to therapeutic impasses.

14- *Venous pressures.*

I define here the different venous pressures by outlining their relationship with physiopathology. They will be explained in more detail in the following chapters.

141-Transmural pressure (TMP) is the key hemodynamic parameter of venous functions.

It is the determining parameter of the main venous function i.e. tissue drainage.

It results from the difference between two pressures

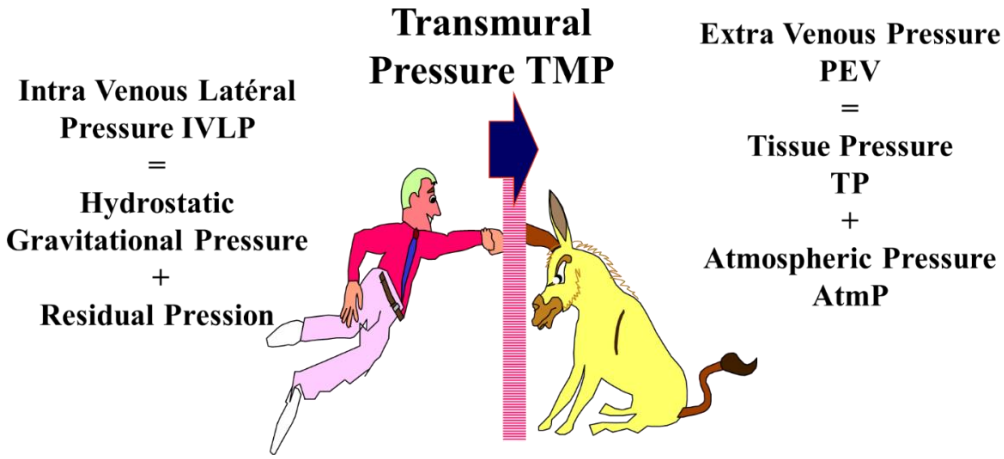
- the lateral intravenous pressure (IVLP) and

-the Extra-Venous pressure EVP,

-which are opposed on either side of the venous and capillary walls.

It is almost always positive, and therefore contrary to drainage.

<u>Fortunately, the venous system keeps it lower than the osmolar (oncotic) drainage force which attracts the interstitial liquid into the capillaries, namely the Oncotic pressure OP</u>.



1411- The lateral intravenous Pressure IVLP should be as low as possible to ensure physiological tissue drainage.

It is produced by

-the static part of the Residual pressure RP and the

-Gravitational Hydrostatic Pressure GHSP.

IVLP = static RP + GHSP, i.e., the pressure against the inner surfaceof the venous wall.

It is opposed to the extravenous pressure EVP, which is the sum of the tissue pressure TP and the atmospheric pressure AtmP TMP= IVLP-PEV= (RP+GHSP - TP + AtmP)

TMP is the result of this opposition.

14111- Residual pressure RP is produced by the Cardiac Pump.

It results from arterial pressure AP decreased by its loss of load (driving pressure) in the microcirculation.

It varies according to the microcirculatory resistance MCR, the Reservoir Effect, the downstream venous resistance to the flow.

These variations are explained by Bernoulli's Theorem which also considers the Gravitational Hydrostatic pressure GHSP.

141111- Microcirculatory resistance decreases with the dilation and recruitment of microcirculatory units, the opening of arteriolo-venous micro-shunts, particularly during muscular effort, when it is hot and in the event of inflammation.

141112- The Reservoir Effect reduces intravenous lateral pressure IVLP

The reservoir effect reduces intravenous lateral pressure IVLP as long as the passive and active elastic compliance of the venous walls can offer little resistance to the increase in volume of the veins (according to the ratio of pressure/volume/resistance).

Thus, the reservoir effect dampens variations in Intravenous Lateral Pressure IVLP to comply with the right ventricle preloading requirement.

141113- Venous Resistance progressively increases Residual pressure RP according to the hemodynamic significance of the downstream obstacles, until it equals the Arterial pressure when the latter achieves a complete obstruction.

The venous flow which has become zero also stops the arterial flow (ischemia of Phlegmatia Cerulea).

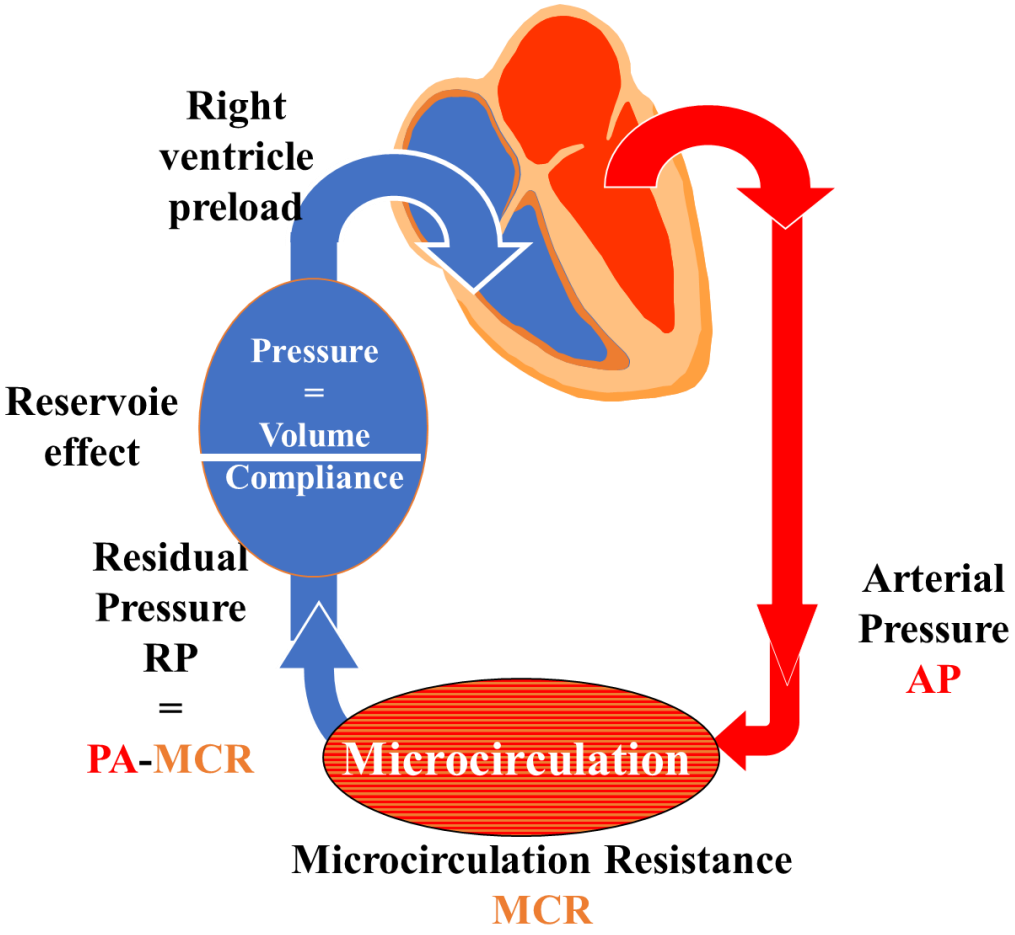
These resistances explain the pulsatility of venous flow when downstream resistances are severe.

They decrease secondarily in proportion to the opening of collaterals that I call vicarious "open shunts" that the CHIVA cure respects.

These open vicarious shunts are often recurrent varicosities and varicose veins after destructive treatments. They are welcome when they compensate for deep venous obstructions.

141114- Cardiac, thoracic, and abdominal pumps reduce the flow resistance, *thus the Residual pressure. Indeed, diastole permanently sucks in the microcirculatory flow (vis a tergo).*

Thus, any suction defect of these pumps increases the Residual pressure, thus the TMP.



141115- Valvulo-muscular pumps (VMP) work only during walking.

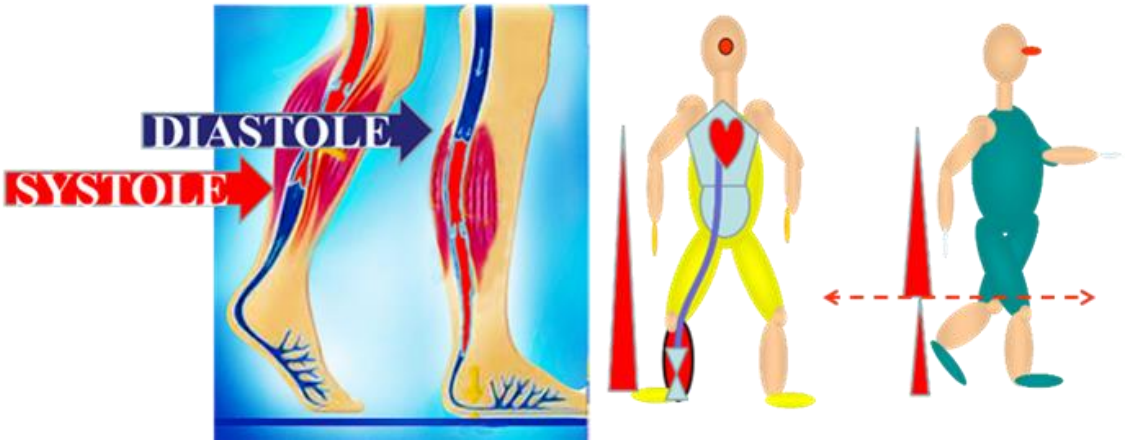
They draw in the excess microcirculatory flow increased by muscular effort.

In case of valvular incompetence*, the flow/pressure supplied by the pump is pushed back upstream and overloads the residual pressure.*

This backflow is direct or indirect*.*

It is direct, *systolic, and diastolic when inlet and outlet veins of the pump are incompetent.*

It is indirect and only diastolic when the competent pump is shunted by incompetent collaterals, which I call "closed shunts".



The CHIVA treatment consists of disconnecting these incompetent veins from the source of the reflux, but without destroying them so as not to create an obstacle to the drainage of its territory (phlebosome), which is a source of skin suffering and varicose recurrence under the pressure of the residual pressure that it has increased.

14112- the Gravitational Hydrostatic pressure GHSP

The Gravitational hydrostatic pressure GHSP is a static, potential energy in Bernoulli' equation. It depends on the Universal Force of Gravity (Newton) and its application to fluid statics by Stevin, Torricelli, and Pascal. GHSP =ρ gh (ρ= Specific mass, g=gravity acceleration, h=fluid height)

It varies according to the vertical height, unfragmented, of the venous blood column, from the feet to the heart, and therefore according to the posture.

In Bernoulli's formula GHSP is measured as the potential energy to account for the Total pressure constant.

It has three remarkable characteristics in humans.

1-The first is the contrast between its almost zero value in the supine position and its very high value in the standing position (90 mmHg). *Its hemodynamic impact is major and dominant over the other pressure variations.*

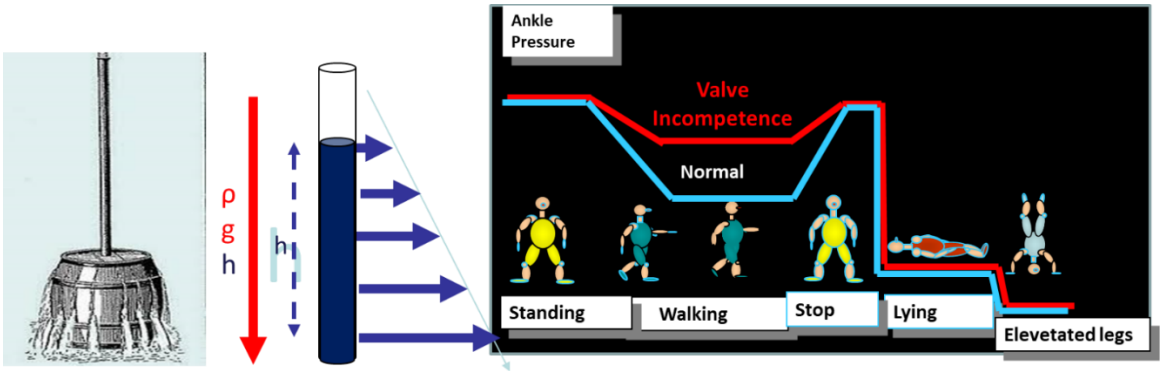
2-The second is its reduction during walking (30 mmHg) which I have related to a Dynamic Fractionation of the Gravitational Hydrostatic pressure DFGHSP *produced by the alternating closures of the valvulo-muscular pumps. The CHIVA cure restores this fractionation when it is impaired by valvular incompetence.*

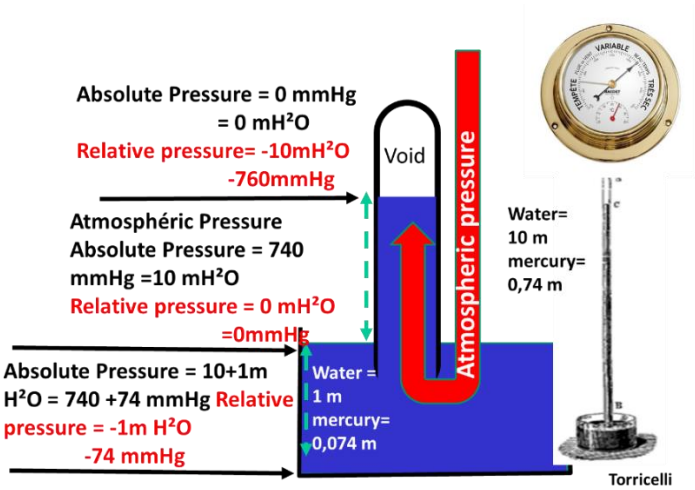
We understand how the Gravitational Hydrostatic pressure GHSP can be determinant as a cause of the Venous Insufficiency.

We also understand the effectiveness of the treatments by simply raising the legs.

3-The third is the value measured at the ankle in a standing *position equal to the ankle-heart height and not to the expected "true" height of the ankle-skull blood column.*

I will explain later the reason related to the transmission of the atmospheric pressure to the human body, part of which behaves like... a barometer*.*

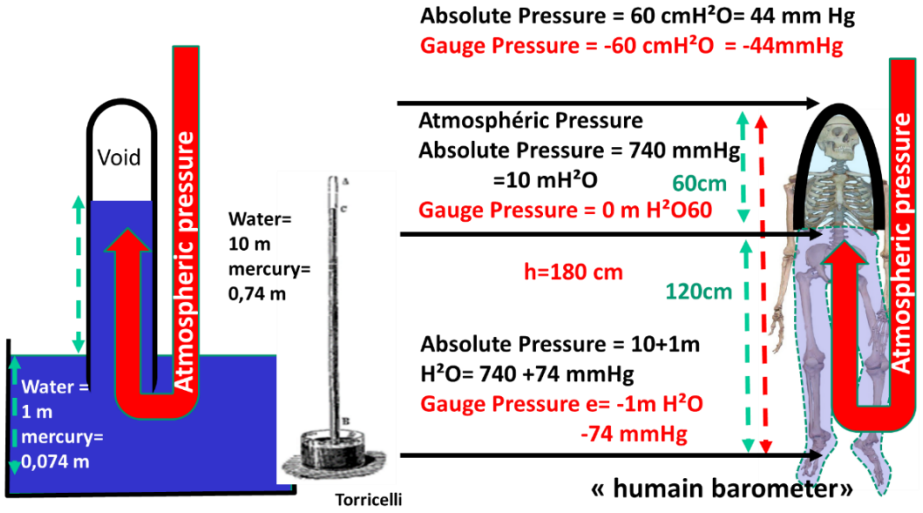




Barometer: absolute pressure

Tensiometer (Sphygmomanometer) and Catheter: Relative pressure (Gauge pressure),

The pressure usually measured is the relative pressure called gauge pressure = absolute pressure - atmospheric pressure.
The atmospheric pressure measured by barometers is the absolute pressure.



Barometer: absolute pressure

In humans, the negative pressures are the pressures lower than the atmospheric pressure. In the standing position, the venous pressure is negative above the diaphragm because the rigidity of the walls of the skull and thorax do not allow the direct transmission of atmospheric pressure, so the skull and thorax behave like barometers. This explains why the pressure at the ankle in standing position is equal to the ankle-diaphragm height and not the ankle-skull height.

14113- Pressure gradient PG.

The pressure gradient PG is the pressure difference ΔP between two points of a continuous fluid, separated by a length D. PG= ΔP/D. It is not the cause of the pressure difference but its measurement *in each hydrodynamic context. For example, between 2 points A and B of a stationary liquid, the pressure measured at the lowest point A is higher than at the highest point B, but the potential hydrostatic pressure(energy) is higher at B than at A.*

Recall that Transmural pressure TMP is the pressure difference between two points separated, not by fluid, but by a wall. So, it is not called pressure gradient

1412- Extravenous pressure EVP

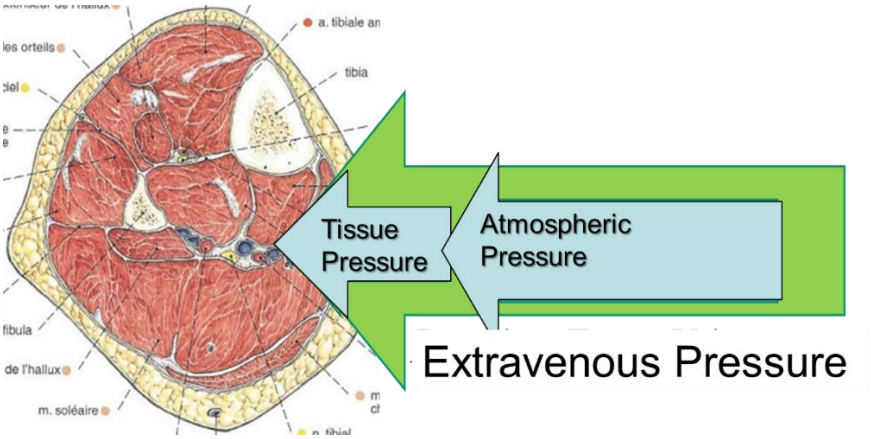
The Extra-Venous pressure EVP reduces the TMP by opposing the lateral Intra-Venous pressure IVLP.

It thus promotes drainage.

It is the sum of

-the atmospheric pressure AtmP (10 kg/cm² at sea level!) which decreases with altitude and

-the tissue pressure TissP trans mitted to Interstitial fluids which varies with the surrounding structures, passive (fascia) and active (muscles).



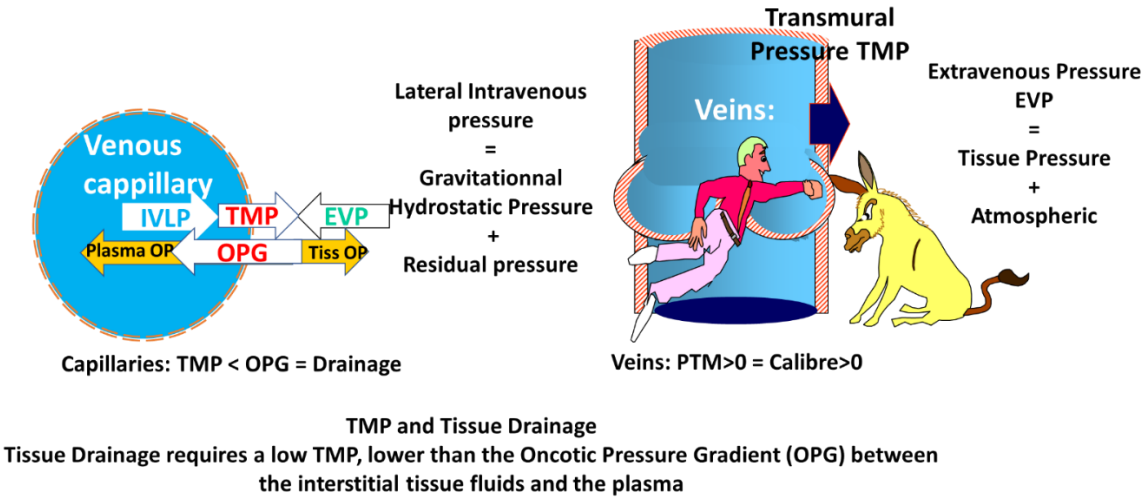
142- *Oncotic pressure OP*

Transmural pressure (TMP), Intra Venous Capillaries Lateral Pressure (positive mechanical pressure) directed towards the tissues, hampers drainage.

However, drainage is possible thanks to a superior opposite force of the Oncotic (osmotic) Pressure Gradient. *It is the plasma oncotic pressure POP of the macroprotéines, higher than that of the macroprotéines IOP of the interstitial fluids, which creates an oncotic pressure gardient OPG favourable to drainage.*

The semi-permeable capillary wall does not allow plasma drainage of the tissue interstitial macroproteins. The latter are drained by the lymphatic system.

<u>We understand the interdependence of the lymphatic and venous systems in their drainage functions, as we will see later.</u>



TMP and Tissue Drainage
Tissue Drainage requires a low TMP, lower than the Oncotic Pressure Gradient (OPG) between the interstitial tissue fluids and the plasma

143- ***<u>Thinking about TMP and knowing its parameters lifts the veil on the main "mysteries" of venous insufficiency.</u>*** ***Remember that Bernoulli***

and Poiseuille were also doctors who established the laws of fluid mechanics to better understand hemodynamic.

<u>To ensure tissue drainage</u>, the TMP must be lower than the trans-capillary oncotic pressure gradient.

<u>To ensure a Reservoir effect</u> favourable to the filling of the heart, the IVLP must remain stable despite variations in the volume of the venous bed. This is particularly the case during postural variations in the Hydrostatic Gravitational pressure GHSP and variations in the thermoregulatory flow rate

144- Waterfall and Starling Resistor The definitions and contexts of Waterfall and Starling Resistor are rarely presented clearly so that they remain unclear and mysterious to many. Here are the definitions I have in mind.

1-If Waterfall is the waterfall from the top of a dike, the physical parameters attributed to it are either the height of the dike behind the dam, or the height of the waterfall in front of the dike. According to the laws of physics, the falling water simply responds to gravity but does not attract the water behind the dike. Thus, the flow of the dam does not depend on the height of the dike but on the height of the water source relative to the top of the dam. The water behind the dam overflows when its surface exceeds the upper limit of the dam. The water can be made to flow when its surface is lower than the upper limit of the dike by using the siphon effect. Thus, a rigid pipe immersed in the water of the dam, which then spans the dike and descends in front of the dam lower than the water surface, allows the flow thanks to the difference of potential gravitational energy between the two ends of the pipe, without resistance to the flow because the atmospheric pressure is exerted in the same way at both ends. These conditions are not fulfilled in the vessels. **They are flexible pipes, which obey the Navier Stokes equations, but more simply, although more roughly, the laws of Poseuille and Bernouilli**. We can however retain an analogy between the minimum height of the water surface in relation to the top of the dam, capable of overflowing the dam and the minimum blood pressure in the vessels, air in the avéoles, to overcome arteriolo-cappilar or bronchiolar obstacle . This is the Critical Closing Pressure that corresponds to the pressure value below which blood or air can no longer flow.

2- The Starling Resistor

In the classisic laboratory model (Holt 1941, Permutt 1962), a liquid flows through a hose with a flexible and collapsible segment (Penrose drain) horizontally subjected to a gravitational energy gradient between a Mariotte bottle (constant level liquid) that supplies it and its lower end that drains it. This showed 2 phenomena, first that **the more the lower end is opened, the more the tube collapses**, creating a resistance that reduces the pressure downstream of the collapsed segment**, second that any external pressure applied on the tube produces the same effect**. This is the Transmural Pressure: Internal Pressure - External Pressure, which decreases with the decrease of internal pressure by any means (including raising the feet above the head) or that we increase the external pressure (by compressible bandage for example**).This is the demonstration of transmural pressure TMP** which shows

that the flow in a flexible vessel is stopped by an external pressure greater than or equal to the internal lateral pressure (static), produced by any means (air pressure, air, tissue etc.. Note that the internal lateral pressure does not change, except when the reduction in caliber becomes sufficiently important to create velocities such that the internal lateral pressure (static) decreases in favor of the dynamic energy, due to the importance of the velocity v^2, according to the Bernoulli equation. This explains the vibrations, because as soon as the duct collapses, the velocity drops to 0 which stops the collapse effect so that the "stenosis" opens again. The return of the high speed reproduces another collapse and so on. This produces a succession of opening-closing, thus a vibration that can also be observed and for the same reason between the lips of the trumpeter and undoubtedly the vocal cords of singers. At the level of the veins, the velocities are rarely high enough to create this phenomenon of murmur as in the jugular gullet and much less frequently than in arterial stenoses and AVFs where the ssystolic velocities can be very high in the frequent "significant" stenoses)

3- The critical closure pressure is the internal pressure at which a blood vessel collapses and closes completely. If the blood pressure falls below the critical closing pressure, the vessels collapse. This occurs when measuring blood pressure with a sphygmomanometer. At rest, the critical arterial closure pressure, i.e. the pressure at which the flow stops, would be ~ 20 mmHg. This means that BPs below 20 mmHg cannot be measured with a sphygmomanometer (stop of Korotkoff sounds)

Chapter 2

Each chapter includes some of the elements of the previous chapters and anticipates those of the following chapters.

2- Forces, pressures, and resistances

21- Force and energy

22- Gravitational force, Archimedes and venous pressures

23- Circulatory regimes, Bernoulli's theorem, Poiseuille's law, Reynolds number and vascular applications

231- Circulatory regimes

232- Bernoulli's theorem

233. Poiseuille's law and Reynolds number

2331-Reynolds number and turbulence

2332- Poiseuille's Law and pressure drop 23321.

23321- Pressure loss and hemodynamically significant stenosis.

23322-Effects of significant stenoses on veins and drainage

233221- Increased residual RP pressure provided by microcirculation and/or systolic pressures of valvulomuscular pumps.

233222- Collaterals and resistance

233223- Measurement of ascending pressures: invasive and and Doppler.

233224- Pseudostenosis: Pseudo May Thurner syndrome.

May Thurner Syndrome MTS and Nutcracker Syndrome NTS.

233225- Stents and Recanalization

233226- Downstream Pressure and Guyotan equation

24- Gravitational Hydrostatic Pressure

25- Dynamic fractionation of gravitational hydrostatic pressure.

26- Paradoxical hydrostatic pressure and atmospheric pressure

27- Pump pressure

271- Cardiac pump

2711- Reservoir effect.

2712-Residual RP pressure and Microcirculatory Resistances.

2713-Right heart failure

272- Thoracoabdominal pump

2721- Respiratory physiological modulation of lower extremities flow and pressure.

2722- Pathologic respiratory modulation of lower extremities flows and veins.

273- Valvulo-muscular pump. Dynamic fractionation of gravitational hydrostatic pressure DFGHS, valvular incompetence and shunts.

2731- Dynamic fractionation of gravitational hydrostatic pressure DFGHSP

2732- Veno-venous shunts, valvulo-muscular pump and cardiac pump

27321- Definition of shunts

27322- Hemodynamic classification of venous shunts. OVS, CS and ODS.

273221-Venous shunts favorable to drainage

273222-Venous shunts against the drainage

2732221-Venous shunts in favor of drainage

2732222-Open deviated shunts ODS hinder drainage

2732223-Open Vicarious Shunt OVS Facilitates Drainage

2732224-A mixed MS shunt associates OVS that facilitates drainage with a CS that hinders drainage.

28- Plasma oncotic pressure POP and interstitial pressure IOP

29- Lateral intravenous pressure (IVLP), driving pressure, pressure gradient and pathophysiology

291- Lateral intravenous pressure IVLP is the sum of the following

292- Motor pressure MP = p + (1/2) mv2, Obstacle and valvular incompetence

293- Pressure gradients

294- Siphon effect 295- Extravenous pressure EVP

295- Extravenous pressure EVP

2951- Atmospheric pressure AtmP and gravitational hydrostatic pressure

2952 Extravenous Tissue Pressure

2953- Extremity compression

29531- Homogeneous compression

295311- Immersion in liquid

295312- Air inflated cuff

29532- Heterogeneous compression.

295321- Non-elastic compression

295322-- Elastic band and compression stocking.

296- Measurement of venous pressure

2- Forces, pressures, and resistances.

Venous pressure P is the result of a force F exerted on a surfaceS, P=F/S.

The venous system provides different types of pressure according to the forces that produces them.

The force of gravity and the force are produced by the various pumps.

The driving (motive) pressure MP *of the pumps that push and pull blood to the heart.*

The gravitational hydrostatic pressure GHSP *attracts the blood down.*

Tissue pressure exerted on veins, interstitial fluid and atmospheric pressure *compress the veins and microcirculation.*

TMP is the result of the interaction of these various pressures*.*

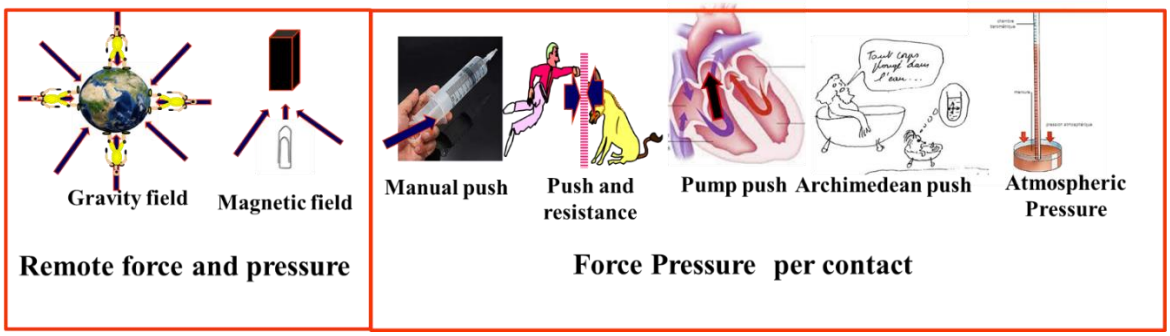
The laws of fluid mechanics have been established throughout the history of science. These laws are the basis of the hemodynamic of the venous system. Force is expressed in newtons and energy in joules. Pressure is expressed in equivalent values that can be converted into each other, either in Pascals, in cm of water or in mm of mercury.

These laws deserve to be well understood *because they allow a better understanding and treatment of venous insufficiency.* ***They are accessible to non-physical physicians if they study them patiently and accept those that are often counterintuitive.***

Bernoulli and Poiseuille established equations *of fluid mechanics which are applicable to blood with a good approximation.*

Bernoulli's equation describes the energies that produce the static, potential and dynamic pressures and that convert one into the other according to the law of conservation, but only under conditions of circulation where the effect of viscosity is negligible.

Poiseuille's equation describes the conditions, particularly of velocity, in which the effect of the viscosity is no more negligible. It measures the loss of charge (hydrodynamic energy and related pressure) converted in mechanical and/or thermal energy, as in stenosis.



21- *Force and Energy*

In its hydrodynamic expression, energy acts in the venous system to ensure its functions.

The forces of gravity and pumps interact with the resistances of the venous and capillary walls, the tissue environment, and the atmospheric pressure. The result is a transmural pressure TMP that is low enough to drain the tissue but high enough to ensure the return of blood to the heart.

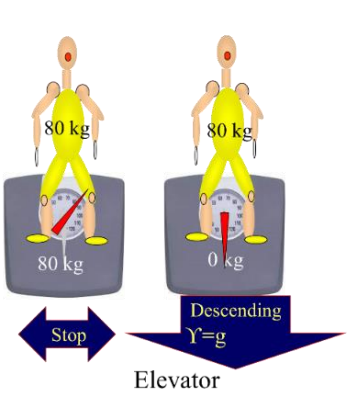
*In physics, energy is the capacity of a system to produce work (Joules) . It exists in many forms, including mechanical and thermal energy, which can be transformed into each other. The force F provides the mechanical energy that can move an object of mass m with an acceleration ϒ in a direction determined by its vector F=mϒ (Newtons). If this object is prevented from moving by a resisting force, this energy is called potential Pe, such as water held back by a dike, a stone a tile placed on the edge of a roof, the arrow held back by the tight string of a bow. According to the law of conservation of energy, it can be transformed into kinetic energy ec (ec = work of the applied forces F necessary to make the body m go from rest to its motion v = ½ mv²) when the resistance is null. The water retained by the dam transforms its potential energy into electricity. When it falls from the roof the potential energy of the tile is transformed into kinetic energy which breaks it against the ground. The string of the bow released transforms its potential energy into displacement kinetic energy of the arrow in motion. Thus, the kinetic energy Ec increases in proportion to the potential energy Pe which decrease**s.***

We must distinguish two forces of different nature.

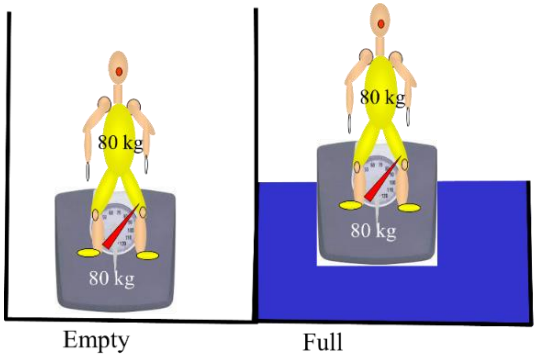
<u>The force of gravity mg</u> *is the force which acts on the tile and the water of the dam, it* ***acts at <u>remote force</u> and produces the <u>gravitational hydrostatic pressure</u>.***

<u>The force by contact</u> *mϒ* ***produced by the pumps acts <u>directly on the fluids and any objects</u>.***

22- *Force of gravity, Archimedes, and Venous Pressures*



Elevator
Weightlessness (weightlessness)
Descent acceleration ϒ = g Gravity
The weight measured in the elevator changes



Small boat in a pool

80 kg

Immersion in water
The weight measured does not change but it is differently distributed = feeling of lightness

The force of gravity acts at a distance on blood as on any other object or liquid. Its acceleration ϒ is that of gravity conventionally designated by g produced by a gravity field. It acts permanently and at a distance on any solid or liquid object (blood) as would a force field (like the magnetic field that mobilizes iron at a distance without touching it) in a strictly vertical direction (vector) towards the centre of the earth. It is inversely proportional to the square of the distance d² between the earth and the human body (Newton)*. This force decreases as we move away from the earth but in minute proportions on earth, including when we climb a mountain, or we move by plane. g remains practically equal to 9.8 m/s. Note that this force acts but is no longer felt when an elevator or an airplane descends to earth with acceleration equal to g (Einstein's thought experiment). Gravity exists everywhere and in any situation. In fact, the weight mg (density) is only felt when a surfacereacts against it (the floor of an elevator at a standstill and a tile that breaks on the ground). It continues to exist, but is no longer felt when the elevator descends at accelerated speed g. There is no absence of gravity (weightlessness) but its feeling because in*

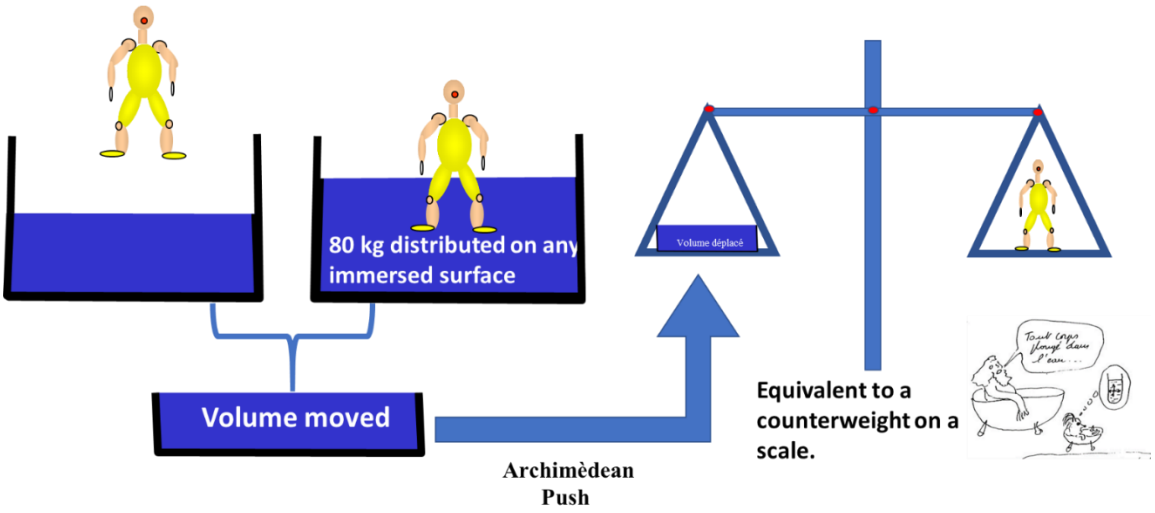
the absence of resisting force, we no longer feel its weight. If we are standing on a scale in an elevator that descends with the acceleration g, the scale marks 0 kg! In this case, the blood no longer suffers its weight and the conditions of application of the laws of fluid statics of Pascal (Gravitational Hydrostatic pressure GHSP) no longer apply and the pressures related to it are no longer modified regardless of the posture. Thus, the blood of the astronauts remains only subjected to the mechanical forces of the pumps of their circulatory system and the air pressure.

On the other hand, the drop in atmospheric pressure between the sea and the mountains or in an airliner, ***decreases the extra-venous pressure EVP sufficiently to significantly increase the transmural pressure.***

The sensation of weightlessness when our body floats in water is not of the same type as that of gravitational weightlessness. Contrary to what we have seen in a descending elevator, our weight (mg) remains the same in and out of the water. We float because "Anybody immersed in water receives from the water a thrust from bottom to top equal to the weight of the volume of water displaced (Archimedes)". Our body is pushed upwards, as when we were in our childhood, sitting on one side of the swing, we were lifted and kept in balance by our fellow of the same weight as ours, sitting on the other side.

The resistive force of the swing seat was clearly felt because it was concentrated on the small surfaceof our buttocks. On the other hand, when we were floating in the pool, we had the illusion of weightlessness. Illusion because the resistant force of the liquid was no longer concentrated on our buttocks, but it was distributed on the whole immersed surfaceof our body. A recent theory, contrary to the laws of physics, attributed antigravitational effects to immersion in water, according to a theory known as "the bags", under the pretext of the sensation of lightness of the body and the reduction in the size of varicose veins of patients with varicose veins in the pool.

<u>However, the explanation in accordance with the laws of physics is sufficient. The calibre of the varicose veins decreases not because the blood is lighter and the intravenous pressure lower, but because the pressure of the water increases the extra-venous pressure EVP, which reduces the transmural pressure TMP, thus the calibre.</u>



23-*Circulatory regimes, Bernoulli's theorem, Poiseuille's law, Reynolds number and their vascular applications.*

Circulatory regime depends on the conditions of pressure, flow, viscosity, calibre, and regularity of the veins.

Ideally laminar in physiological conditions, the regime becomes turbulent and pathogenic in the conditions of stenosis, arteriovenous fistula and veno-venous shunts, where the friction is important because of the viscosity at high speeds. The Bernoulli equation does not apply alone because the total pressure is no more constant along the circuit. Then we use the generalized Bernoulli theorem, including the parameters responsible for the pressure drop described in the Poiseuille equation and the Reynolds number that we will explain below with the resistances.

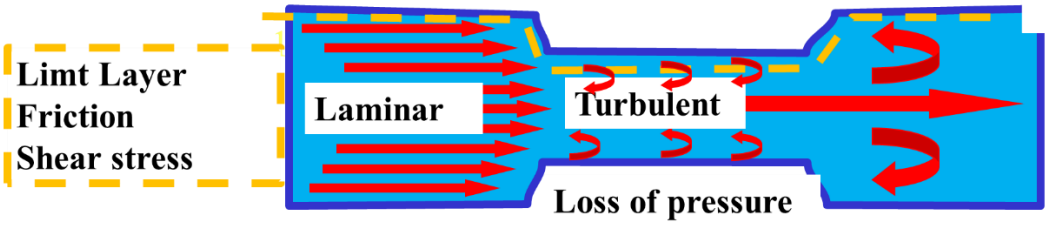
The Navier Stokes equations would allow a more precise description, but it is almost impossible to measure precisely all the hemodynamic parameters that would be necessary.

All these laws must be retained as tools of an indispensable, although approximate, model to describe and understand sufficiently the hemodynamic of the venous system, an indispensable condition for a better management of the disease.

231- *Circulatory regimes.*

Reynolds number (Re) varies with the velocity V, the gauge L and the kinematic viscosity. Re = VL/ .

When Re > 2500, the regime becomes turbulent .



Laminar flows.

The laminar flow of a fluid in a blood vessel is the mode of flow of concentric blades of blood, in the same Parallel direction, with a front of maximum velocity in the centre that reduces regularly to the walls. The blood being not Newtonian, its kinematic viscosity allows this regime only for low speeds for which we can apply Bernoulli's theorem. Beyond these speeds, the regime becomes turbulent (Reynolds) and the energetic charge of pressure is Partially dissipated (Poiseuille). The blades of blood in contact with the walls make the boundary layer where shear stress, friction, and transitions from laminar flow to turbulent flow.

Turbulent flows.

Turbulence is a vortex that appears in the bloodstream when the velocity increases until the Reynolds number reaches a value of 2000-3000. Their size, location and orientation vary constantly. They cause the wall to vibrate by as many shocks and stresses which participate in varicogenesis, and which can sometimes be heard with the stethoscope as a murmur (noise) as in arterial stenosis.

***In laminar flow, the pressure drop is proportional to the flow rate, it becomes proportional to the square of the flow rate when the flow is turbulent**. This indicates a high loss of load in caloric energy but especially mechanical energy against the walls which Participates in varicogenesis.*

This may explain why varicose veins progressively dilated by aggressive turbulent flow remain stable for many years when increasing the size, without changing the volume of the flow, reduces the velocity below the Reynolds number, which removes the aggressive constraints of turbulence.

Boundary (limit) layer

The boundary layer in vessels is the interface zone between the wall and the moving blood. It is due to the viscosity of the blood.

It is the place where we find the highest shear stress, friction, and laminar-turbulent transition of the flow.

Shear stress and friction.

The shear stress τ = F/A *is the* ***applied force F per unit area A*** *(τ expressed in Pascals because it has the dimension of a pressure) that mobilizes the boidart layer) of a fluid tangentially to another blade or wall (boundary layer) in addition to the forces that apply perpendicularly to it. The speed and the deformation of the resulting blade depend on its viscosity. It Predominates at the boundary layer, i.e., in contact with the walls. This tangential friction tends to tear away the intima like water erodes the edge of a river, just as it increases its effects when turbulence occurs.*

In addition to their mechanical effects, these constraints trigger chemical, neuro-hormonal and structural reactions of the walls, notably in varicogenesis.

232- *Bernoulli's Theorem*

Bernoulli's equation helps to understand and correct the energies, gravitational hydrostatics, static and dynamic produced by universal gravity and venous pumps.

Knowing how to identify the pressures that dilate the veins, that drain the blood, that cause varicose veins and ulcers, allows to apply a rational treatment.

According to the law of conservation, energy does not disappear but is transformed (Lavoisier). Thus, the potential energy Pe is transformed into kinetic energy and vice versa E= pe + ce. It is the same for its expressions of static and dynamic pressures.

Pascal's law only concerned fluids in equilibrium. In 1643 Torricelli established that the square of the velocity v^2 of a fluid flowing under the effect of gravity g is proportional to the height h of the fluid above the bore. $v^2 = 2gh$. If we multiply the denominators by the specific mass ρ of the fluid, we obtain $\rho v^2=2\rho gh$ or $1/2\rho v^2=\rho gh$. ρgh is the potential energy of still water converted into $1/2\rho v^2$ energy by flowing.

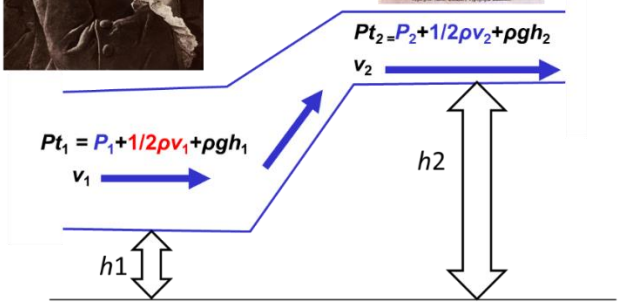
91 years later, Daniel Bernoulli" extended it to fluids in motion with the formulation of the theorem that bears his name. ***Ref: "****Hydrodynamica, sive de Viribus et Motibus Fluidorum commentarii. Opus Academicum... Strasbourg Dulsecker, 1738*

Total pressure Pt = p + ½ρv² + ρgh shows that a Newtonian fluid (perfect, with constant viscosity behaviour) does not lose total pressure Pt throughout a circuit because its components convert into each other.

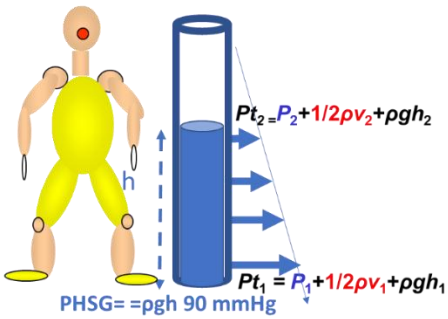
p = density Kg/m3 v = velocity m/second, g = gravity of the earth 9.81 m/s. h = vertical drop of the pipe in meters m.

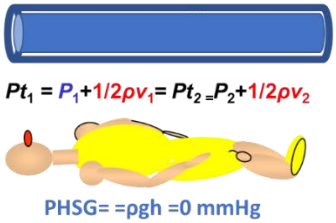
P= static pressure energy in Pascals, $\frac{1}{2}\rho v^2$ = kinetic pressure energy, ρgh = potential energy.

It applies to Newtonian fluids i.e., of linear viscosity. The viscosity of blood does not perfectly fulfill these conditions, so it is considered non-Newtonian. However, it is commonly accepted that it remains applicable to blood under the conditions of circulatory regime and low physiological



Total pressure Pt : Pt1 = Pt2
P1+1/2ρv1+ρgh1=P2+1/2ρv2+ρgh2= Cte
p = static pressure is the volume density of energy due to the work of pressure forces
½ρv² = dynamic pressure ρ = density (weight/volume)/g also called mass density
v= velocity
ρgh = hydrostatic gravitational pressure PHSG. is the volume density of potential energy of gravity.
Pm = driving pressure (charge) = p+½ρv² .



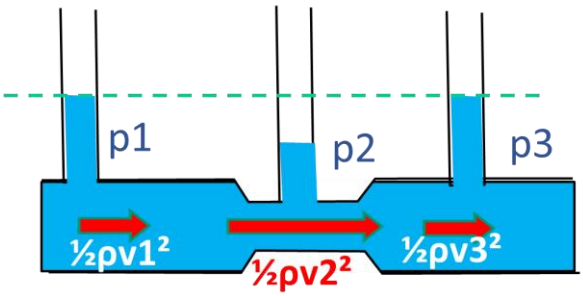


When velocity v increases (v2) increases and lateral static pressure p decreases (p2)
ρgh = gravitational hydrostatic pressure is negligible when the duct is horizontal BUT becomes largely dominant in the upright position ρgh = 90 mmHg vs p + ½ρv² = 10 mmHg

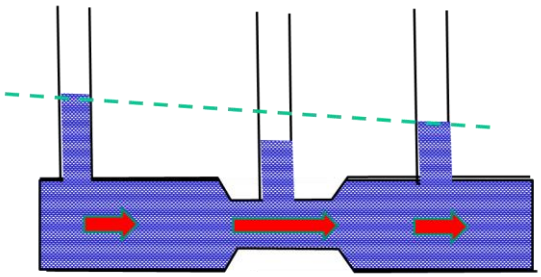
Pt1= Pt2=p1 + ½ρv²1 + ρgh1 = p2 + ½ρv²2 + ρgh2.

In the previous figure, we see that at 2 distant points 1 and 2 of different gauge and height h, h1 and h2, Pt1= Pt2. h increases in h2 by h2-h1. At Pt2, the static pressure energy p1 is found in part as potential energy proportional to the vertical drop ρgh1-ρgh2. The increase in velocity v2 -v1 increases the kinetic energy (1/2ρv²2) -(1/2ρv²2), which reduces the static pressure energy accordingly.

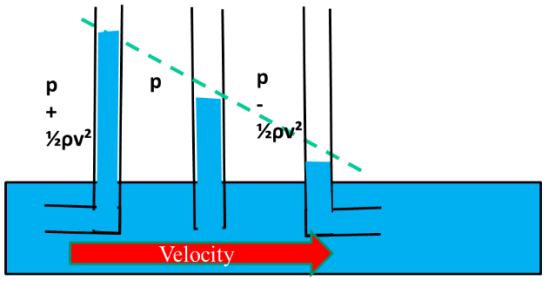
Measuring the pressure with a manometer, does not give the value of Pt, but p2 + 1/2ρv²2 and p1 + 1/2ρv²1 which we can call the load or driving pressure PM. Here, ρgh is the potential energy related to the height, that is expressed in part of the pressure P1 and P2.



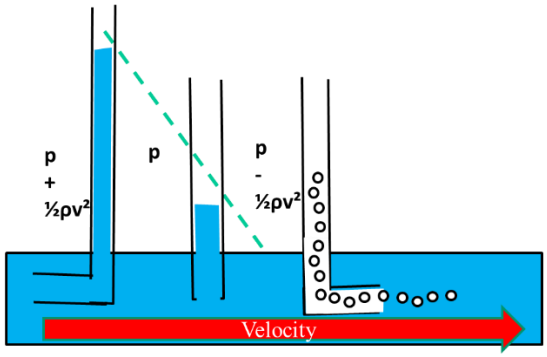
Bernoulli : Newtonian fuid
Total pressure Pt = p + ½ρv² + ρgh = Cte
When the velocity v increases (v2) the dynamic pressure ½ρv² increases and the lateral static pressure p decreases (p2)
ρgh = hydrostatic pressure negligible here because the duct is horizontal BUT becomes largely dominant in the standing position ρgh = 90 mmHg vs p + ½ρv² = 10 mmHg



Non-Newtonian fluid
The viscosity of blood reduces the total pressure by progressive pressure drop along the vessels. However, the Bernouilli equation is considered applicable to hemodynamics by approximation



Bernoulli and Pitot tubes
Total Pressure Pt = p + ½ρv² + ρgh
When the catheter faces the flow, it measures the Total Pressure p + ½ρv²
When it is perpendicular to the flow, it measures the only lateral static pressure p.
When it is in the direction of the flow, the pressure is equal to p- ½ρv²
This is to be taken into account when measuring venous pressure with a catheter and also to understand the behavior of the flow in the perforators, depending on their orientation with respect to the veins to which they are connected.



Bernoulli ,Pitot tubes and Venturi effect
Total pressure Pt = p + ½ρv² + ρgh
When the velocity v (v2) is very high, ½ρv² is > p, which draws the outside fluid (blood or air) into the vessel.
This pipe may be a vein with flow velocity v that draws blood from a collateral as sometimes during systoles of valvular-muscular pumps.
This phenomenon can also combine with the depression of the veins of the blow to cause gas embolisms.

Total pressure Pt = p + ½ρv² + ρgh and conservation of energy p1 + ½ρv²1 + ρgh1 = p2 + ½ρv²2 + ρgh2

½ρv² = dynamic pressure PD (kinetic energy) is the kinetic energy density (kinetic energy per unit volume, m being the mass of the volume V of fluid

(ρ (ro) = Mass density and v velocity),

p = static pressure is the volume density of energy due to the work of pressure forces

ρgh = hydrostatic gravitational pressure GHSP is the volume density of potential energy of gravity.

ρ = density (weight/volume)/g also called mass density.

Pm = driving pressure (load) = p+½ρv².

In fluid mechanics, pressure drop is the dissipation, through friction, of the mechanical energy of a moving fluid. Most often, the term pressure drop is used to quantify the pressure loss within a pipe generated by the friction of the fluid on it.

As the liquid flows up and down, under the sole force exerted at a distance by gravity, ½ρv² represents the portion of the liquid's potential GHSP transformed into kinetic energy (dynamic pressure) and p portion of ½ρv² transformed back into potential energy (static pressure p). As the liquid flows, the gravitational potential energy ρgh changes only if the liquid column changes in height h

If we add a pump contact force, such as my calf valvulo-muscular pump, we must increase the Pm = p+½ρv² = Pump power (heart, muscle...)/ flow rate by that much.

*Recall that the principle says that for the same energy of Pm, the sum of dynamic pressure energies DP and static is constant, because when one decreases, the other increases by the same amount (principle of conservation of energy**).***

This law finds its practical clinical and diagnostic applications. (See figure above)

*The static pressure p and the Hydrostatic Gravitational pressure GHSP ρgh are exerted in all directions, including against the walls. The dynamic pressure ½ρv² is that part of the total pressure exerted only in the direction of flow when the regime is laminar, and in whole or in part against the walls when the regime is turbulent, and Bernoulli's theorem is no longer applicable**.***

<u>These measures have a practical application in venous pathology</u>

In laminar (non-turbulent) regime (if the velocity remains low).

In a regular vertical tube such as a great saphenous vein at rest with a height h2, standing, valves open, supplied at constant pressure and flow by the Residual Capillary pressure.

If h1 = 0 at the ankle, p1 + ½ρv²1 = p2 + ½ρv²2 + ρgh2. p2 + ½ρv²2 is given by the Residual pressure and ρgh2 by the U-tube effect that is imparted by the supplying arterial pressure column.

If v = 0 due to an obstacle, p1=p2 + ρgh2

If the direction of the velocity v2 reverses (backflow) without changing its value, v1 and p1 do not change.

If the velocity v2 reverses (reflux) and increases (decrease of the resistant forces by suction by the diastole of the calf pump), ½ρv²2 increases, p1 decreases. <u>The end of diastole abruptly converts ½ρv²2 into an additional pressure shock p at the re-entrant perforator.</u>

This model must be tempered for 2 reasons.

Because of the large calibre of the veins, blood velocities are sufficiently low, to keep the flow laminar. <u>These velocities are also too low, including during exercise, to reduce very significantly the lateral static pressure and suck in the tributaries significantly by the Venturi effect</u>*. If we assume that the systolic flow of the calf pump in the popliteal vein of diameter = 0.01 m can reach a peak velocity of O.6ms², the lateral static pressure is reduced by 2.6 mm Hg or 3.6 cm H²O for a peak pressure of 90 mm Hg or 120 cm H²O.*

The relative slowness of the speeds modifies little the measurement of the pressures according to the orientation of the intravenous sensor in the subject at rest where the highest speeds are of the order of 10cm/s², which can reduce the static pressure of 100 Pascals, that is to say 1cm H²O or 0,74mmHg, that is to say approximately the 29th of a usual venous pressure which does not exceed 10 to 20 mm Hg in supine position.

<u>Beyond these "physiological" speeds, Bernoulli's theorem is no longer applicable. It is replaced by Poiseuille's Law and the Reynolds Number</u>

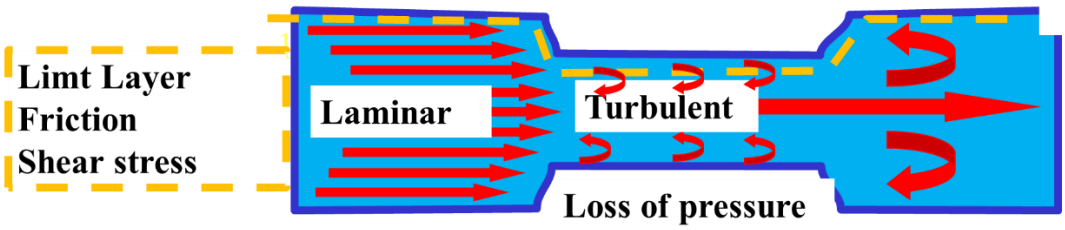
233. Poiseuille law and Reynolds number

Bernoulli's theorem is no longer applicable because of <u>the non-Newtonian character of blood</u> when velocities are too high and/or calibres are too low, especially in the overloads in flow / pressure of superficial veins are arteriovenous and veno-venous shunts and in venous stenosis.

2331-Reynolds number and turbulence;

Reynolds number (Re) varies with the velocity V, the gauge L and the kinematic viscosity. Re = VL/ .

When Re > 2500, the regime becomes turbulent .



The viscosity is responsible for turbulent regimes when the conditions of velocity and size are met. Turbulence occurs when the <u>Reynolds Number Re, a dimensionless number</u>, due to Osborne Reynolds 1883, is reached under specific conditions of velocity, size, and viscosity of the fluid. Re=VL. V = speed, L = size and kinematic viscosity. It is about 2500 in humans.

We must also add the effect of parietal irregularities that deform the boundary layer.

Physiopathological and clinical effects.

These turbulences redistribute all or part of the hemodynamic load against the walls. They increase the aggression (shear stress), dilate, and deform the walls, which they stimulate biological reactions such as the secretion of chemical factors and modifications of the histological structures.

It is understandable that the conditions of overloading of the veno-venous shunts during ***walking aggravate, or even are the almost exclusive conditions for the development of varicose veins****. When velocities decrease due to the increase in calibre for which they are responsible, Reynolds number is reduced below 2500 and the regime becomes laminar again, and dilation stops progressing. This is seen in* ***patients whose varicose veins remain stable*** *in size for years. It should be noted that* ***the elimination of the pressure flow overload (disconnection of the shunts responsible) leaves a physiological pressure flow which leads to a progressive remodelling which results in a normal calibre adapted to normal pressure/flow****.*

2332- *Poiseuille's law and pressure loss (resistances and stenoses)*

Viscosity also leads to resistances and load losses, especially in stenoses depending on the calibre and extent according to Poiseuille's law (Jean-Léonard-Marie Poiseuille, 1797 - 1869). *His law also requires a Newtonian liquid, which is to say of linear viscosity, but it remains a good approximation in the measurements of blood flow pressure drop. It measures the pressure gradient due to the pressure drop P1-P2 ΔP of a Newtonian fluid (linear viscosity) flowing between two points 1 and 2 of a vessel as a function of its flow rate Q, its radius r, the distance L between 1 and 2 and its viscosity μ.*

P1-P2= ΔP = Q 8 L μ / π r4 = pressure load loss - P

The measurement is made with the following international units:

ΔP =P1-P2= pressure gradient= PA(Pascal)

1PA= 1/98,0638 cmH²O = 0,74/ 98,0638 mmHg

Q=flow rate: m3/s

L=length in meters

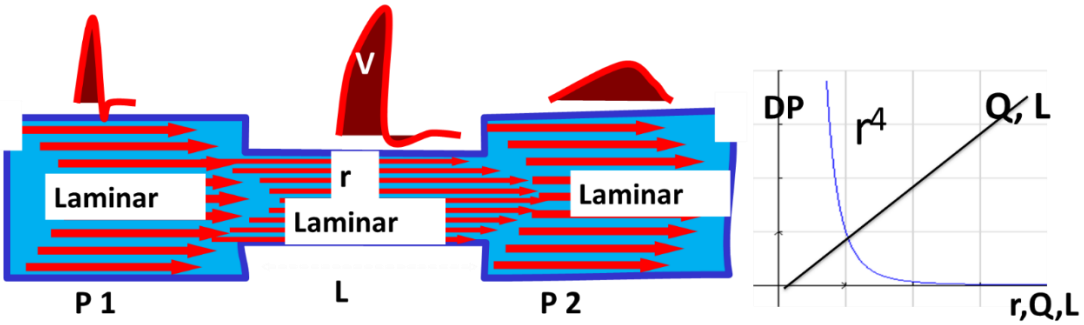
r=radius =meters

μ=Viscosity of the blood (poise)= 6.10-3

Poiseuille's law dP = P1-P2= 8 L μ Q/ π r4.

The pressure drop increases exponentially with the reduction of caliber of radius r :1/ r4.

Allows to evaluate the ideal stent size for a given flow rate Q and length L



23321- *Loss of pressure and hemodynamically significant stenosis.*

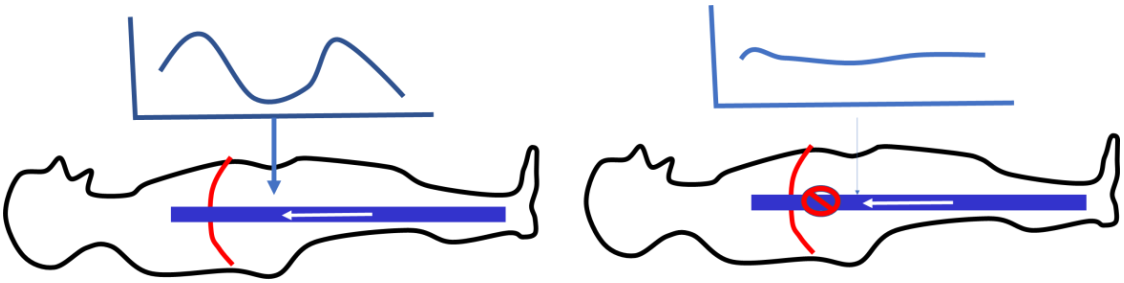
Physiologically, the shear stress opposes resistance to the normal flow which progressively lowers the downstream pressure, but in physiologically negligible *proportion because the viscosity and velocity are normally low enough to maintain a Reynolds number below 2000.*

Hemodynamically significant is not defined by geometry (stenosis, dilation) but by hemodynamic changes as turbulences, pressure drop (loss of charge), damping flow modulation).

Hemodynamically significant stenosis occurs when the pressure drop causes a pressure gradient PG (pressure difference ΔP between 2 points separated by a distance D (PG= ΔP/D)

In addition to quantitative pressure measurement, stenoses can be assessed by Doppler velocimetry. Indeed, the degree of demodulation (loss of amplitude) of the velocity of a periodic flow is proportional to the significance of the stenoses. This is because, still in accordance with Poiseuille's law, the resistance reduces more the velocity as the flow is higher. ***The decrease in maximum velocities is explained by the fact that the resistance increases with velocity.*** *This is not only the case for arteries, but also for veins* ***when iliac and/or ilio-caval obstructions reduce the modulation of breath-timed velocities****, as measured with femoral vein Doppler in the recumbent patient.*

This is also the reason why a not significant at rest stenosis can become significant when the flow is increased by effort. Justifies a Doppler measurement of the femoral veins in the supine position at rest and immediately after a walking effort (or free pedalling movement in the supine position**).**



Iliocaval obstruction: Loss of respiratory modulation of femoral flow on Doppler

23322- *Effects of significant stenoses on veins and upstream drainage.*

In addition to the loss of pressure, the significance of a stenosis for venous hemodynamic takes on its full venous meaning when considered as a resistance to upstream flow

233221-*Increase in residual pressure RP provided by the microcirculation and/or the systolic pressures of the valvulomuscular pumps.*

Contrary to arterial stenosis, which is serious because of the reduction in downstream pressure (ischemia), ***venous stenosis is serious because of the increase in upstream pressure (including the Transmural pressure (TMP) via the increase of Residual Pressure RP.***

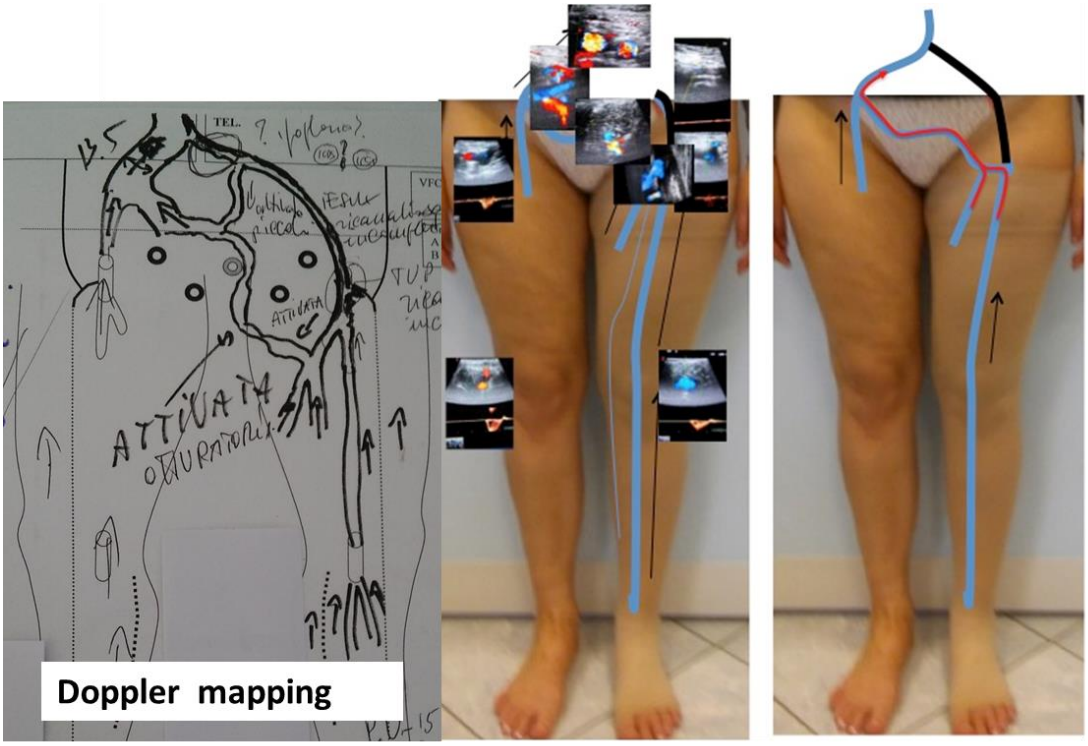
233222- *Collaterals and resistance.*

The upstream hemodynamic impact of occlusions and stenoses is reduced in proportion to the compensatory collateral veins (open vicarious shunts OVS) which reduces the global resistance by opening up parallel resistances.

This compensation can be accelerated by walking under strong compression, which increases residual pressure and forces the opening and dilation of compensatory collaterals.

If this evolution is not sufficient to correct the functional clinical signs of venous insufficiency, or if the compensatory varicose veins are not accepted for aesthetic reasons,

dilatation and stenting of these stenoses can be performed.



Compensated Iliac Vein Occlusion: clinical and hemodynamic: Doppler Left Posterior Tibial Vein Pressure = 20 mmHg

233223- *Measurement of upstream pressures: invasive and non-invasive Doppler.*

The measurement of upstream pressures is the only criterion that allows us to confirm the significance of a venous obstacle at rest and during exercise.

<u>However, due to lack of pressure measurement, too often unnecessary revascularization procedures are performed although the pressure is not severely impacted.</u>

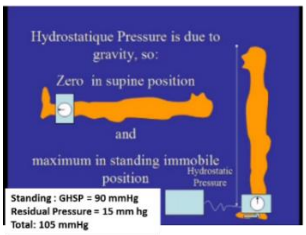
Invasive measurement by catheter is well known, ***but <u>measurement by Doppler effect is sadly unknown.</u>***

However, it is non-invasive and "physically" rational as measuring blood pressure at the same level! <u>It must be performed in decubitus position</u>*, in order not to integrate the Hydrostatic Gravitational pressure GHSP which is not involved in the obstacles. In this position the GHSP is negligible, which makes it possible to* ***selectively evaluate the excess pressures due to the obstacles, without considering the effects of valvular incompetence on the pressure. Indeed, valvular incompetence doesn't increase the venous pressure int that position.***

This is mandatory to ***avoid recanalizing an obstacle well compensated,*** *but to which one falsely attributes the cause of a venous insufficiency when it is* ***causedonly by the associated***

valvular incompetence. It is however the case in the badly hemodynamic documented treatment of the post thrombotic disease and the venous malformations.

One can also, as we have seen, assess, though not quantifying it, <u>the hemodynamic significance of ilio-caval obstacles by the respiratory demodulation of Doppler flows</u> at rest and under effort (pedalling lying down supine with legs in the air) of the femoral veins.



233224- Pseudo-stenosis: Illusory Pseudo May Thurner

May Thurner Syndrome MTS and Nutcracker syndrome NTS

May Thurner syndrome consists of a permanent stenosis of the left iliac vein, clamped between the right common iliac artery and the lumbar rachis.

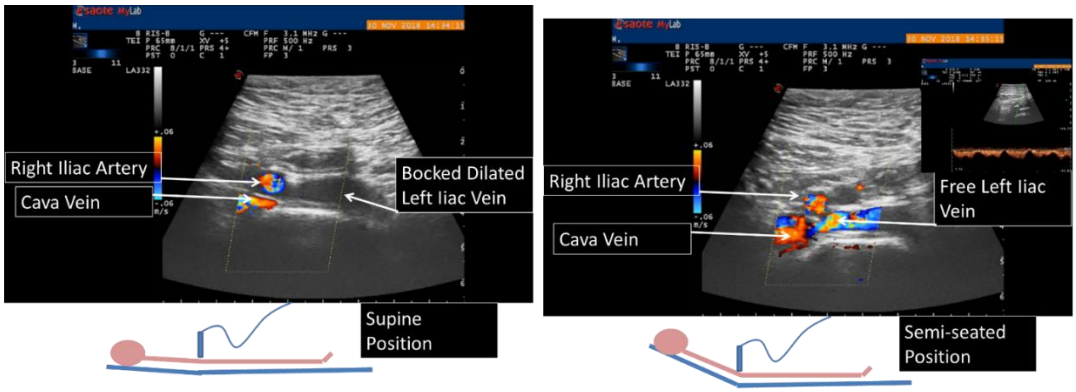
However, <u>in most cases,</u> ***<u>this stenosis is not always permanent but only occasional and in supine postures</u>*** *<u>that are not very frequent in real life</u>****.*** *This is the case of* ***the illusory pseudo-May Thurner syndrome which shows a notable stenosis of the left iliac vein, but only in the strict supine position required by phlebography and MRI techniques.*** *Indeed, this* ***postural artifact disappears*** *as soon as the subject is* ***in a semi seated position as I have shown with the echodoppler.***

Ref: Paolo Zamboni, Claude Franceschi, Roberto Del Frate.The overtreatment of illusory May Thurner syndrome Veins and Lymphatics 2019; volume 8:8020

This may explain the finding of "illusory" MTS assessed by horizontal supine phlebography in young asymptomatic subjects.

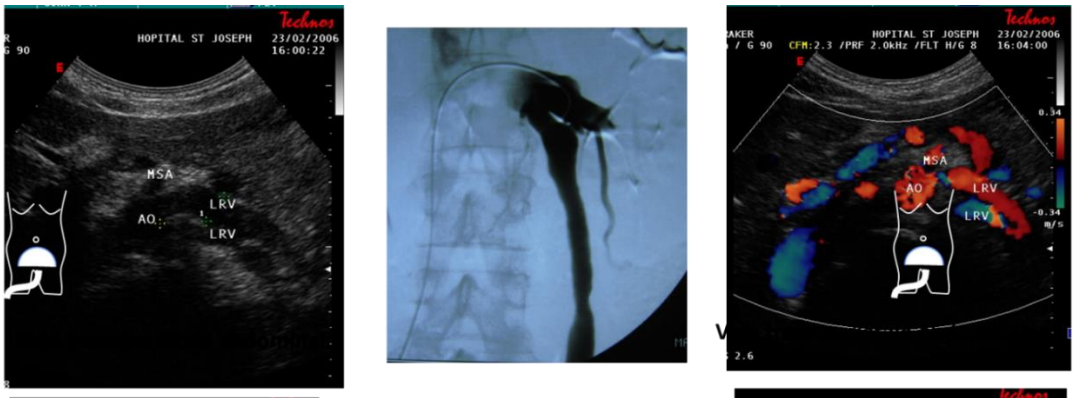
Ref: van Vuuren TM, Kurstjens RLM,Wittens CHA, et al. lllusory angiographic signs of significant lliac vein compression in healthy volunteers. Eur.J Vasc Endovasc Surg 2018;56:874-9. video: Pseudo MTS : https://www.youtube.com/watch?v=h931XXo2hdk&t=23s

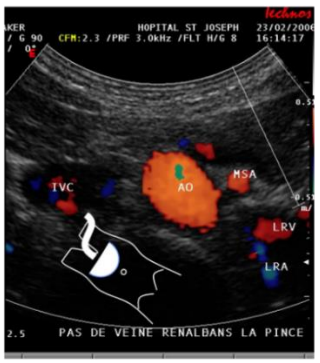
Similarly, the <u>Nutcracker syndrome NTS may be artefactual</u> in the presence of a varicocele considered to be compensatory for a stenosis of the left renal vein. The <u>disappearance of the reflux of the ovarian vein in the Trendelenburg supine position</u> (head lower than the feet) on the echodoppler proves its absence of compensatory effect, whereas its permanence confirms it.



May Thurner Syndrome at phlebography but
Pseudo May Thurner Syndrome on Echodoppler: only postural

This knowledge should reduce the still too large number of unnecessary stentings of the left iliac and renal veins.

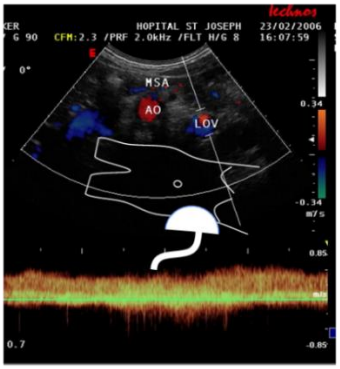




Trans-abdominal scan in Trendelenburg position.

Aorto-Mesenteric clip.
Venous flow = 0

No reno-azygo-lombar compensation
PERMANENT left ovaric vein reflux in Trendelenburg position (head lower than the feet).



Trans-abdominal scan in Trendelenburg position.

Nutcracker Syndrome: Total Aorto-Mesenteric clip
Single bypass through the left ovarian vein

233225- *Stents and Recanalization*

Poiseuille's law can be used to evaluate the calibre r and length L for a flow rate Q to provide the size of recanalization, stent, or by-pass. As the flow is not easy to assess in the iliac vein, I suggest to measure the flow rate of the common iliac artery which supplies it.

$\Delta P = 8\ QL\ \mu / \pi\ r^4$ = pressure Drop. The detailed measurement is in the treatment chapter8.

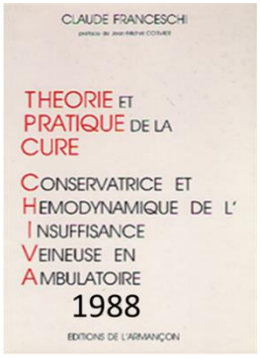
233226- Downstream Pressure and guyotan equation.

The downstream hemodynamic impact of venous stenosis has an effect when it significantly reduces the flow/pressure, necessary to the reservoir effect on the right ventricle preload. This is the case of obstacles to the vena cava (ligation, laparoscopic surgery, compression by the pregnant uterus when the pregnant woman is supine).

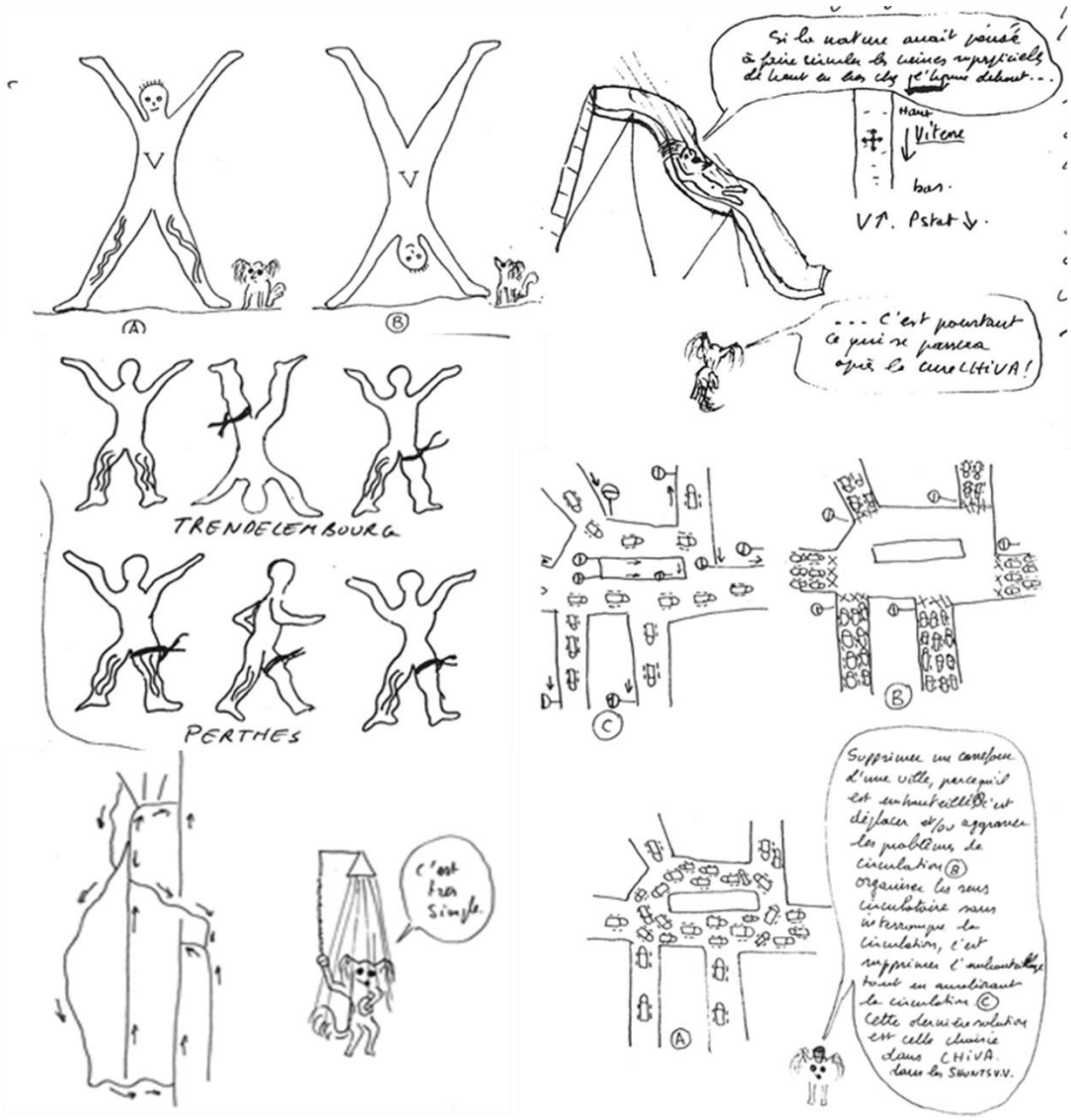
The Guyot equation is intended for resuscitators and anaesthesiologists to avoid cardiac complications of "too full and too empty" of the cava vein. ***The argument that the vena cava may be "too empty" to justify the removal of chronic occlusion loses its meaning if one understands that the deficit in direct venous blood supply to the lower limb is compensated by the collaterals and represents only 150 to 200 ml/ minute compared to the 5000 ml of cardiac output.***

For the same reason, justifying the ablation of an associated incompetent great saphenous vein to increase the iliac flow/pressure is not relevant.

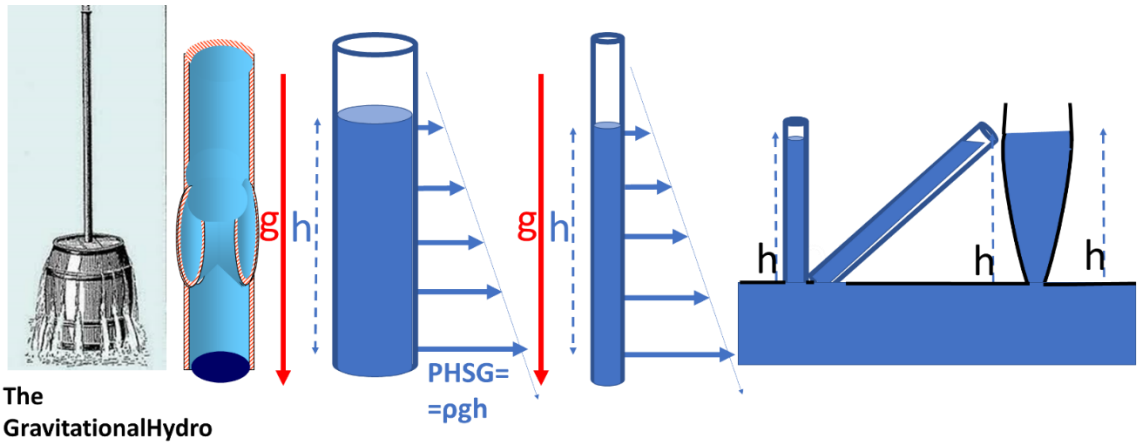
However, varicose veins may disable the reservoir effect when they move down a great blood volume as when standing up.



The order and disorder of venous circulation is a matter of clinically evident fluid statics and dynamics. Understanding this informs diagnosis and treatment



24- *The Gravitational Hydrostatic pressure GHSP*



The GravitationalHydrostatic Pressure GHSP= =ρgh depends only on the height of the pipe whatever the gauge and "bursts" the barrel.

GravitationalHydrostatic Pressure GHSP = =ρgh whatever the vein size

GHSPρgh depends only on the height h independently of the shape , size and volume

The gravitational hydrostatic pressure GHSP will refer to the hydrostatic pressure HSP*, because HSP is often confused with the static pressure p which can also be supplied by other energies than gravity such as pumps.*

It applies to blood of density ρ (ro) subjected to the force of gravity g= 9.8 m/s, from a height h. GHSP = ρgh.

It does not depend on the volume but only on the height of the liquid above the point of measurement. (Pascal's principle*). This was demonstrated by the experiment of the barrel burst (Torricelli, Pascal). The barrel was filled with water through a vertical pipe of small diameter, 10 meters high. It burst when the water height reached the 10 meters, so a pressure= 10kg/cm². **Changing the pipe calibre does not change the experiment.** The pressure increases with h even though the weight of the volume of water in the barrel + pipe remains the same. **Gravitational forces and pressures are distributed differently in liquids than in solids** according to laws discovered by Stevin, Torricelli, and Pascal. In particular, the Gravitational Hydrostatic pressure presents characteristics that explain the **counterintuitive facts in phlebology. Indeed, one can think intuitively, but against physics, that the gravitational hydrostatic pressure in a vein increases with its calibre.***

As we have seen, this pressure only increases with the height of the column of blood overlying the vein, regardless of its calibre! Therefore the pathogenic character of a vein should not be judged by its calibre, because a small-calibre vein will have the same effect as a large one if the height of the blood column is identical.

*We will see later that **the gravitational hydrostatic pressure when standing still decreases considerably during walking** thanks to the **closure of the valves which divides the height of the blood column h** as one **would interpose shelves between piles of books to reduce the***

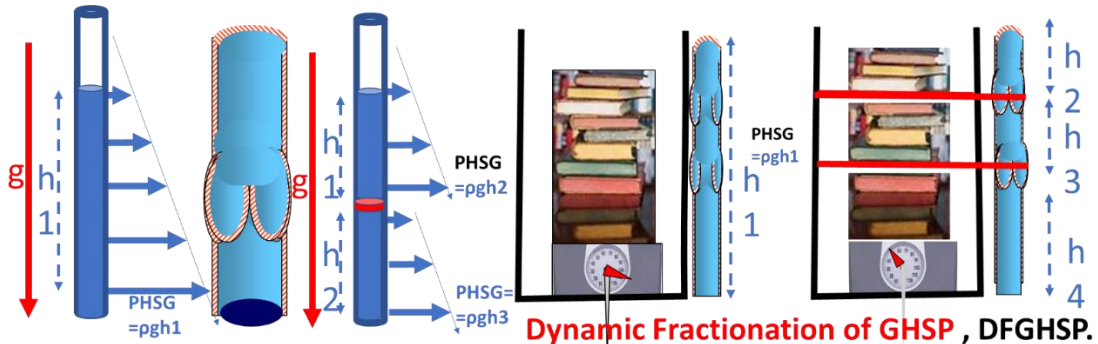
weight *supported by the lowest shelf****. We will also see the hemodynamic consequences of the non-closure of these valves.***

*When this column is too high, it is therefore rational to fraction it, whatever its calibre****.***

The calibre only acts on the pressure when the blood is set in motion. *By decreasing, it increases the pressure drop by friction and viscosity, which reduces the dynamic pressure without changing the gravitational hydrostatic pressure. But the parietal constraints by the load of the flow of the shunts, dilate progressively the vein which appreciably reduces the loss of load and ends up stabilizing the calibre of the varicose veins.* ***In fact, it is mainly the flow/pressure load of the shunt flows much more than the gravitational hydrostatic pressure that "makes the varices". For this reason, the reduction (not the loss) of the load of the shunts by disconnection of the shunts is rational and reduces "naturally" the calibre.*** *Note that the load is the whole of the energies which take part in the hydrodynamic state of a fluid as much as the dynamic pressure without changing the hydrostatic gravitational pressure****.***

25- Dynamic Fractionation of the Hydrostatic Gravitational pressure.

Ref: 1)-Franceschi C. Théorie et pratique de la cure conservatrice de l'insuffisance veineuse en ambulatoire. Precy-sous-Thil. France:Editions de l'Armançon, 1988.2-C Franceschi, M Cappelli, JM Escribano, E Mendoza - Dynamic Fractionation of Gravitational Hydrostatic pressure. Journal of Theoretical and Applied Vascular Research (Page 1) - JTAVR 2020;5(2) - DOI: 10.24019/jtavr.100 Corresponding author: Dr. Claude Franceschi, claude.franceschi@gmail.com



Gravitational Hydrostatic Pressure PHSG= =ρgh reduced because the height is split and GHSP 1 and GHSP 2 by closure of the valve or ligation

Dynamic Fractionation of GHSP , DFGHSP. Diastolic valve closure splits PHSG ρgh1 (open valves) into ρgh 2,3, and 4 like shelves split the weight of books

The Gravitational Hydrostatic pressure GHSP (ρgh: ρ= mass volume, g=Gravity acceleration, h=height) depends on posture and therefore on h, which can vary at the ankle from 0 in the supine position to 90 mmHg in the immobile standing position because

the valves are held open by the flow that, pushed by the residual pressure, comes from the microcirculation.

Arnoldi measured simultaneously by puncture the pressure in the posterior tibial, popliteal, and great saphenous veins in 10 young healthy subjects

Ref:1-Arnoldi CC: Venous pressure in the legs of healthy human subjects at rest and during muscular exercise in nearly erect position. Acta Chir Scand 1965; 130:570-583).2-Bjordal R. Simultaneous pressure and flow recordings in varicose veins of the lower extremity Acta Chir Scand 1970;136:309-317 16-

At rest standing.

-posterior tibial vein = 83 mm Hg

-popliteal vein = 61 mm Hg

-great saphenous vein 83 mm Hg

These values correspond to the unfractionated hydrostatic pressure column when the valves remain open.

During calf contraction, the pressure increased on average by:

- *75 mm Hg in the posterior tibial vein in addition to the 83 mm Hg at rest,*

- 29 mm Hg in the popliteal vein in addition to the 61 mmHg at rest, and 34 mmHg in the great saphenous vein in addition to the 83 mm Hg at rest.

The ejected volume varies according to the muscle mass and the volume of the flushing chambers of the calf of each individual. On average, it is around 70 ml.

<u>But, during walking the pressure of the posterior tibial and great saphenous veins goes from 90 mmHg to 38 mmHg +-6 mmHg.</u>

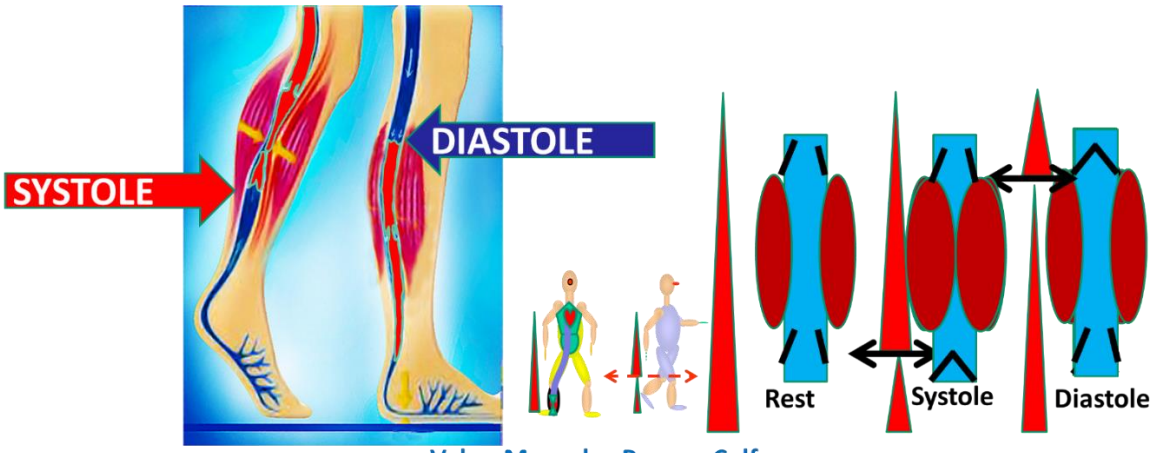
*This distal venous pressure of 90 mmHg maintains a Transmural pressure TMP unfavourable to tissue drainage****. <u>Thus, the prolonged immobile standing position is the cause of chronic insufficiency called "physiological</u>»*** *because it causes trophic disorders while the venous system is normal. In the course of time, stasis in the valves can degrade the venous system by destroying the valves.*

<u>We understand that to avoid this evolution, it will be sufficient to reduce the Transmural pressure TMP in 2 ways</u>: either by avoiding standing still for a long time (or even sitting still) while reducing the Intravenous pressure by walking, or by increasing the extra venous pressure by compresssion.

On the other hand, walking is much less effective when the valves are incompetent.

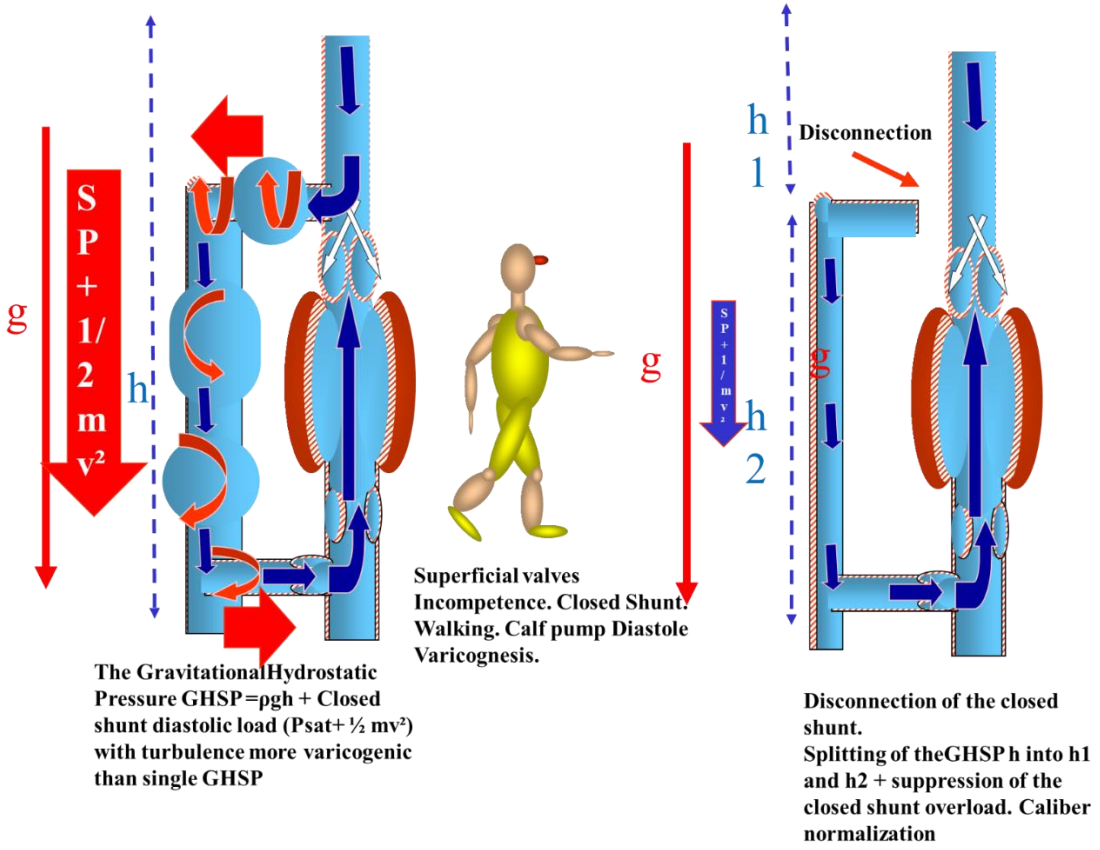
These facts led me to propose ***<u>the concept of Dynamic Fractionation of Gravitational Hydrostatic pressure (DFGHSP).</u>*** *The mechanism would be the successive and alternative closure of the calf valves, upstream during systole and downstream during diastole. This fractionation is therefore prevented by valvular incompetence.* ***By analogy, the weight of stacked books supported by a single shelf is reduced if other shelves are interposed that split the stack of books. One can imagine the effect of books falling onto the bottom shelf if the intermediate shelves on top are quickly removed****. This reflux will be more aggressive for the walls as its volume will be increased and its regime will become turbulent as it occurs in the superficial veins in closed shunts as it will be explained later.*

We will see later that this Dynamic Fractionation of Gravitational Hydrostatic pressure DFGHSP can be impaired by the incompetence of superficial veins despite good functioning of the valvulomuscular pump of the calf (competent inlet and outlet valves). *This is the frequent example of the incompetent great saphenous vein connected downstream to the femoral vein via the saphenofemoral junction and upstream to the gastrocnemius veins via a calf perforator. The diastolic non-closure of the incompetent valves impairs the effect of closure of the pump valves.*



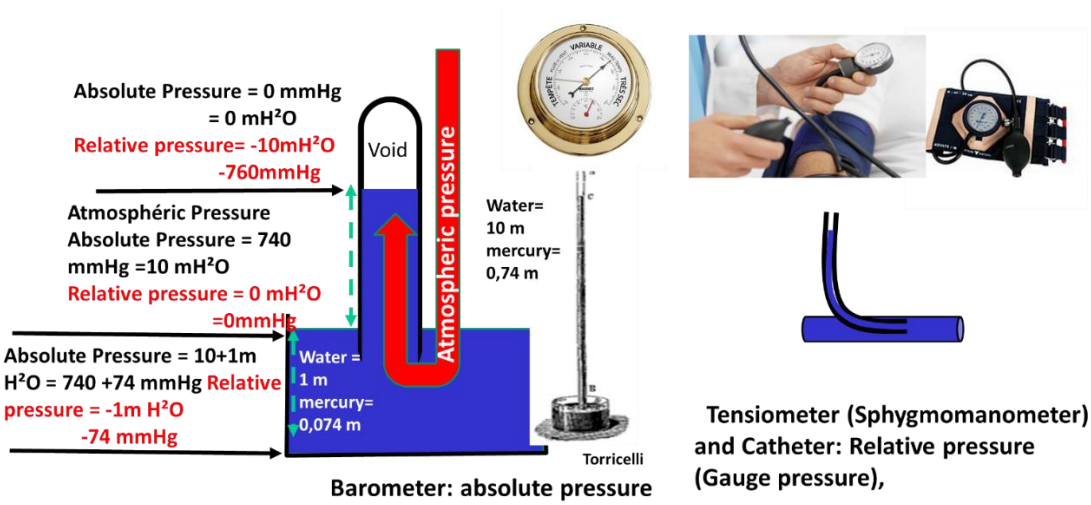
Valvo-Muscular Pump : Calf

Dynamic Fractionation of Gravitational Hydrostatic Pressure (DFGHSP):
Successive and alternate closure of the upstream and downstream valves of the muclar pumps during walking.

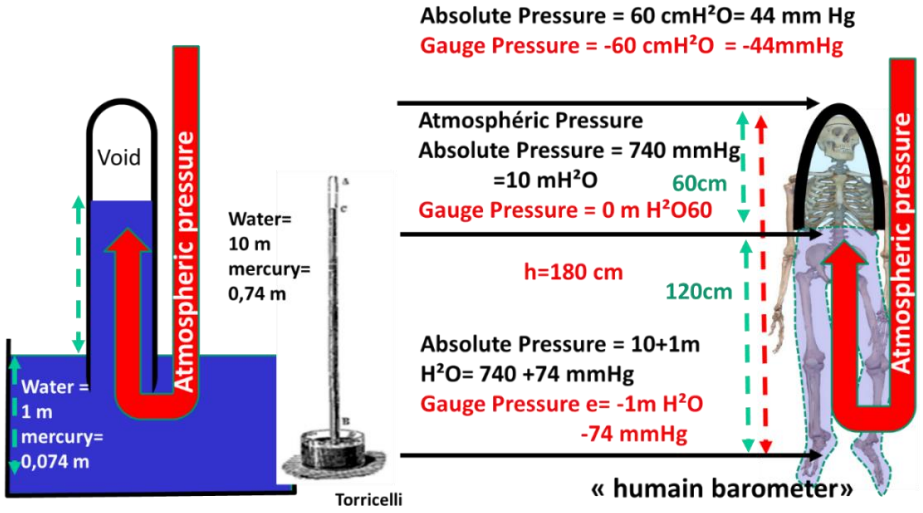


26- Paradoxical hydrostatic pressure and atmospheric pressure

Ref: C. Franceschi. Paradoxical ankle venous pressure in standing and walking compared to the venous blood column height. Journal of Theoretical and Applied Vascular Research (page 32) - JTAVR



The pressure usually measured is the relative pressure called gauge pressure = absolute pressure - atmospheric pressure.
The atmospheric pressure measured by barometers is the absolute pressure.



Barometer: absolute pressure

In humans, the negative pressures are the pressures lower than the atmospheric pressure. In the standing position, the venous pressure is negative above the diaphragm because the rigidity of the walls of the skull and thorax do not allow the direct transmission of atmospheric pressure, so the skull and thorax behave like barometers. This explains why the pressure at the ankle in standing position is equal to the ankle-diaphragm height and not the ankle-skull height.

The venous pressure at the ankle pgh in standing still (venous valves open) should be in cm H^2O, equal to the height of the top of the skull or 180 cm in a subject who is 180 cm tall. However, as it is the measured pressure is lower because it is equal to 120 cm, which corresponds to the height of the heart.

This Paradox is due to the method of measurement which does not consider the atmospheric pressure. *Let us first recall that the measurements of pressures are usually those of the so-called Relative pressure (also called Gauge pressure) which is by convention equal to the Absolute pressure AP minus the Atmospheric pressure Patm (GP=AP-Patm).* ***The devices we use to measure the blood pressure, or the tires of our car are relative pressures.*** *It gives us negative values when the pressure is lower than the atmospheric pressure. So, there is no such thing as negative pressure, but pressures that are lower than atmospheric pressure are called negative****. The negative pressure is called "vacuum pressure" and is equivalent to the absolute pressure because in absolute value, a pressure is always positive****. The devices for measuring atmospheric pressure, such as barometers, give the values of the absolute pressure and cannot give negative values.*

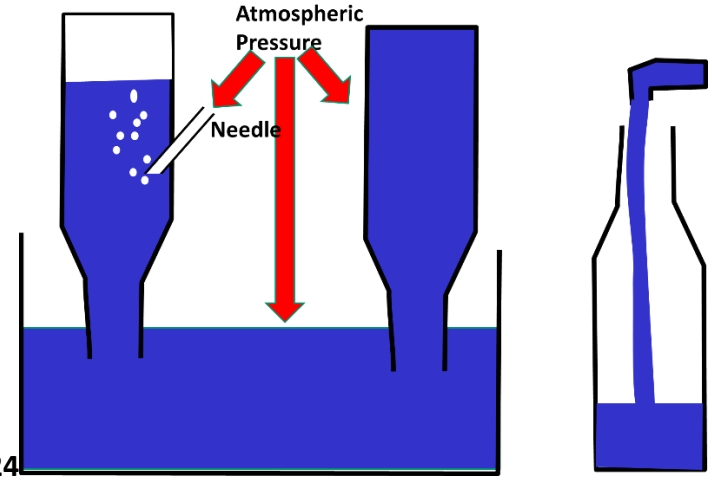
Experimentally, when we plunge a part of the open end of a long test tube previously filled with water in a basin full of water, we notice that the water of the test tube does not empty*. Indeed, the water in the test tube is retained by the atmospheric back pressure transmitted to its immersed end by the water in the basin. The relative pressure of the water in the test tube is equal to 0 at the level of the water surface where it is equal to the atmospheric pressure. It is negative above the water surface because the closed, rigid glass prevents the transmission of atmospheric pressure. If the test tube was more than 10 m long, it could be full up to 10 meters (equal height of water in equilibrium with the atmospheric pressure), but not in its higher part which would remain empty of air and water. The pressure*

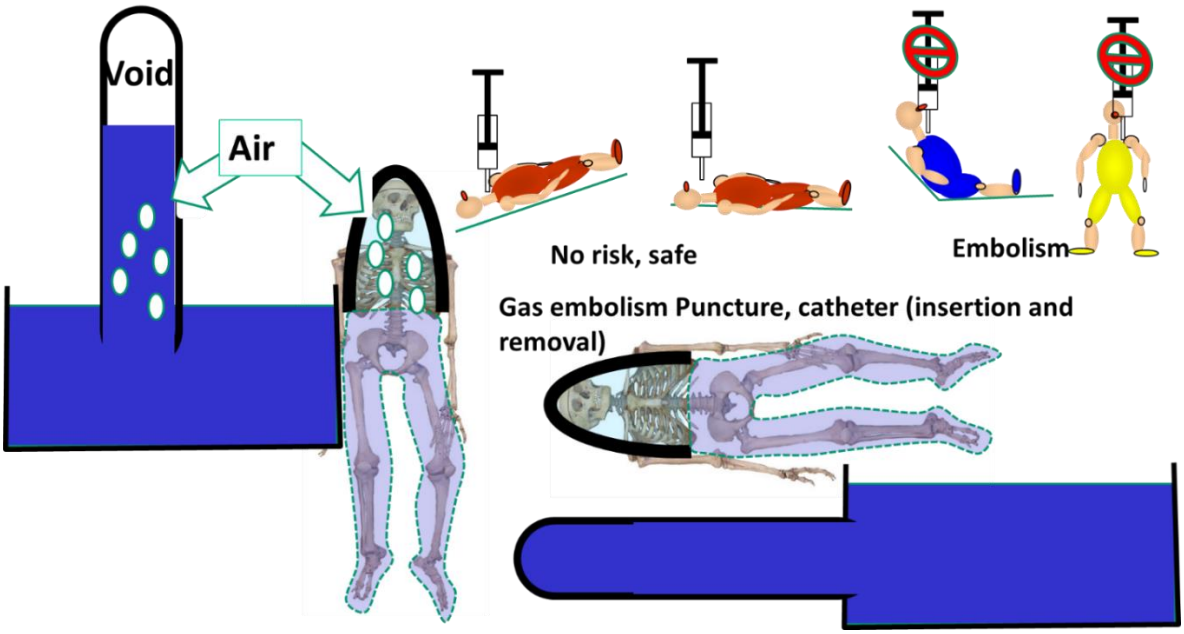
= 0 of the vacuum is the absolute pressure. It is equal to a negative relative pressure = -10 m H^2O or -740 mmHg and 0 Pascals in absolute pressure. If we pierce the closed end of the test tube, we get into contact with atmospheric pressure, which empties the water down to the surface of the water of the basin.

Thus, by analogy with the test tube model, the skull and thorax can be considered as rigid bony shields which, like the glass of the test tube, protect the veins they contain from the transmission of atmospheric pressure. By contrast, abdomen and extremities doesn't protect their veins because they are flexible and depressible. *Thus, we can understand why, in a subject measuring 180 cm standing still, the relative venous pressure is close to 0 + Residual pressure at the level of the typhoid appendix, decreases negatively towards the top of the skull to - 65 cm H^2O + Residual pressure and increases progressively to 120 cmH^2O + Residual pressure at the level of the ankles 120 cmH^2O + Residual pressure). The additional lowering of pressure during walking is due to the Dynamic Fraction of Gravitational Hydrostatic pressure DFGHSP.* ***The negative pressure above the heart explains the gas embolisms when the jugular vein is catheterized in a sitting position,*** *i.e., when the intravenous pressure is lower than atmospheric pressure, sucking in outside air. The Venturi effect is probably added when the needle is placed in the same direction as the blood flow.*

Do the test yourself. Dip the neck of a plastic bottle full of water into the water of a sink...the bottle does not empty. It empties if you pierce the side with a needle. The air is pushed in the vacuum where the relative pressure = -10mH^2O, and the absolute pressure = 0 mH^2O) by the atmospheric pressure where the relative pressure = 0, absolute pressure= 10m H^2O.

This theoretical model is illustrated by a simple video experiment. **Ref:** C Franceschi - Paradoxical ankle venous pressure in standing and walking compared to the venous blood column height. Vasculab Journal of Theoretical and Applied Vascular Research (Page 31) - JTAVR EPub Ahead of prints). ***VIDEO*** Venous pressure discrepancy https://www.youtube.com/watch?v=Udsg8hIzPu8&t=59s




Void
Air
No risk, safe
Embolism
Gas embolism Puncture, catheter (insertion and removal)

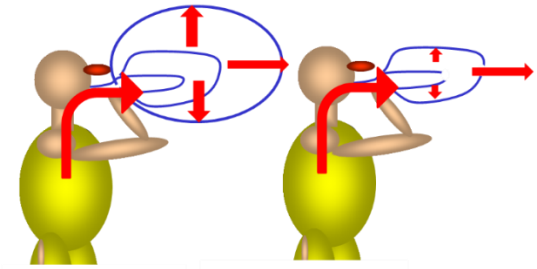
27- *Pump pressure.*

Venous pumps are alternative pumps made up of cavities limited by valves and surrounded by muscles whose relaxation called diastole sucks the venous blood and contraction called systole ejects it downstream. These are the cardiac pump PC, thoraco-abdominal pump TAP and valvulo-muscular pump VMP

271- *Cardiac pump PC*

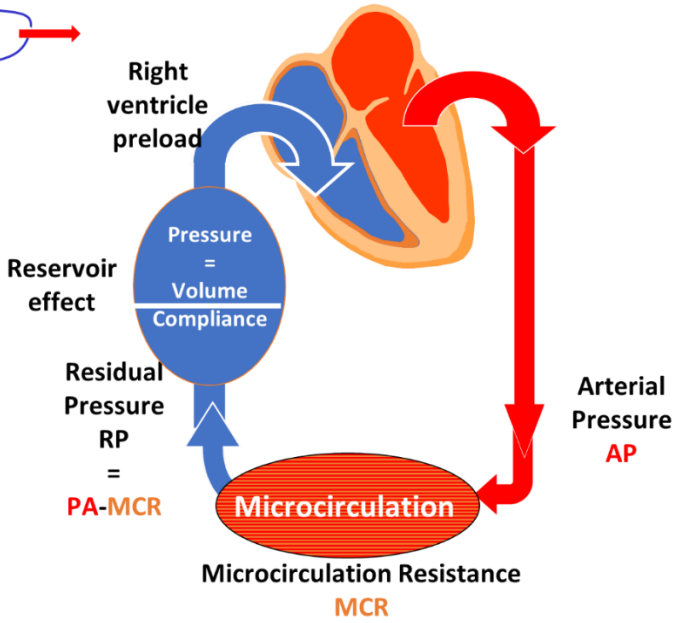
The systole of the left ventricle pushes every minute 5 litres of arterial blood with an average pressure of 90 mmHg. This blood passes through the microcirculation where it exchanges anabolic nutrients and catabolic waste products with the tissues. The microcirculatory resistances reduce the arterial pressure (loss of pressure energy) equal to 75mmHg so that the blood ends up in the venules with a pressure called "residual" RP = 90-75=15 mmHg at rest.

Assisted by atrial systole, the diastole of the right ventricle sucks in the venous vena cava blood available in the appropriate volume and pressure thanks to the reservoir effect RE. Then, systole pumps it into the left ventricle via the pulmonary circulation.



Balloon inflates with progressive pressure until it equals the thrust of the thoracic pump

Pierced balloon does not inflate completely because the balloon pressure stops increasing when it exceeds the resistance of the orifice



The reservoir effect is due to the Compliance of the venous system, which allows variations in volume to absorb pressure variations

Residual Pressure is the arterial pressure minus the pressure drop in the microcirculation.

.

These interactions between venous volume- pressures and the cardiac pump, namely the reservoir effect RE and residual pressure, produces clinical, pathophysiological, and therapeutic effects

The left heart transmits the arterial pressure of 90 mmHg reduced by the 75-mmHg pressure drop in the microcirculation to the left venous system

This pressure of 15 mmHg is called residual RP. It must remain low to ensure vital tissue drainage. It increases with downstream resistance (venous obstacle, right heart failure) and the decrease with microcirculatory resistance (vasocontraction). In case of venous obstruction, the residual pressure can increase until it stops the arterial flow (ischemia) when it equals the arterial pressure (phlegmatia cerulea).

Q=RP/R. The right heart stabilizes residual pressure RP at low values by maintaining low cardiac resistance R to flow Q. By evacuating at each diastole, the additional volume of blood supplied by each systole of the left ventricle, the right ventricle stabilizes the venous volume.

If the aspirated volume is less than the injected volume, the residual pressure increases the venous bed volume and the residual pressure increases*. The increase in RP pressure is less rapid than the volume as long as the compliance of the walls allows the veins to dilate (capacitance of the reservoir effect). This is how the venous volume is around twice the arterial volume****. **The reservoir effect RE maintains a stable pressure thanks to the variation in volume**. This pressure stability and available volume ensures a ventricular preload corresponding to the needs of the right heart.* ***Failure of the reservoir effect can lead to cardiac consequences: preload decrease (haemorrhage, " blood steal" by the veins of the lower extremities in the standing position) or excess (arteriovenous fistulas, perfusions).***

So, when, due to downstream resistance, the upstream venous bed dilates up to a maximum then cannot dilate anymore. The Residual Pressure can increase according to the downstream resistance, up to the arterial pressure which makes the venous flow pulsatile (heart pace). The consequences on the Transmural Pressure TMP excess (oedema, varicose veins, trophic disorders) can be treated by lowering resistance and/or increasing extravenous pressure (bandage) and/or GHSP reduction (lower extremities elevation).

2711- *Reservoir effect RE*.

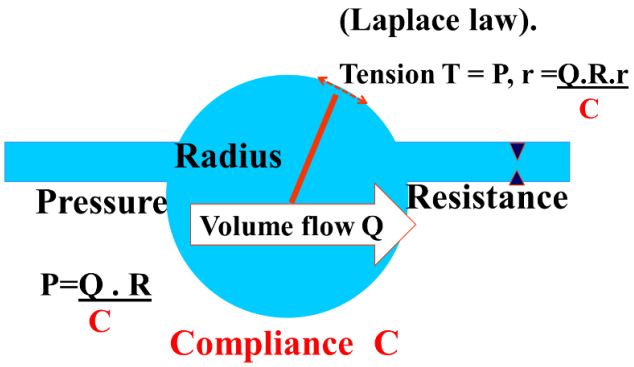
Besides its cardiological effects, Reservoir effect has a phlebological relevance.

In physiology, the ER reservoir effect is the compliant capacity of the venous system to damp pressure by varying its volume, neither too high nor too low, in accordance with the needs of the right heart preload for the cardiologist, and to damp the intravenous lateral pressure IVLP variations for the phlebologist. It is improperly attributed to varicose clusters wrongly supposed to "suck" the downwards the venous blood. As a matter of fact, varicose veins are passive while the blood flows change with the pumps work and posture changes.

It is related to physics as the capacity of a compliant viscoelastic container subjected to a flow Q and an upstream pressure P, to increase its pressure less quickly than its volume. Compliance is the ability of a container to change volume by elastic deformation when subjected by the contents to pressure forces, which has the effect of damping the pressure changes according to the relationship pressure P on volume V. ***This is the RE reservoir effect, which reduces the volume to increase the pressure and increases the to reduce the pressure.*** *This capacitive effect is the equivalent of the windkessel effect for arteries and of a capacitor of an electrical network.*

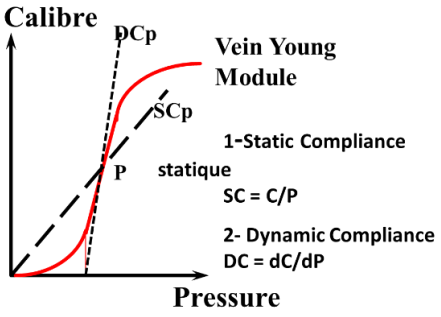
It stops when the limit of its compliance C (expansion capacity) is reached. *The Young's modulus is characteristic of the variation of the compliance according to the length and the limits of stretching of the wall.* ***When the stretching limit is reached, the volume can no longer progress, and the pressure then progresses as the upstream pressure.***

In the venous system, the variation in calibre ΔS related to Compliance C, limits excessive pressure variations ΔP ΔP= ΔS/C.



Reservoir effect and Compliance.

Pressure P and Pressure T decrease and radius r increases when compliance C increases.
The Resistance R decreases the flow Q and increases the Pressure P, and the radius r of the caliber as C is smaller



Compliance and caliber

Variation in S of the caliber C according to the Young's modulus which modulates the compliance according to the degree of dilation

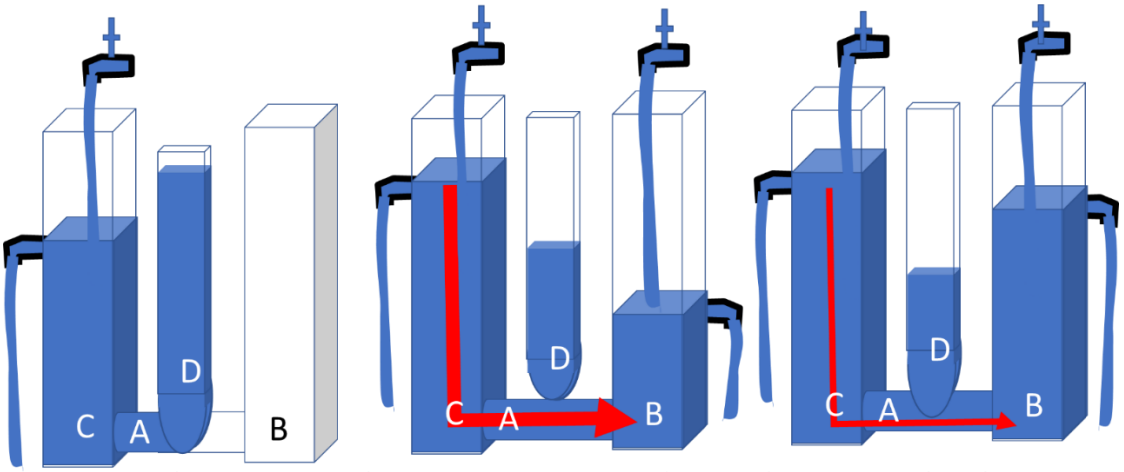
*When the vein is subjected to an Extra-Venous pressure EVP that opposes the Intravenous Lateral pressure IVLP, the Transmural pressure TMP (IVLP-PEV) must be considered. ΔTMP = ΔP/****C.***

Thus, for the cardiologist, an abdominal EVP greater than 20 mmHg can totally compress the inferior vena cava and deactivate the heart (Abdominal compartmental syndrome and compression by the gravid uterus).

On the other hand, for the phlebologist, an EVP by external compression, is welcome at the level of the extremities to reduce the TMP when the excessive IVLP, in particular because of a venous obstacle cave and / or iliac, compromises the drainage.

The concept of the Starling Resistor *is more in line with the concerns of the cardiologist, because it describes more the conditions of flow towards the heart and less the upstream pressures which are more the concern of the phlebologist.*

It consists of (below figure) of a flexible conduit A (vena cava,) flowing into B (right atrium?), crossed by the flow of a container of water C (upstream venous pressure?) and compressed by the water of a container D (extra venous pressure?). If the pressure C is lower than D, the flow A stops. If B is greater than D and less than C, the flow flows along a gradient equal to A-B. If D is greater than B and less than A, the flow flows according to an A-D gradient.



Starling Resistor: Flow resistance C according to the pressure loss by the compression of the flexible conduit A through D and the pressures C and B

The volume and pressure of venous blood is ,on the one hand, increased continuously upstream by the systoles of the left heart pump, via the microcirculation. This additional supply is absorbed by the diastoles of the right heart pump and thoracoabdominal pump, which suck it up.

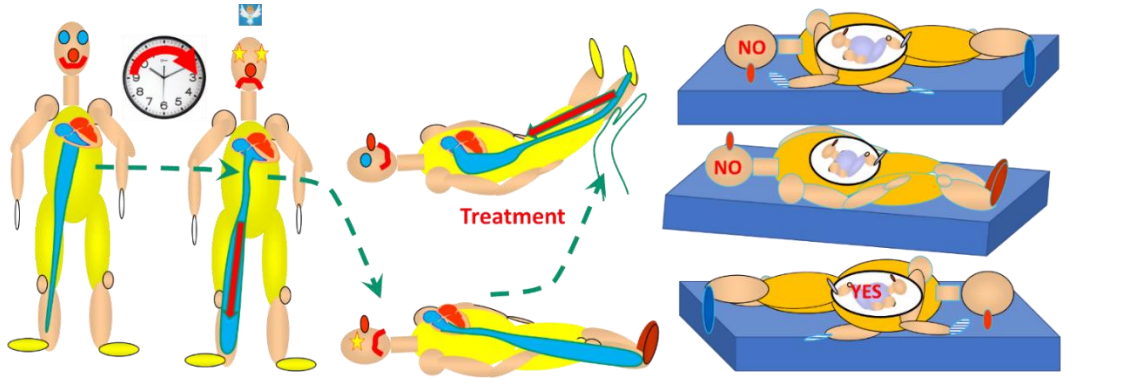
The demand for venous blood varies with cardiac output. *These variations are rapid and the immediate availability of venous blood in volume and pressure for the necessary preload of the right ventricle is possible thanks to the ER reservoir effect. The available venous volume is important because it is 2/3 of the total blood volume (blood volume) and therefore double that of the arterial volume. Unlike the arterial volume, which varies little (arterial Wind Kessel effect), the venous volume can vary considerably, either globally according to the microcirculatory flow rate, or sectorial (particularly in the lower extremities during changes in posture or obstacles to the flow). Venous compliance maintains, within the stretching limits (elongation and shortening) of the walls, pressures that are satisfactory for the heart.*

This effect can be observed by inflating a latex balloon. *At the beginning, the volume of air that I transmit to it at each expiration, is done without effort until it has reached a certain volume beyond which I must force more and more. Thus, by expanding easily thanks to its high compliance, the balloon has accepted the volume of air Q pushed by the systole of my*

thoracic pump with a low resistance R therefore without appreciably increasing its pressure P (Q =P/R and P = Q/R) until the limit of elastic extensibility (easy compliance). Beyond this limit, the pressure of the balloon increases rapidly until it equals the pressure of my expiratory systole.

***This is what happens in case of a total venous downstream obstruction**. They inflate until the limit of their distensibility, and the residual venous pressure RP then increases until it reaches the value of the arterial pressure PA. Indeed, the loss of load in the microcirculation reduces the venous pressure in proportions to its flow rate (see Poiseuille equation below). In the case of a total downstream obstacle, the flow rate and therefore the pressure drop become zero so that the residual venous pressure rises until it equals the arterial pressure*

If I pierce the balloon, it inflates less as the hole is larger, i.e., the resistance is lower**. **This is what happens in the veins upstream of an obstacle (resistance).** It swells without significantly increasing the residual pressure until it equals the resistance of the obstacle. Therefore, obstacles must be evaluated by measuring the upstream venous pressure. **Too often, therapeutic decisions are made on the appearance of obstacles rather than on their hemodynamic impact on pressure, and therefore on drainage function. If the pressure is normal, the obstacle is not hemodynamic, regardless of its anatomical and structural aspect.



Deactivation of the heart pump by default of the Reservoir Effect

It is easy to understand how the reservoir effect can be compromised when the limits of variation in vein calibre are exceeded. Either the venous bed can no longer reduce its calibre when the volume of blood is too small, or it can no longer dilate when the volume is too large.

The volume/pressure of available blood is too low in the vena cava in 3 exemplary conditions.

*The first, in case of **venous haemorrhage**.*

The second is when too much ***blood leaks from the vena cava to the lower extremities*** *during the rapid transition from recumbent to standing position. It leads to an "****intravenous haemorrhage"*** *which deactivates the cardiac pump with fainting and reflex vagal bradycardia. It is quickly corrected* ***by elevating the legs in decubitus position which operates an "intravenous re-transfusion"*** *by the blood accumulated in the lower extremities. Some people, especially* ***young people****, are more prone to these symptoms, and they react to the slightest lack of reservoir effect with a vagal shock. These discomforts* ***are well known to echodoppler practitioner who examines patients in an extended immobile standing position****. Prevention consists in* ***limiting the time of immobile station and/or administration of atropine****. The volume of varicose veins increases the volume of this intravenous haemorrhage in the lower extremities and accounts for the discomfort when standing. Their effect can be evaluated by* ***the "tilt test"*** *which consists in maintaining the patient in a quasi-upright position on an examination table and under electrocardiographic control. The preventive treatment consists in* ***reducing the Transmural pressure by elastic and inelastic compression of the lower extremities which increases the Extra-Venous pressure EVP. But also, by advising the patient to remain seated for a while, before getting up, the time to balance his volumes and pressures****.*

The third is the ***compression of the inferior vena cava by the uterus of the supine pregnant woman****, which is* ***prevented by the right lateral decubitus*** *and corrected by elevating the legs in the right lateral decubitus.*

The volume/pressure of available blood may be too high in the vena cava relative to the possibilities of the heart, especially in case of heart failure*. It was reduced by "bleeding" before the advent of diuretics.*

The reader will have understood that this physiological reservoir effect has nothing to do with the reservoir effect falsely attributed to varicose veins and which is just as falsely held responsible for aspiration and varicogenesis, as I will show later*.*

2712*-Residual pressure RP and Microcirculatory Resistances*

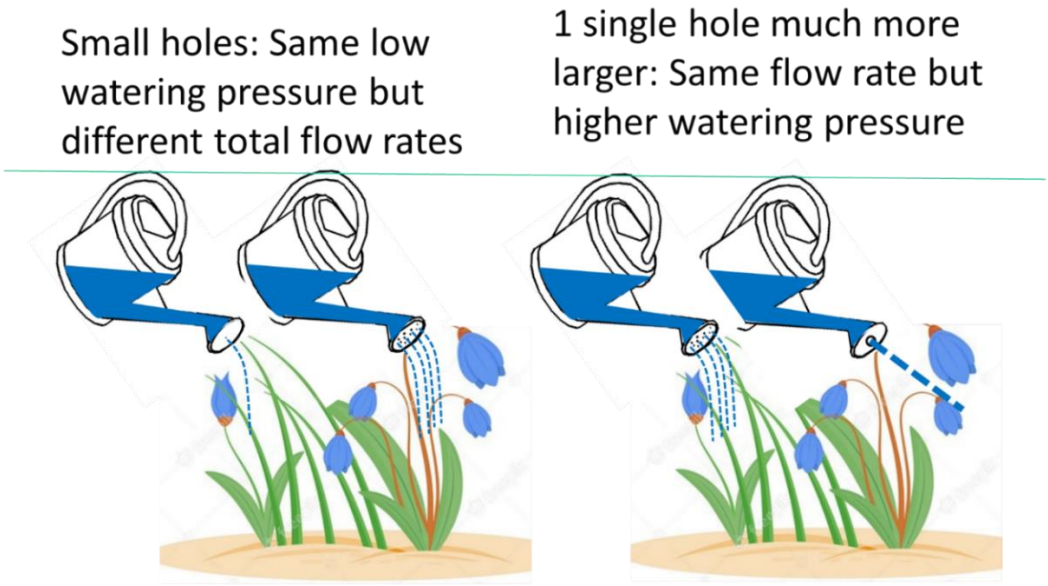
As we have seen, the residual pressure RP is the "vis a tergo" (from Latin “force which acts by pushing from the back’)of Richard Lower (1670).

The residual pressure is = 15 mmHg transmitted to the venules by the left ventricle via the arterial pressure 90 mmHg. *This important pressure drop (75 mmHg) corresponds to the loss of load of the motive pressure in the microcirculatory resistances****.***

It varies with the arterial pressure PA, the microcirculatory resistances, the calibre of the arteriovenous fistulas FAV and the venous flow resistances (venous obstacles, right heart failure). These variations are dampened within the limits of the possibilities of volume variation of the ER Reservoir Effect

Microcirculatory flow and Residual pressure.

It may seem counterintuitive that the drop in pressure does not lead to a corresponding drop in flow*. In fact, the flow rate is maintained, because although each of the microcirculatory units has a high resistance, their arrangement in large numbers and in parallel, reduces the overall resistance, in accordance with Ohm's law: the inverse of the overall resistance is equal to the sum of the inverses of each resistance. 1/R= 1/N1+1/N2/+ 1/N3 +1/Rn. To each resistance corresponds a Conductance C (capacity to leave a certain quantity of current, i.e., C =C1+C2+C3+Cn). Thus, the more resistors are added in parallel, the more the overall resistance is reduced.* ***To convince yourself of this, just imagine a watering can apple. Although each small hole resists strongly, the watering can empty all the better as the holes are more numerous, without increasing the pressure of the water poured, which avoids destroying the flowers.*** *Similarly, the arterial flow empties all the better in the arteries, as the microcirculatory units are more numerous, without increasing the venous pressure.* ***The water pressure increases with the calibre of each hole*** *of the watering can at the risk of destroying the flowers and the residual venous pressure with the calibre of the micro vessels at the risk of overloading the TMP, thus increasing the calibre of the veins and reducing the drainage of the tissues****.***

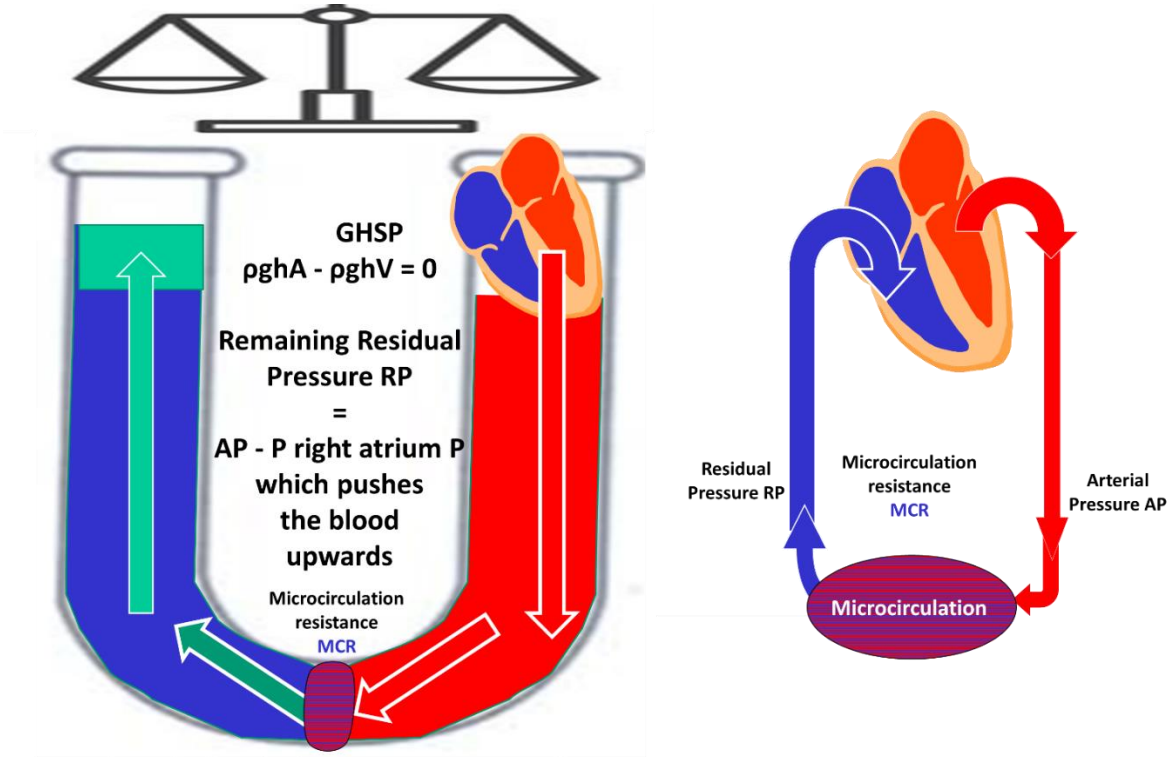


Thanks to its many resistive microcirculatory units arranged in parallel, the microcirculation provides a high flow rate at low pressure (Ohm's law on parallel resistances).

Residual pressure and return of blood to the heart in standing position.

Why does the Residual pressure return blood to the heart even though it is lower than the GHSP in the standing position? *Because the motive residual pressure does not need to be higher than it. In fact, the venous GHSP column is in equilibrium with that of the arteries because they communicate via the microcirculation, according to the physical model*

of the U-tube. ***This is equivalent to a balance with 2 pans balanced by the same weight on each side. Lifting a weight along with the pan requires much less force than lifting the weight off the pan.***



U-shaped tube. The hydrostatic gravitational pressures cancel each other out like two weights on either side of a scale. What remains is the residual driving pressure provided by the left heart which moves them

The Microcirculatory Resistances MCR cause a pressure drop which is imperfectly but sufficiently expressed by Poiseuille's law.

RP= AP- (Q 8 L μ / π r^4) = pressure drop

Q=flow rate, L=length, r=radius μ=Viscosity

We can see that we can increase the residual pressure RP by two means: either very quickly by dilating the microvessels (r^2) which transmits more volume and arterial pressure, or by reducing the flow rate Q if we add a resistance downstream of the microcirculation (venous, thoracoabdominal or cardiac obstacle. This is a resistance R2 which is added in series to the microcirculatory resistance R1, i.e. R = R1+R2. This reduces the intensity of the current, and therefore the flow Q. This microcirculatory flow Q can therefore decrease when the resistance increases until it is cancelled when R2 equals the arterial pressure AP. (uncompensated major venous occlusion leading to an arrest of arterial circulation as seen in the fortunately rare phlegmatia cerulea which associates major oedema and ischemia). This

is undoubtedly the case of frequent "matting" where the "therapeutic" procedure has completely occluded the drainage veins or venules and the residual venous RP pressure approaches the arterial pressure. This is surely the case of "recurrences" that follow destructive treatments of the veins, thus of the drainage routes. In these cases, the R2 resistance increases the residual pressure, which hinders local drainage of the skin, favours telangiectasias, and then forces and dilates other compensatory veins (see Open Vicarious Shunts).

2713: Right heart failure.

If right atrial resistance (tricuspid stenosis and incompetence, pulmonary hypertension) increases the left heart-right heart pressure gradient decreases and residual pressure RP and venous volume increase.

Tricuspid reflux leads to a systolic pressure/volume surge in the upstream veins, which results in a synchronous venous stop or reflux, depending on whether the veins are competent or incompetent.

***This pulsed venous flow reflux must not be confused with the anterograde pulsed flow produced by the microcirculation**. Microcirculatory resistances are low during inflammation, or shunted by arteriovenous FAV fistulas, but also when downstream resistances are exceedingly high (severe venous occlusions).*

272- Thoracoabdominal Pump TAP

The thoraco-abdominal pump is interposed between the extremities and the heart. It consists of the muscles of the thorax and abdomen whose respiratory movements vary the volumes and pressures of the veins they contain. The systole and diastole of the TAP, which compresses and dilates them, affect the venous pressures and volumes of the lower extremities.

2721- Physiological respiratory modulation of flow and pressure of the lower extremities.

It is usually the effect of diastole that dominates. ***Any limit to inspiration reduces venous drainage of the extremities.***

The velocity of the flow of the veins of the lower extremities therefore increases during inspiration and slows down during expiration).

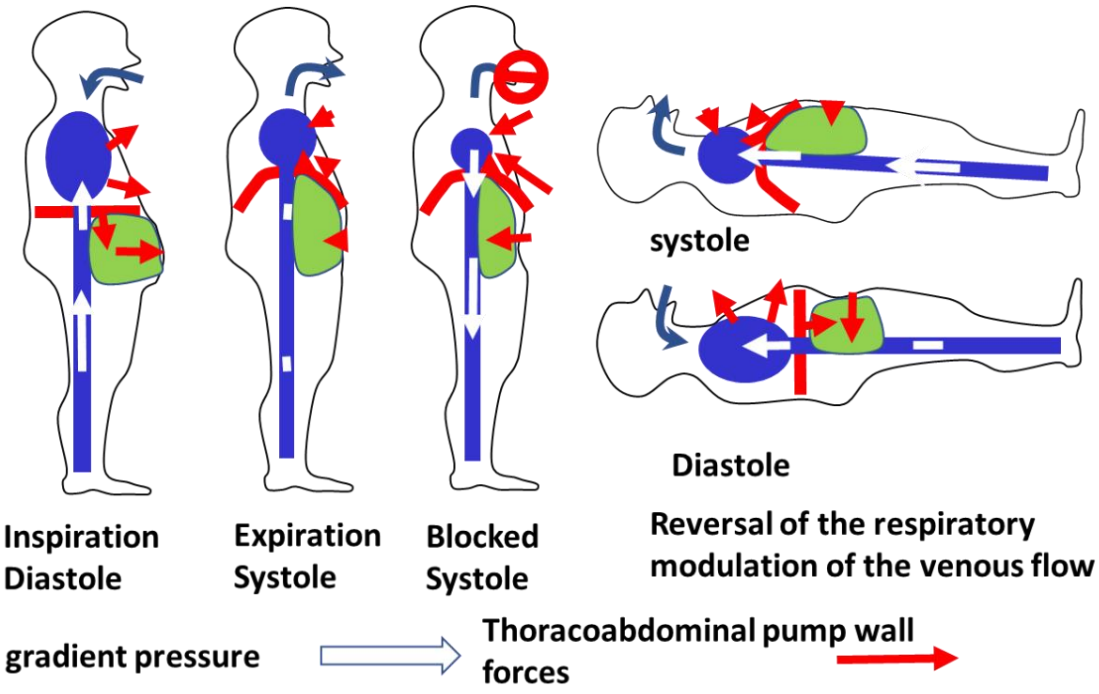
***This modulation is reversed physiologically by posture**. Indeed, in decubitus, the femoral flow is reduced by inspiration and restored by expiration. This is due to the compression of the inferior vena cava by the weight of the abdominal viscera associated with the reduction in volume of the abdominal cavity the diaphragmatic thrust (see figure below)*

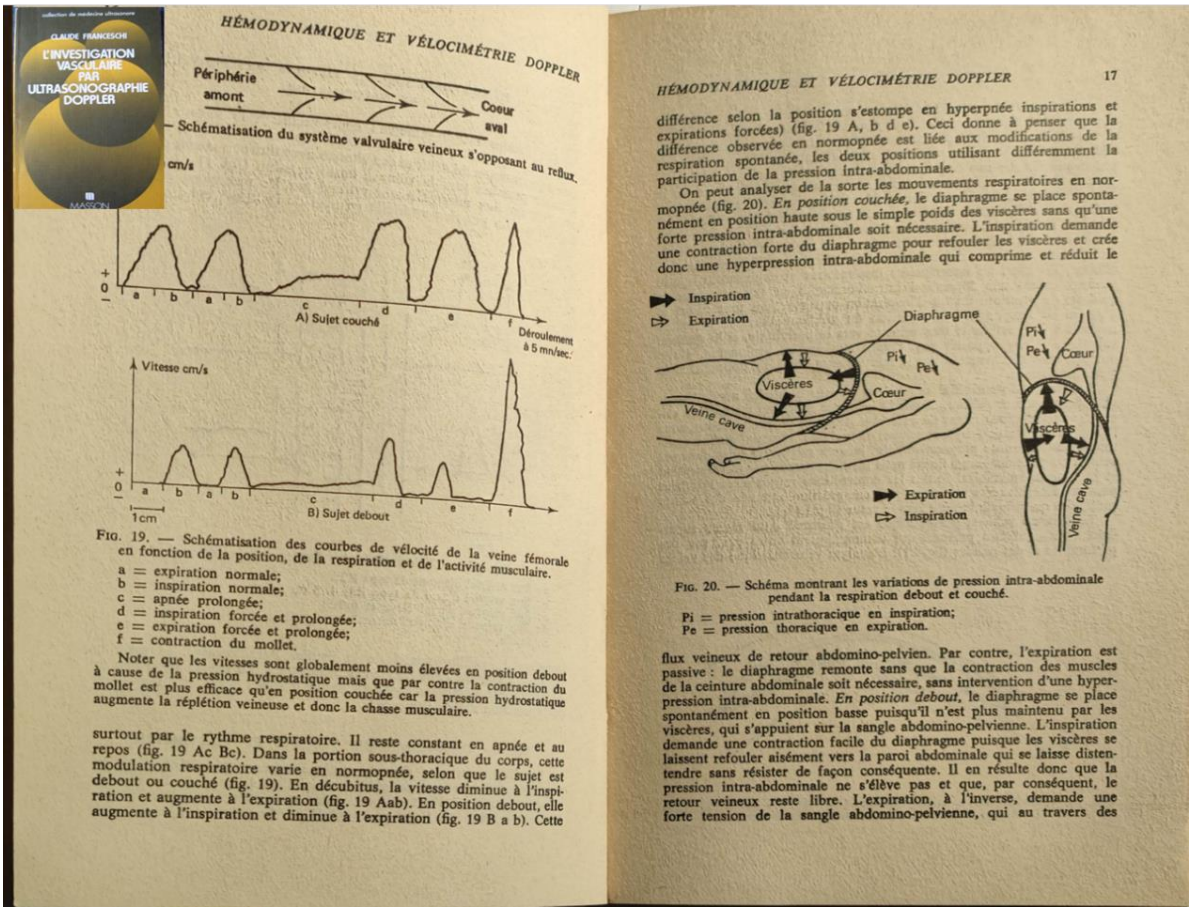
2722- *Pathological respiratory modulation of flow rates and venous of the lower extremities*

If venous pressure increases due to an iliac and/or cava obstacle, the respiratory flow modulation no longer varies. ***This explains the lack of respiratory variation in femoral flow velocities on Doppler, which thus supports the diagnosis.*** *This non-variation at rest can occur only by increasing the flow/pressure in the veins of the lower extremities when the obstacle is hemodynamically significant in these conditions alone.* ***This increase can be achieved by 20 free pedalling in supine position.***

Forced Expiration.

Forced expiration against an obstacle (blockpnea) ***reverse downwards the pressure gradient****. These conditions occur during the* ***wearing of heavy weights and during defecation efforts****, which normally results only in dilation and pathologically in reflux when the veins of the lower extremities they are incompetent.* ***The forced blockpnea is used for the Valsalva manoeuvre which is mandatory to assess the venous competence.***





HÉMODYNAMIQUE ET VÉLOCIMÉTRIE DOPPLER

Périphérie amont — Cœur aval

Schématisation du système valvulaire veineux s'opposant au reflux.

cm/s

A) Sujet couché

Déroulement à 5 mn/sec.

Vitesse cm/s

1 cm

B) Sujet debout

FIG. 19. — Schématisation des courbes de vélocité de la veine fémorale en fonction de la position, de la respiration et de l'activité musculaire.

a = expiration normale;
b = inspiration normale;
c = apnée prolongée;
d = inspiration forcée et prolongée;
e = expiration forcée et prolongée;
f = contraction du mollet.

Noter que les vitesses sont globalement moins élevées en position debout à cause de la pression hydrostatique mais que par contre la contraction du mollet est plus efficace qu'en position couchée car la pression hydrostatique augmente la réplétion veineuse et donc la chasse musculaire.

surtout par le rythme respiratoire. Il reste constant en apnée et au repos (fig. 19 Ac Bc). Dans la portion sous-thoracique du corps, cette modulation respiratoire varie en normopnée, selon que le sujet est debout ou couché (fig. 19). En décubitus, la vitesse diminue à l'inspiration et augmente à l'expiration (fig. 19 Aab). En position debout, elle augmente à l'inspiration et diminue à l'expiration (fig. 19 B a b). Cette

différence selon la position s'estompe en hyperpnée inspirations et expirations forcées) (fig. 19 A, b d e). Ceci donne à penser que la différence observée en normopnée est liée aux modifications de la respiration spontanée, les deux positions utilisant différemment la participation de la pression intra-abdominale.

On peut analyser de la sorte les mouvements respiratoires en normopnée (fig. 20). *En position couchée*, le diaphragme se place spontanément en position haute sous le simple poids des viscères sans qu'une forte pression intra-abdominale soit nécessaire. L'inspiration demande une contraction forte du diaphragme pour refouler les viscères et crée donc une hyperpression intra-abdominale qui comprime et réduit le

Inspiration
Expiration
Diaphragme
Pi
Pe
Viscères
Cœur
Veine cave
Expiration
Inspiration

FIG. 20. — Schéma montrant les variations de pression intra-abdominale pendant la respiration debout et couché.

Pi = pression intrathoracique en inspiration;
Pe = pression thoracique en expiration.

flux veineux de retour abdomino-pelvien. Par contre, l'expiration est passive : le diaphragme remonte sans que la contraction des muscles de la ceinture abdominale soit nécessaire, sans intervention d'une hyperpression intra-abdominale. *En position debout*, le diaphragme se place spontanément en position basse puisqu'il n'est plus maintenu par les viscères, qui s'appuient sur la sangle abdomino-pelvienne. L'inspiration demande une contraction facile du diaphragme puisque les viscères se laissent refouler aisément vers la paroi abdominale qui se laisse distendre sans résister de façon conséquente. Il en résulte donc que la pression intra-abdominale ne s'élève pas et que, par conséquent, le retour veineux reste libre. L'expiration, à l'inverse, demande une forte tension de la sangle abdomino-pelvienne, qui au travers des

273- Valvulo-muscular pump. Dynamic Fraction of the Gravitational Hydrostatic pressure DFGHS, valve incompetence and Shunts.

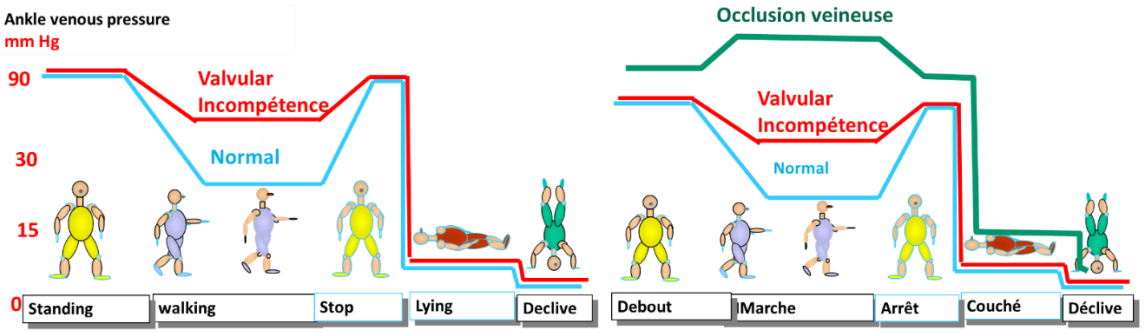
2731 *Dynamic fractioning of the Gravitational Hydrostatic Pressure DFGHSP is the segmentation of the venous blood column by the systo-diastolic alternate closure of the inlet and outlet valves of the muscular pumps of the lower limbs.*

Pressure and DFGHSP

During walking, the valvulo-muscular pumps *of the lower extremities, primarily those of the calf, combine 2 effects.*

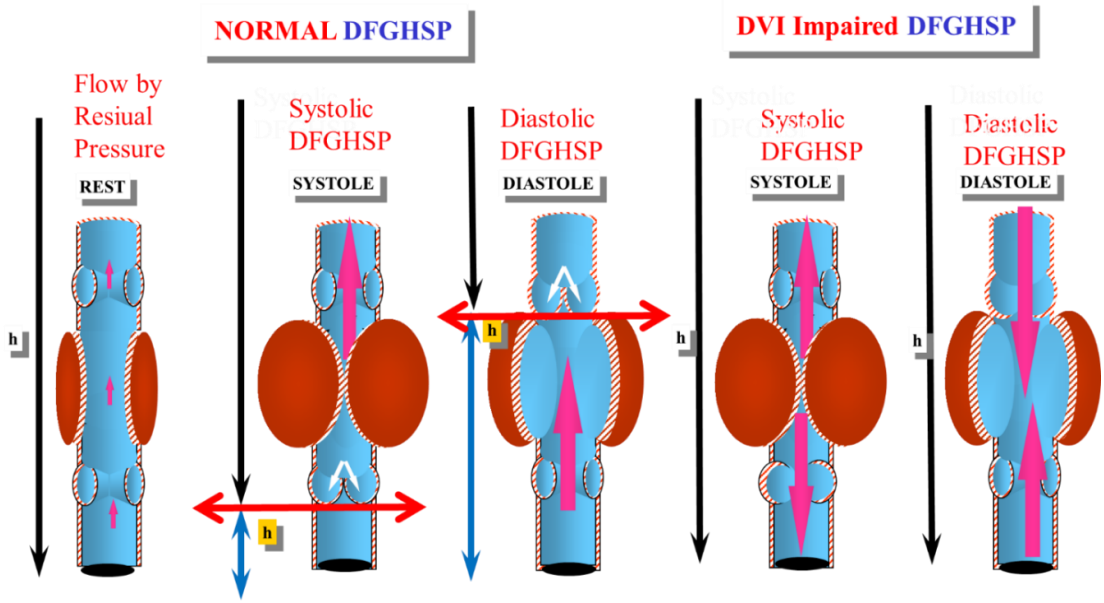
-The first is the ***dynamic fractioning of the gravitational hydrostatic pressure DFGHSP****.*

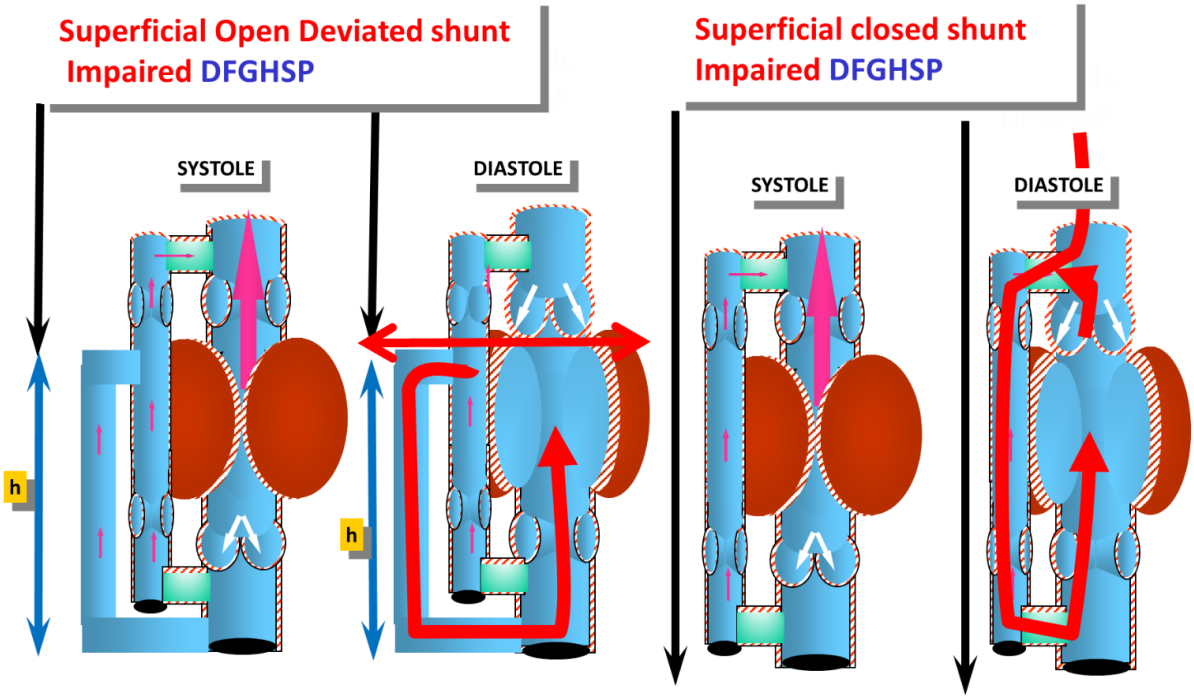
-The second is the ***evacuation of the flow-pressure generated by the muscular activity*** *(increase of the cardiac flow transmitted to the veins by reduction of the microcirculatory resistances).*



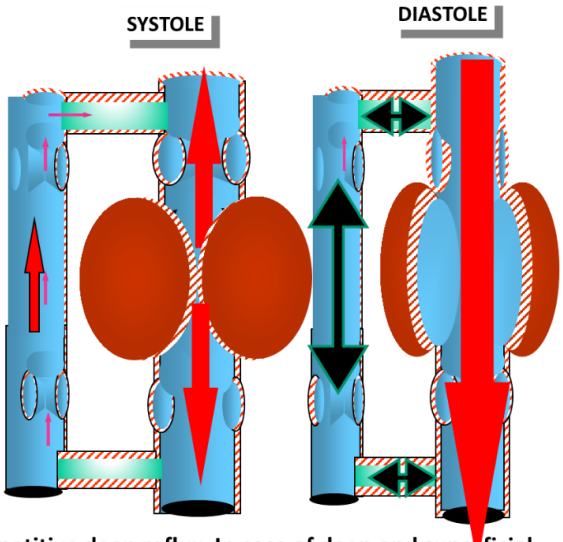
Valvular Incompetence impairs the Dynamic Fractionation of the Gravitational Hydrostatic Pressure DFGHSP . It is not pathogenic at rest, but only during walking !!!!

The obstacles are pathogenic at rest and especially during walking: venous claudication





Deep competitive reflux



Competitive deep reflux. In case of deep and superficial valvular incontinence, deep reflux dominates and prevents, when it is major, the reflux of the greater saphenous vein, even though it is varicose.
No reflux of the great saphenous vein on Doppler and the Perthes test does not result in its collapse

The volume ejected varies according to the muscle power and the venous volume of the calf, so according to each individual conformation. Its average volume is 70 ml.

The effect of systole on the pressure of the posterior tibial, popliteal, and great saphenous veins was measured by Arnoldi in 10 young healthy subjects.

Ref: Arnoldi CC: Venous pressure in the legs of healthy human subjects at rest and during muscular exercise in nearly erect position. Acta Chir Scand 1965; 130:570-583).

During calf contraction, the pressure increased on average by 75 mm Hg in the posterior tibial vein in addition to the 83 mm Hg at rest, 29 mm Hg in the popliteal vein in addition to the 61 mm Hg at rest, and 34 mm Hg in the great saphenous vein in addition to the 83 mm Hg at rest.

The pressures at rest correspond to the height of the overlying unfractionated gravitational hydrostatic pressure column (open valves), during walking the pressures of the posterior tibial and great saphenous veins, drop from 90 mmHg to 38 mmHg +-6 mmHg. This is consistent with the levels of dynamic fractionation of the gravitational hydrostatic pressure DFGHSP that I propose, produced by the successive and alternating systo-diastolic closure of the upstream and downstream valves of the pumps. The absence of reduction of GHSP during walking in proportion to valvular incompetence confirms this model.

The Léjars pump, i.e., the venous network of the sole of the foot, produces little flow (few ml) and pressure compared to the other pumps of the lower extremities.

2732- Veno-venous shunts, valvulo-muscular pump and cardiac pump.

The concept of veno-venous shunts marks a decisive turning point in the progress of knowledge and treatment of venous physiopathology.

27321- Definition of shunts

<u>Veno-venous shunts are present in most pathologies of the venous system</u>

In medicine, the term shunts usually refers to communications between arteries and veins through "fistulas", "niduses" or surgical anastomoses.

<u>If we refer to the physical definition of shunts</u>, we see that this term can be attributed to other models.

Indeed, a shunt defines in fluid mechanics, a ***pathway that deviates by an escape point EP all or part of the flow of a network and then restores it by a re-entry point RP****.*

An arteriovenous shunt is an open shunt that diverts blood from an artery to a downstream vein bypassing the capillaries. Arteriovenous shunts that by-pass the capillaries are called

micro-shunts. Direct truncal communications between arteries and veins, which shunt the microcirculation downstream of the venules, are called arteriovenous fistulas.

***I call veno-venous shunt a vein whose physiological drainage flow is overloaded by blood from a vein from which it diverts (steals) all or part of the flow**. It takes blood from an escape point EP and gives it back by a re-entry point RP. **We have already understood that removing this shunt, is certainly removing the overload, but it is also removing a physiological draining flow**. This is like throwing the baby out with the bath water. **Conservative hemodynamic treatments such as the CHIVA cure avoid this mistake**.*

27322- Hemodynamic classification of venous shunts. Venous shunts. OVS, CS and ODS.

Some favour the drainage, others impair it.

273221-Veno-venous shunts favourable to drainage.

An open vicarious shunt OVS by-passes and compensates a venous obstacle. It is activated by the heart through the residual pressure and/or by the systole of the valvulo-muscular pump during walking. The escape points EP overloads it with the flow/pressure of the shunted vein, and the re-entry point RP empties and returns it beyond the obstacle. The more the calibre of the shunt, the less the upstream pressure and transmural pressure TMP for better drainage.

I call them vicarious (which means compensatory of the defective normal pathway) and open because they are don't recirculate not in closed circuit. They are activated by the cardiac pumps at rest and when walking, and by the systole of the valvulo-muscular pumps when walking.

273222-Veno-venous shunts hostile to drainage.

2732221-The Closed CS shunts hampers the drainage

The Closed CS shunts impairs the DFGHSP, overloads shunting veins, the microcirculation and the valvulo-muscular pump.

The valvulo-muscular pump activates a closed circuit made of the closed shunt. At each diastole, it aspirates via the closed shunt all or part of the volume that it has previously ejected downstream during the systole. So, the physiologic draining flow of the shunting vein is overloaded by the diastolic pressure/volume fed by the deep N1 shunted veins through an escape point EP and returns it back into the entry of the valvo-muscular pump through a re-entry point RP. This overloaded diastolic recirculation dilates the shunt and increases the superficial transmural pressure, which is the cause of most of varicose veins and skin damages especially at the re-entry point RP. Nevertheless, a large re-entry point

MUST be preserved because of its physiological draining function. The overloading flow is stopped at the EP by a disconnection.

Trendelenburg *suspected this closed circuit which he called "private circulation".*

Although innocent at rest because activated only by the valvulo-muscular pump (particularly calf) , and unrelated to the cardiac pump, they are highly pathogenic during walking. *Indeed, they overload the venous system, only when we activate the valvulo-muscular pump, usually when we walk. So, it is more the shunts than the dynamic fractionation of the gravitational hydrostatic pressure DFGHSP that develops varicose veins,* ***as proven by their "disappearance" in who becomes paraplegic.***

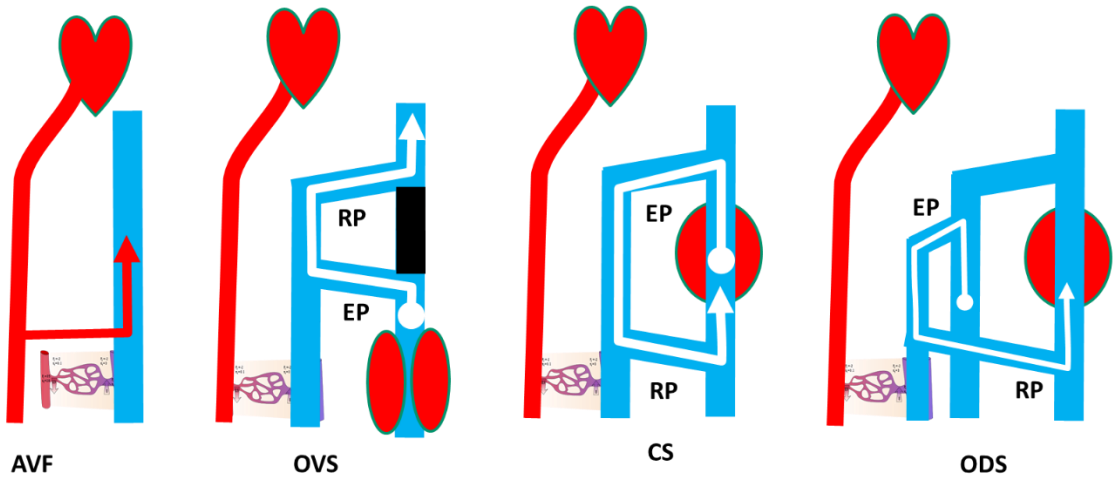
The pump thus tends to increase the venous volume of the calf instead of reducing it and increases the intravenous part of the transmural pressure accordingly. Its diastole pumps in the deep blood overloaded by the shunt through the re-entry point RP. Then, the volume ejected by the systole that follows is partly sucked once into the shunt by the diastole. The more the power and output of the pump, the more the shunt overload and dilation.

This is also why walking as well as sports should be discouraged to varicose subjects*...unless they correct the transmural pressure by using compression socks.*

*One can also imagine how, at the end of diastole****, the sudden stop of reflux will brutally transform the dynamic energy into static energy which will shock and widen the re-entry perforators****. This explains why the calibre of the* ***re-entry perforators is not the cause but the effect of the shunt****.* ***Closing these re-entries*** *instead of disconnecting the escape point, stops the overloaded flow but leaves behind a high non-fractioned column and hampers the physiologic flow in drainage.* ***Destroying the shunting vein increase the drainage impairment. CHIVA ablates the overloading flow without destroying the veins nor occluding the re-entry. This anti-physiological practice, which consists, as I have already emphasized, of "throwing the baby out with the bathwater", is still very frequent***

2732222-The open deviated shunts ODS hampers the drainage

The open deviated shunts ODS are, like the closed shunts CS, activated by the diastole of the valvomuscular pump but they are not "closed". Indeed, they function in open and not closed circuit. They are made of tributaries N3, which are overloaded by the blood they deviate from the saphenous veins' trunks N2 through an escape point EP N2>N3, instead from the deep veins N1. N1>N3. Then they drain into the entry of the valvulo-muscular pump through a re-entry point RP. Their overload, although not as heavy as that of the CS, dilates them and reduces the skin drainage.



AVF: arteriovenous fistula.
Veno-venous shunts: physiological drainage flow overloaded by the flow that it diverts from another vein by an escape point EP that it restores by a re-entry point PR.
OVS: vicarious open shunt bypasses an obstacle Activated by the systole of the valvulo-muscular pump and drainage.
CS: closed shunt by valvular incontinence. Activated by the diastole of the valvular pump veins. Self-feeding in closed circuit.
ODS: Open shunt by bypass, incontinent tributary diverts flow from an saphenous trunk without self-supply.

2732223- *Open Vicarious shunt OVS eases the drainage, as it passes the obstacles.*

An open vicarious shunt SOV is activated by the heart and/or the systole of the valvulo-muscular pump. The Escape point EP which overloads it and the re-entry point RP which empties it are on either side of the obstacle. Its compensatory quality depends on its calibre and its length.

2732224-*A mixed shunt MS associates OVS that eases the drainage with a CS that hampers it. So, it combines the good and the bad. The challenge is to save the good and eliminate the bad.*

Mixed shunts MS are activated by the systole for the open vicarious shunt OVS then by the diastole for the closed shunt. *They share the same escape point EP and the same first pathway. Then they diverge in different veins that lead to different re-entry points.* ***One can understand that treating MS without respecting the OVS part is nonsense. CHIVA disconnects selectively the CS from MS where it diverges from the OVS, which leaves behind a patent OVS and disconnected CS.***

I will detail these distinct types of shunts in their functional and anatomical pathological context in the chapter 5 (55216) devoted to venous insufficiency

28- Plasma Oncotic Pressure POP and Interstitial Oncotic Pressure IOP

The Transmural pressure TMP, a positive mechanical pressure directed towards the tissues, opposes drainage. However, drainage is possible thanks to an opposing force that is greater than it.

It is the Oncotic pressure of the plasma macroproteins POP superior to the Interstitial Oncotic Pressure IOP which creates a Oncotic Pressure Gradient OPG= POP-IOP directed towards the capillary lumen.

Drainage is reduced in 3 circumstances.

*-When **TMP is higher than the normal OPG** i.e., when the **lateral intravenous pressure IVLP is too high or when the extra venous pressure PEV is too low.***

*-When **OPG too low** because*

POP is too low. (hypoproteinaemia) or

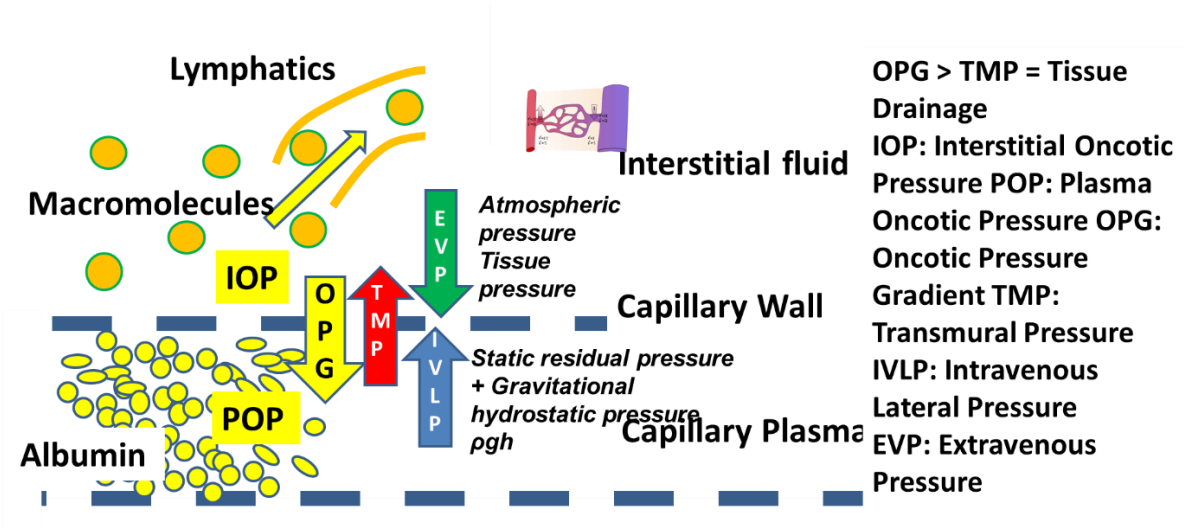
IOP is too high** (**lymphatic insufficiency, inflammation, tissue trauma).

Plasma oncotic pressure is the osmotic pressure of blood.

It attracts fluids,** through the capillary wall, **from Interstitium of the tissues where protein concentration. Is lower.

This difference in concentration is the oncotic pressure gradient OPG.

*It attracts the interstitial tissue fluid to the plasma through the capillary wall. This wall is a semi-permeable filter which allows liquids to pass through and prevents macroproteins from passing through. The oncotic pressure of plasma albumin is 22 mmHg, i.e., 80% of the mean plasma oncotic pressure which is equal to 28 mmHg. The oncotic pressure gradient OPG allows tissue drainage when it is higher than the capillary transmural pressure **TMP pressure which opposes it. If TMP is too high or the capillary oncotic pressure is too low, tissue drainage is disrupted, with its tissue consequences (oedema, trophic disorders, ulcers). Compression** increases the static pressure of the interstitial fluid, which, by **reducing the TMP, restores drainage** but not changing the oncotic pressure. **This is the true effect of compressive bandaging and not the veins compression.** Here again, the concept of transmural TMP and its regulation remains central to venous function. **The interstitial oncotic pressure is kept low by the lymphatics** which drain the macro proteins. This **OPG-TMP interaction accounts for drainage disorders** such as oedema due to **hypoproteinaemia** or excess macroproteins in the interstitial fluid due to **lymphatic insufficiency**. The **excess of TMP** can indirectly overload the lymphatics. Indeed, the **tissue suffering that it produces, releases waste and proteins into the interstitial fluid,** which reduces the OPG which can even less oppose the TMP.*



29- *Intravenous Lateral pressure (IVLP), Motive Pressure, Pressure Gradient and Pathophysiology.*

The calibre of veins depends on the physical characteristics of their wall (Hook's, Young's and Laplace's laws, Compliance) subjected to the transmural pressure TMP but also to shear stress.

291- *The Intravenous Lateral pressure IVLP is the sum of:*

-Static Pressure p +

- Gravitational Hydrostatic Pressure ρgh

that push outwards the venous walls

The Dynamic Pressure ½ρv² does not exert pressure against the wall in laminar regime. In turbulent regime, turbulence aggravates the aggression against the walls.

Note that these regimes are not permanent and change from one to the other depending on the circumstances (rest and walking according to the conditions of the venous system, normal, incompetent, obstructed).

Reminder:

Bernoulli equation: Total pressure Pt = p + ½ρv² + ρgh

½ρv² = dynamic pressure PD (kinetic energy)

ρ= Density and v velocity),

p = static pressure

ρgh = hydrostatic gravitational pressure GHSP.

ρ = Density (weight/volume)/g also called mass density.

Pm = Driving pressure = p +. ½ρv²

IVLP = Intravenous Lateral pressure = p + ρgh = Pt- ½ρv²

The static pressure P increases when the velocity V decreases and vice versa (P°+ ½ρv² = constant by conservation of energy);

The Gravitational Hydrostatic pressure GHSP increases with h, so according to the posture, lying, sitting, standing.

When the blood column is immobile, Total pressure Pt = Lateral Intravenous pressure IVLP = ρgh

***During walking ρgh decreases due to** the blood column fragmentation DFHGP. At the same time, the **motive pressure Pm (p +. ½ρv²)** increases with two mechanisms. **Residual pressure RP increased** by Arterial pressure rise+ Microcirculation resistance decrease (Vasodilatation) and. **valvulomuscular pumps output**.*

*It is understandable why, **during walking**, the **resistance R of venous obstacles** increases **the intravenous lateral venous pressure** IVLP, which **dilates the veins and tense their wall**, leading to pain and claudication. **Nevertheless,** walking is beneficial in these cases. By "**forcing" the collaterals, it progressively reduces downstream resistance, especially since it is performed under restraint, which reduces TMP**.*

*On the other hand, we understand **the "harmful" effects of walking, which by successive diastoles, overloads the veins of the closed CS shunts and the Open ODS shunts between the escape point and the re-entry point.** The flow and pressure energy are distributed into driving pressure (p, ½ρv² and turbulence) that **dilates the shunt**. We also see that the maximum surcharge is at the re-entry point as approximately shown by Bernoulli's theorem. Let's take for **example a shunt closed by incompetence of the Great Saphenous vein of the thigh and leg**.*

Total IVLP1 at the point of escape = p1 + ½ρv1²+ = Total IVLP2 at the point of re-entry = p2 + ½ρv2²+ ρgh2

Either if the shunt is a great saphenous vein with an EP at the saphenofemoral junction and a re-entry perforator at the ankle, i.e., h = 80 cm, h1 = 80 cm, and h2 = 10 cm show that p2 is increased by ρgh1 -ρgh2- compared with p1. If the femoral and iliac veins above the phenol-femoral junction, h is increased to the height of the heart

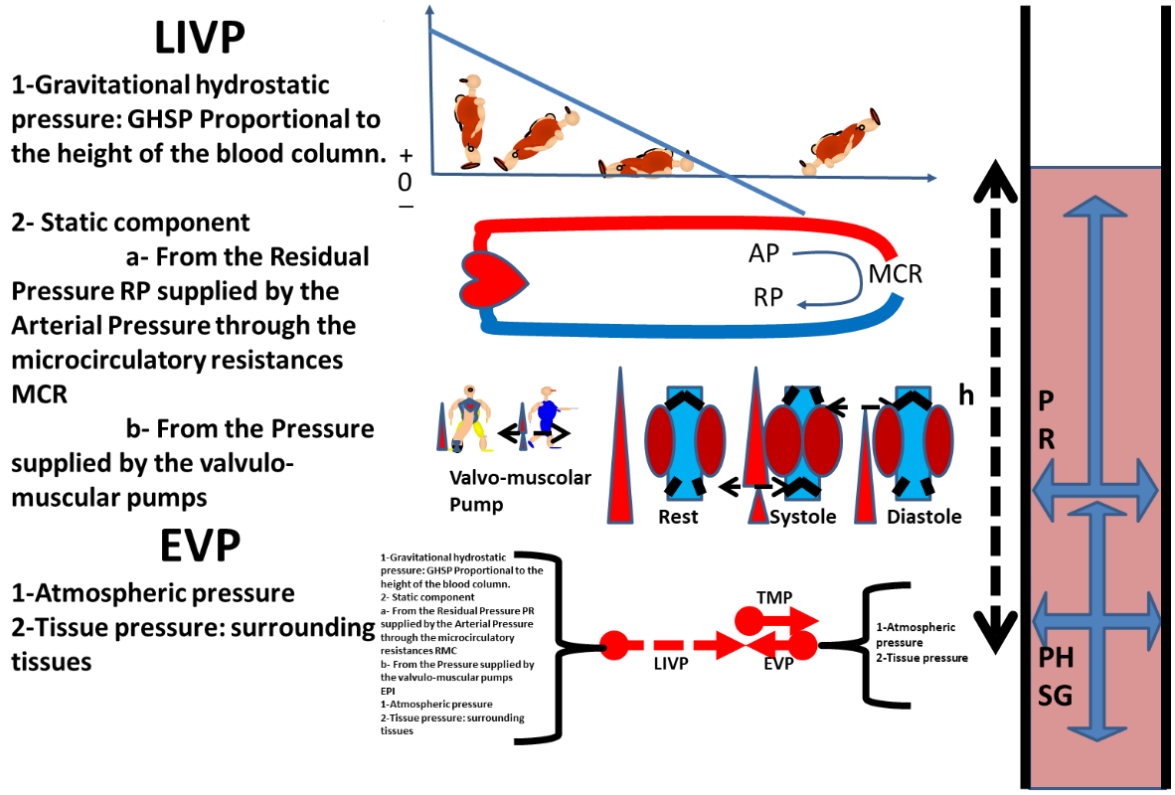
One can also imagine how, at the end of diastole, the sudden stop of the blood mass m of kinetic energy ½ mv² (v² = 2gh Torricelli) will shock and dilate the perforating vessels of re-entry (inwards water hammer physically due to Pressure surge wave usually caused by a liquid forced to stop or change direction suddenly) traumatize the surrounding tissues, and create the bed of the reactive hypodermitis and the ulcer (combination of trauma and obstacle to drainage) Classically, in phlebology books, the water hammer was described outwards by perforator reflux due to deep venous disease. Then, any dilate perforator was and is still considered pathogenic by some physicians even if it is NOT outwards refluxing, which leads to ablate them, so hampering drainage. The hemodynamic treatment aims to suppress the flow-pressure overload while the physiologic draining flow is preserved.

*It is also understood that the **dilation of the great saphenous vein stabilizes when it has reached a calibre which, for the same flow rate reduces the velocity, and therefore the turbulence and the parietal constraints**.*

*Conversely, the **disconnection of this shunt** at the EP escape point will **reduce the diastolic driving pressure and fragment the gravitational hydrostatic pressure column**. The saphenous vein reduces its calibre **and "repairs" itself by reactive remodelling** to the new hemodynamic conditions.*

Rational hemodynamic correction. In case of venous obstruction, it** must suppress the **obstacles** or encourage their by-pass (open vicarious shunt **OVS**) by collateralization (**walking under strong bandaging compression**). In case of closed shunt **CS** or Open deviated shunt **ODS**, It must treat the gravitational hydrostatic pressure DFGHSP impairment and the source of the overload, **by CS and ODS disconnection at the escape points.

*This leads to **2 clinical consequences**. Because walking and running increase the calibre of varicose veins more than standing still, **proper compression** during these activities is indicated. **Occlusion by ligation or sclerosis of large calibre re-entry perforators must be "forbidden"** because not only is this calibre not the cause but the consequence of the reflux. The overloading flow blood must be suppressed without suppressing the physiological draining flow. CHIVA cure preserves these perforators, splits the gravitational hydrostatic pressure column and eliminates the escape point responsible for their diastolic overload, while preserving the drainage flow which, although retrograde, ensures its physiological function.*



292- *The motive pressure MP = p + (1/2) mv², Obstacle and valvular incompetence.*

The motive pressure Mp = p + (1/2) mv² is low at rest = 15-15 mmHg at the ankle but increases significantly during the muscular effort of walking due to the increase in flow and pressure supplied by the left heart via the microcirculation and by the valvulo-muscular pumps.

It is constant in the sense that for the same force that produces it, the static pressure p and dynamic pressure are converted into each other (law of conservation of energy), so that one increases by the value of the decrease of the other, without their sum changing. This constant shows how the static pressure p increases dilates the vessel as the velocity v

decreases due to downstream resistance. It also shows that p can reduce until it collapses the vessel when, due to the reduction of downstream resistances, the velocity increases. ***This is contrary to a first false intuition according to which the velocity would rather tend to dilate. The Pitot tubes are the experimental proof of this, showing in addition the suction effect of the velocity v on the walls known as the Venturi effect.***

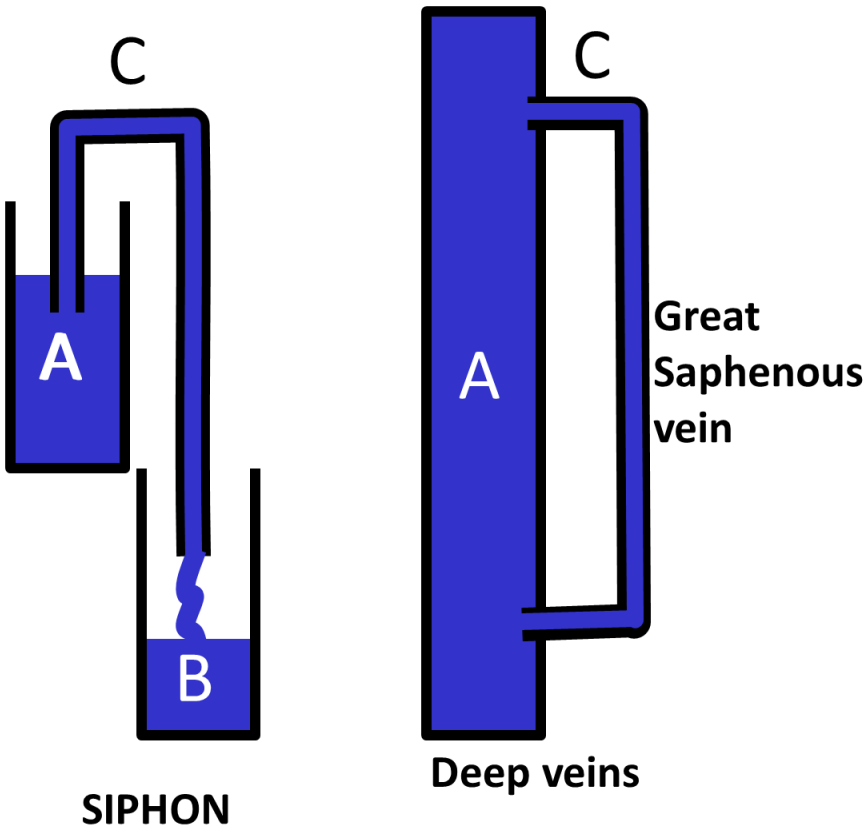
293- Pressure gradients.

The pressure gradient PG is misinterpreted as the cause of flow. It is simply the measure and not the cause of the pressure difference Pa and Pb between two points a and b distant by a length L (PG= (Pa-Pb)/L)*. It is zero between two points of horizontal flow of a liquid if there is no intermediate pressure drop or if the liquid is at rest. It is greater than 0 in proportion to the pressure drop between the 2 points. It is not zero between two points of height of a liquid where it is proportional to the height difference between the 2 points, h1 higher than h2. Gravity force and potential energy gradient (ρgh1 – ρgh2) are downwards vertical while the pressure gradient is upwards. The driving pressure gradient DPG is not the cause, but the measure of charge loss, i.e., the hemodynamic significance of a stenosis.*

294-Siphon effect:

The reflux of the saphenous veins is not the result of a siphon effect.

The true siphon effect allows to transfer the liquid from a container A to a container B located lower, without any other energy than the difference of hydrostatic gravitational potential. See fig. below. So, without pumping. They are both in the open air and separated by a height Δh. They communicate in an open circuit, by means of a pipe with a part C of its path higher than the surfaceof the liquid in container A. Thus, the siphon effect is not applicable to reflux from the great saphenous vein, because its supposedly C branch is not higher, but lower than the supposedly A deep venous blood column to which it is connected. Moreover, the reflux is not spontaneous, but must be activated by a pump (calf). It lasts only the time of diastole and not until the complete emptying of the deep blood. Finally, the reflux returns, in closed circuit (not open) by a re-entry. in the same supposed to be A and not in B. ***Contrary to a car whose tank can be emptied without a pump, the saphenous reflux requires that we walk to activate the pump. It is not the vein that passively draws blood, but the calf pump. However, this abuse of language is often used to justify an erroneous conception of venous hemodynamic.***



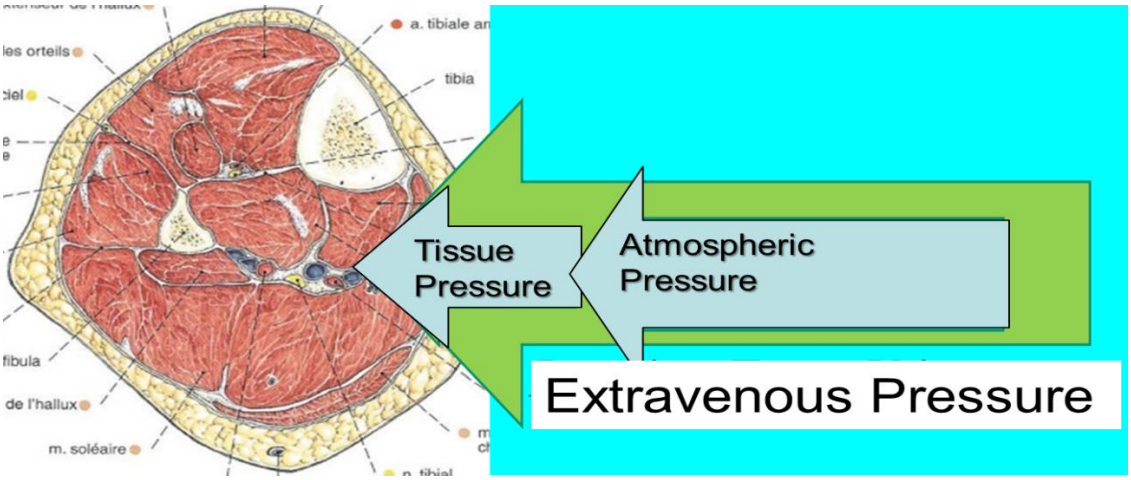
The reflux of the Great Saphenous vein is not a syphon effect

295- Extravenous pressure EVP

Extravenous Pressure Extra venous pressure is the external part of the transmural pressure TMP. Added to the oncotic pressure gradient OPG, it opposes the intravenous lateral pressure IVLP.

<u>It is the sum of the atmospheric pressure and the tissue pressure</u>.

The Atmospheric pressure AtmP (10 kg/cm² at sea level!) decreases with altitude and the Tissue pressure Ptiss varies with the surrounding structures, passive (fascia) and active (muscles).



2951- *Atmospheric pressure AtmP and Gravitational Hydrostatic pressure of water.*

The atmospheric pressure AtmP sea level is on average 1,013.25 hecto Pascals or 1 atmosphere. This is equivalent to 10 kg (10 m of water or 740 mmHg) which weighs on each cm² of our skin, which is counterintuitive because we do not feel it! It decreases with heat and altitude, which dilate the air, decreasing its density so its specific weight ρ (ρgh).

When we swim, the hydrostatic gravitational pressure GHSP of the water is added to the atmospheric pressure on the submerged part of our body. The sensation of feeling lighter in the water is also counterintuitive because it does not mean that we are lighter, but that the weight of the water is distributed over the entire surfaceof our body instead of being concentrated on the soles of our feet when we are standing out of the water. This illusion of lightness is increased by the fact that the water pushes us upwards with a force equal to the volume of water that our body has displaced (Archimedes). Thus, we only float if our specific weight is less than the water's, i.e., the volume of water corresponding to the part of our body immersed weighs as much as our whole body. This is also why we only float if we immerse a sufficient volume of our body.

Thus, the venous TMP increases when the atmospheric pressure decreases, thus with heat and altitude, and decreases on the part of the body that we immerse in water. The calibre of the veins varies like the TMP while the Intravenous pressure does not change! We can only imagine that the venous calibre reducing, the velocity increases v, reducing the static pressure p ($p + ½\rho v^2$) but in negligible proportion in front of the GHSP ρgh which does not change. Total pressure $Pt = p + ½\rho v^2 + \rho gh$ = Total pressure This is also the case of the restraint that decreases the TMP but does not significantly change the IVLP

In an airliner flying at 10,000 meters altitude, the air is compressed for a value equivalent to an altitude of 2,500m, which corresponds to a loss of1/4 of the atmospheric pressure, or 25 cm H^2O and increases the TMP accordingly. One compensates for part of this deficit by the compression and the activity of the valvulo-muscular pumps of the lower extremities (exercises in sitting or standing position).

2952 Extra-venous tissue pressure

The extra-venous tissue pressure TP is applied by the skin, muscles and fascia on the interstitial fluids, capillary walls, and venous walls. It is passive at rest and active, and therefore more marked during musculoskeletal movements.

It explains why the veins contained by fascia such as the trunks of the great and small saphenous veins are less dilatable than other superficial veins which do not have them. Muscles contained in inextensible aponeurotic spaces are more effective during their volume variations. This effect is amplified by non-elastic compression*.*

2953- Limb compression

Compression deserves to be considered, not only therapeutically, but also preventively, because of current living conditions. These conditions are, especially, the long immobile standing stations which maintain an excessive gravitational hydrostatic pressure and favours stasis which is thought to favour the destruction of the valve by inflammation,

External compression of the lower extremities reduces transmural pressure, which favours tissue drainage and reduces the calibre of the veins. *The main hemodynamic effect is often attributed to an increase in flow velocity and a reduction in stasis volume****. Certainly, the reduction in vain calibre increases flow velocity and reduces stasis volume, factors that reduce the risk of thrombosis. But the flow rate and intravenous pressure do not change! The more marked reduction in calibre in deep veins than in superficial veins is counterintuitive and may seem paradoxical****. However, it can be explained very well because superficial venous pressure must necessarily be greater than deep venous pressure to make its drainage possible.*

The mass, volume and compressibility (bulk modulus) of tissues must be considered for the transmission of pressure forces to the veins and tissue interstitial fluids around the capillaries, by any means of compression.

The transmission of the pressure complies the Laplace's law and the modulus of compressibility.

Thus, for the same transmission of pressure, compression must be stronger the larger the limb and the less compressive the tissue.

So, compression must consider the shape, volume, and compressibility of the limb tissues.

The distribution and homogeneity of compression varies depending on the external compression medium and the compression/elasticity of the leg.

29531- Homogeneous compression

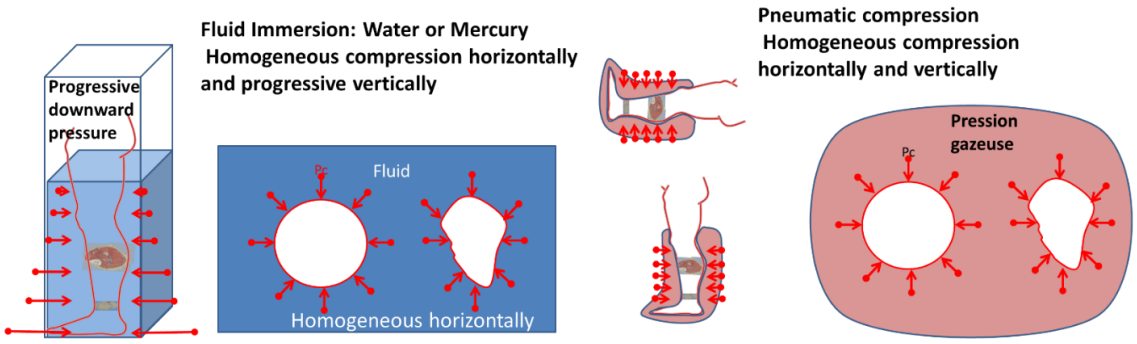
295311- Immersion in a liquid

. Immersion in a liquid (water, mercury) creates a homogeneous circumferential compression that progresses vertically from top to bottom proportional to height h and specific mass ρ (ρgh). Thus, this compression depends on gravity.

As we have seen, the sensation of feeling lighter in water does not mean that the body is lighter, but that according to Archimedes' law, the weight of the water is distributed over the entire surface of our body instead of bearing all of it on our feet when we are out of the water. In the same way, the superficial veins shrink in water not because the column of Hydrostatic Gravitational pressure GHSP of the venous blood is magically lighter than in the open air, but because the hydrostatic gravitational pressure of the water in the pool compresses them in proportion to the depth of their immersion.

295312- *Air-inflated sleeve*

An air-inflated sleeve exerts a compression by circular and homogeneous contact force independent of gravity, thus of the posture*. It can be uneven if the sleeves are staggered and inflated differently. When it is inflated and then deflated intermittently, it produces a pump effect like the valvulo-muscular pumps.*



29532- *Heterogeneous compression.*

The compression bandages elastic or inelastic by the contact force independent of the earth's gravity. It is heterogeneous by the spatial irregularity of the pressure force that it transmits.

The compressive bandages transmit a force of pressure P in accordance with the law of Laplace that establishes the fact "counterintuitive" that, for the same force of tension T, it is stronger when the radius r of the member is smaller. Laplace's law: P=T/r T=Pr. Therefore, bandaging with the same tension force transmits more pressure to the ankle than to the thigh.

The pressure transmitted is equal if the bandaged segment is perfectly circular. If the segment has an irregular surface, the pressure increases with the tension as the radius of curvature of each bumpy irregularity decreases (Laplace).

This transmission can be adapted by modifying the circular profile of the limb with pads that increase or reduce the pressure depending on the required pressure at a given point.

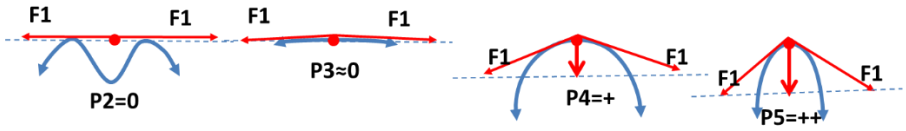
We can thus avoid compressing the arteries, especially the pedal artery, by affixing pads on each side that prevent them from being in contact with the bandage.

In all cases, the risk of ischemia must be feared and prevented by ensuring that the Doppler or plethysmography flow of the forefoot is equal to that which preceded the bandage at the end of the bandage and in decubitus.

LAPLACE's law
Pressure P against the skin
F: tension force F
w: width of the band
R: Radius of the cylinder Pc
Transmitted pressure = F/wR = F/R when w=1cm
P: hecto Pascal, F: Newton 1mmHg = 1,333 hector Pa = 1,359 cm H^2O = 0,00131 atm

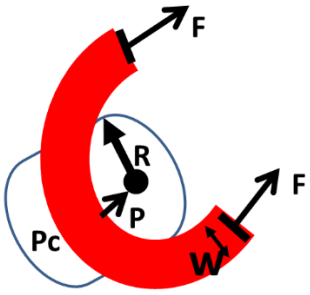
INHOMOGENEOUS COMPRESSION

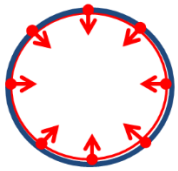
Bandage



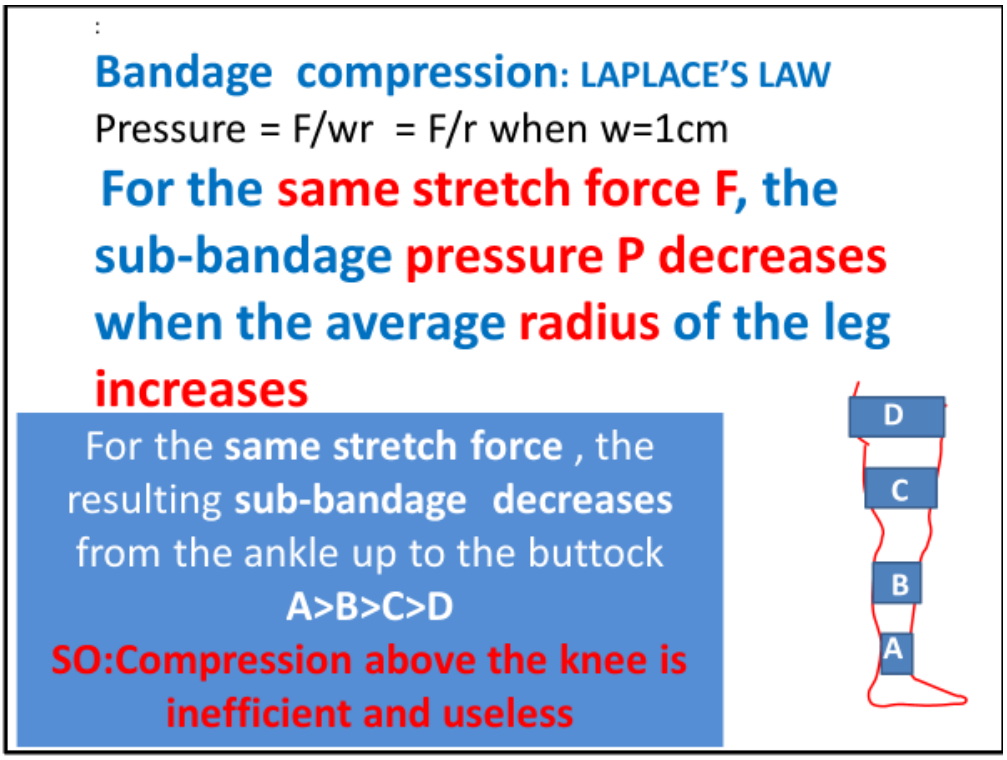
P5>P4>P3>P2: Pressures transmitted by the tension F according to R.

depends on the arc angle where the compression is applied





Circular: Homogeneous Transmission	Not Circular : Inhomogeneous Transmission



295321- Non-elastic compression

Non-elastic support is passive because it is a resistive force and does not exert any active pressure by itself.

The terms "support or constraint" are more appropriate for inelastic means because they are more suggestive of a passive resistive force, whereas the hysteresis of elastic "compression" is an active force until it returns to its resting length.

It simply returns the force of the pressure of the leg it contains. The pressure force transmitted to the muscle mass modulus is that provided by the muscles of the practitioner who tightened the band. When the bandage is elastic, the practitioner muscles have transmitted energy not only to the muscle mass modulus, but also to the bandage, which exerts a compressive force until it has returned to its resting length.

Thus, the non-elastic and non-stretchable bandage resists and returns the pressure of the limb. This happens especially during walking, when the contraction of the calf shortens its length, which increases the pressure-volume of its middle part*. In the same way, these pressure returns make the microcirculatory TMP vary like a massage that is rhythmically generated by walking. This is one of the reasons why* ***it is called "work pressure" where work is done by a force on an object to produce a certain displacement, in that case the force of the calf to tense the bandage".*** *So, the less the calf volume-pressure, the less the tension and the returned pressure. This is what we see during the day. Walking under support improves the "massage" of the microcirculation and the power of the calf pump, which drains part of*

the oedematous volume of the leg and reduces the work pressure. ***This loss of back pressure is avoided if the oedema is reduced before applying the non-elastic bandage.***

The passivity of the non-elastic bandage causes the counter pressure to decrease with the lateral intravenous pressure IVLP of the leg. This is particularly the case in supine position where it is reduced by the 80-90 mmHg of the gravitational hydrostatic pressure GHSP. This advantage, especially in subjects with arterial pathology with low leg arteries pressure, avoids decubitus ischemia. By contrast, elastic compression remains active in the supine position and can aggravate distal ischemia.

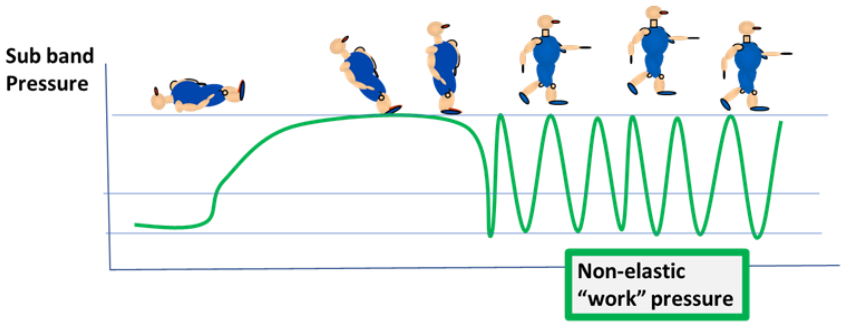
Taking all these parameters into account, we can indicate the optimal conditions for the application of non-elastic compression.

To avoid the loss of compression during the day, in the subject without arterial obliteration, the leg is strongly elevated in the supine position, which reduces IVLP accordingly. Moderate elastic restraint ≤ 20 mmHg is added. After 2 hours, the volume of the leg is reduced to the maximum. To avoid ischemia and its neurological consequences, a Doppler or Infrared plethysmography probe is used to check that the arterial flow of the forefoot is not reduced compared to that measured before the bandage was applied. The Doppler can be replaced by an infrared oximeter that measures pulse and oximetry. This measurement solves the problem of venous compression in case of associated arterial obstruction. This bandage can be kept on overnight and renewed only once a week.

Non-elastic compression

Non-elastic bandage is a passive support because it does not exert any active pressure. It is a resistive force to the pressure produced by limb when its volume tends to overwhelm the volume of the bandage.

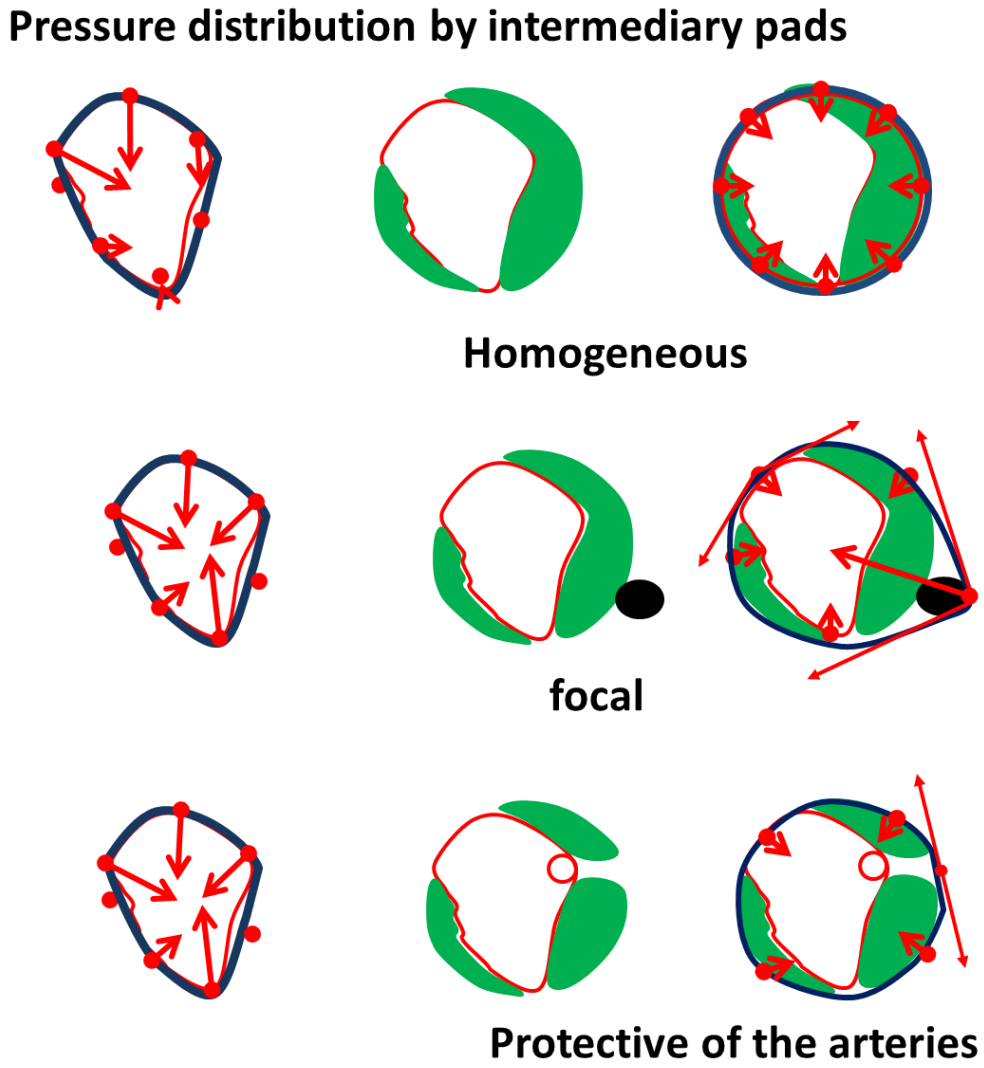
Thus, the non-elastic and non-stretchable bandage resists the volume/pressure of the limb and returns it. This happens especially during walking.



A disadvantage of non-elastic compression is that it does not adapt as well as elastic bands to the irregularities of the limb. This can be corrected by filling with pads the hollows beforehand to make the limb more homogeneous and circular. Otherwise, a compromise can be sought in the form of a semi-elastic bandage.

Particular indication for anelastic support:

<u>Light non-elastic support allows critically ischemic subjects to sit for long periods of time without the development of oedema. This position reduces pain and improves vascularization as shown by the oximeter.</u>



295322-- *Elastic band and stocking compression*

<u>Elastic compression is not passive but active</u> in the sense that, while they are being stretched, they receive a <u>potential shortening force</u> which is gradually released during relaxation (hysteresis) and becomes zero when it reaches its resting length in accordance with their young's modulus

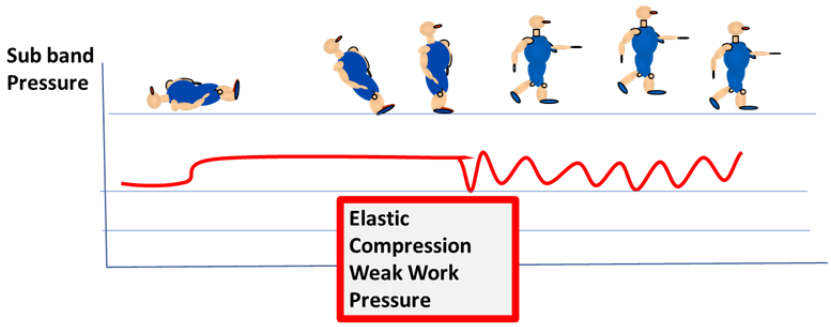
<u>This compression does not have the same therapeutic virtue as non-elastic compression</u> because, for a compression in the supine position equal to that of a non-elastic compression, <u>it will be less compressive when walking</u>. In fact, its elasticity allows the volume to increase, which reduces the "work" effect of non-elastic compression. In

addition, its permanent elasticity continues to act in decubitus position when the blood pressure is much lower than in standing position (reduced arterial GHSP) with the <u>risk of ischemia in case of associated arteriopathy.</u>

<u>The advantages of elastic support are its better conformability</u> to the irregularities of the limb's surface and its use in the form of stockings. This is why it remains preferable for all cases where the TMP is not too high, which is fortunately the most frequent case.

Elastic band and stocking compression

Elastic compression is not passive but active due to its potential shortening force (Hysteresis) and stores instead of resisting part of the pressure volume variations of the limb.

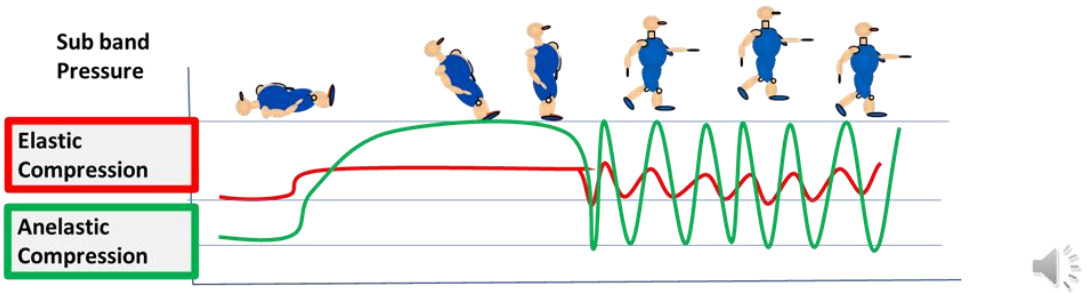


Anelastic and elastic compression does not have the same therapeutic virtue.

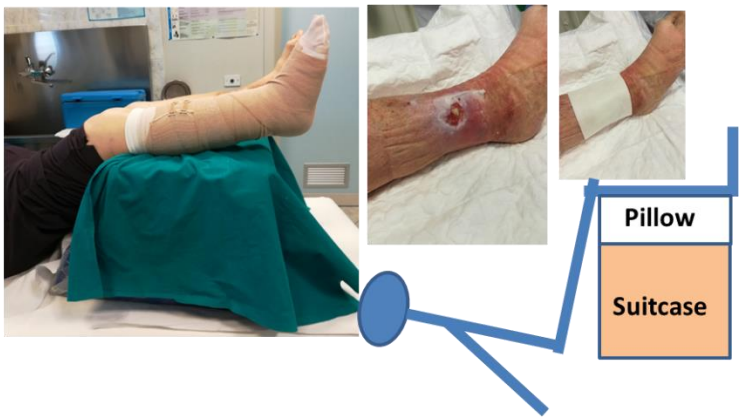
For a lesser sub-bandage pressure in lying position, anelastic bandage reduces more Transmural pressure than elastic bandage during standing and walking.

It reduces the risk of ischemia in lying position, which is essential in case of associated arteriopathy.

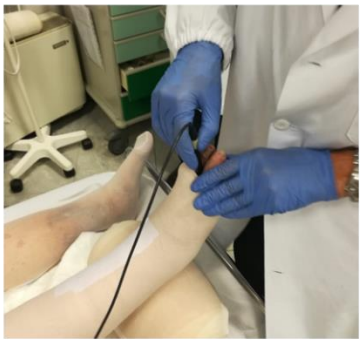
Nevertheless, due to its better conformability , elastic compression remains preferable for all the cases where the TMP is not too high, which is fortunately the most frequent case.



Non-elastic compression after 2 hours of elevation + light elastic compression



Distal arterial Doppler control



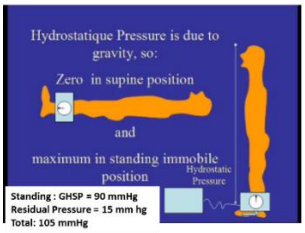
296- *Measurement of venous pressure.*

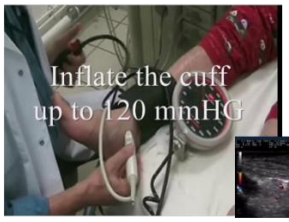
Venous pressure of the posterior tibial vein:

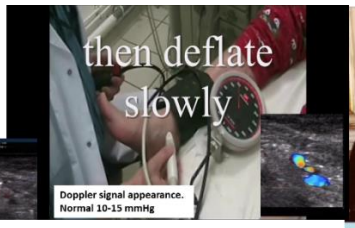
The venous pressure of the posterior tibial vein, measured by Doppler and tensiometer (sphygmomanometer), proposed by Mauro Bartolo.

Ref: 1- Bartolo M. Phlebodopplertensiometry, a non invasive method for measuring venous pressure. Folia Angiol. 1975; 25:199-203. 2-M. Bartolo. Non-invasive Venous pressure Measurements in Different Venous Diseases Angiology. Journal of vascular Diseases November 1983

It is the value displayed when the venous Doppler signal reappears. Its rationale is the same as that of the arterial pressure measurement in the same conditions. This value measures the total pressure and can be artefactually increased when the compressibility/elasticity of the ankle tissues is compromised by the surrounding sclerosis and hypodermitis. To avoid this artifact, the cuff should be placed on healthy tissue. The decubitus measurement specifically evaluates the residual pressure, thus the obstacles to flow, without considering the GHSP which is practically null in this position and nor the valvular incompetence which is inactive in decubitus

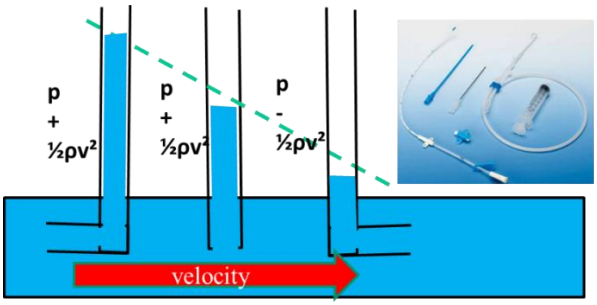






<u>Venous pressure can be measured by strain gauge plethysmography or by infrared.</u> The probe is placed on the foot or the big toe, on a subject in dorsal decubitus. Then the leg is raised vertically by the operator then lowered very slowly to the horizontal. The venous pressure is the value of the height of the foot when the venous filling signal appears.

The pressure measured by catheterization is the motive pressure when the operator faces the flow ($p + \frac{1}{2}\rho v^2 + \rho gh$), without the dynamic pressure $\frac{1}{2}\rho v^2 = 0$ when it is oriented towards the wall ($p + \rho gh$) and reduced by the dynamic pressure $\frac{1}{2}\rho v^2$ ($p + \rho gh - \frac{1}{2}\rho v^2$) when it looks downstream as shown by the Pitot tubes.



Venous pressure with the catheter
Total pressure Pt = $p + \frac{1}{2}\rho v^2 + \rho gh$
When the catheter faces the flow, it measures the Total Pressure $p + \frac{1}{2}\rho v^2$
When it is perpendicular to the flow, it measures the only lateral static pressure p.
When it is in the direction of the flow, the pressure is equal to $p - \frac{1}{2}\rho v^2$

Chapter 3

Each chapter includes some of the elements of the previous chapters and anticipates those of the following chapters.

31- The Starling model

32- The Glycocalyx model

33- Oedema, hypodermitis, ulcer.

331- Oedema is simply related to the excess of TMP that opposes fluid.

332- Hypodermitis is a chronic inflammation of the skin and subcutaneous tissue.

333- Venous ulcers

3- *Microcirculation Drainage and trophic disorders. Ulcers*

The essential function of the venous system is drainage, which is vital for the tissues. Drainage defects range from simple oedema to severe trophic disorders and ulcers.

In the most advanced forms, the accumulation of waste products and toxic catabolites *leads to dermal-hypodermal inflammatory reactions, cells necrosis, and ulcers aggravated by superinfections****. Inflammation, necrosis, and superinfection interact and aggravate each other.***

Local treatments of complications have ***transient successes with recurrences, if the hemodynamic cause is not corrected.***

The clinical presentations of venous insufficiency are essentially due to drainage reductions which are always due to an excess of Transmural pressure (TMP). *Excessive intravenous lateral pressure (IVLP) is related to obstructions and/or valvular incompetence. Extra venous pressure (EVP) defects are usually related to the decrease of the atmospheric pressure AtmP.*

Living conditions can also be responsible for an excess of TMP without anomalies of the venous system. *The PHHSG remains unfractionated for too long during prolonged standing or sitting with an excess of superficial motive pressure in hot environments and a deficit of extra venous pressure EVP at altitude or in airliners****.***

Leg support and elevation are effective, non-invasive, and preventive and curative hemodynamic treatments. *They act directly on the microcirculation, regardless of their*

effect on the veins. Therefore, compression should be focused on the tissues that are suffering and ***the elevation of the legs the highest possible.***

Surgical or endovenous destructive treatments are not hemodynamic *because they correct shunts by means that* ***secondarily compromise drainage and destroy potential vital venous grafts****.*

The CHIVA treatment treats shunts and restores dynamic fractionation of the gravitational hydrostatic pressure, without destroying the overloaded veins*, thus preserving tissue drainage. It splits the gravitational hydrostatic pressure column, disconnects the Closed CS and Open ODS shunts while preserving tissue drainage and avoids disconnecting the compensatory open vicarious shunts****.***

Drainage is also lymphatic.

Lymphatic vessels are specialized in the drainage of tissue products and macromolecules that cannot pass through the capillary wall.

Not only capillaries and lymphangions collaborate in drainage, but they interact. This interaction can be compensatory or aggravating depending on the filtering quality of the capillary walls and on the distribution of macromolecules between the plasma and the tissue interstitial fluid. This distribution determines the oncotic pressure gradient OPG directed towards the capillary. ***Thus, drainage is only possible if the OPG is greater than the TMP.***

31- Starling's model.

Although discussed, Starling's model remains relevant in practice.

According to Starling, the fluid passes from the capillaries into the interstitial spaces at the arterial portion of the capillary where the hydrostatic pressure is higher than the oncotic pressure. Then it returns into the venous portion of the capillary where the hydrostatic is lower than oncotic pressure gradient. So, drainage depends on 7 factors:

Capillary venous drainage (CVD) depends on 6 factors to which we add the Transmural pressure TMP = (IVLP - EVP)

Transmural pressure TMP, oncotic pressure and capillary permeability constitute the determining triad for exchanges between intra and extravascular compartments*.*

A: Transmural pressure TMP

1 - IVLP (Intravenous Lateral pressure) corresponds to the capillary hydrostatic pressure CHS.

2- Interstitial hydrostatic pressure IHP which depends on the tissue environment and the atmospheric pressure, thus on the Extra-Venous pressure EVP

3-The transmural pressure TMP = (IVP - EVP) is the mechanical pressure resulting from the difference between Hydrostatic Capillary Pressure HCP and EVP.

B: Oncotic pressure Gradient OPG

4- Capillary oncotic pressure COP

5- Interstitial oncotic pressure IOP

C: Capillary permeability

6- Filtration coefficient of the capillary wall Fc that is the mechanical filtration capacity of liquids by the capillary wall. It is a constant of capillary permeability (Product of capillary surface and capillary hydraulic conductance).

7- Reflection coefficient Rc (0 to 1) varies according to the ultrafiltration capacity of the semi-permeable capillary wall to retain large molecules and allows water and small molecules to pass. With a value between 0 and 1, it corrects the oncotic pressure gradient according to the parietal permeability to protein versus liquid.

DVC = FC ([PHC - PHI] - Rc [POV- IOP]) or DVC = Fc ([TMP] - Rc [POV- IOP]) (in French)

CVD = Fc ([CHP - IHP] - Rc [COP- IOP]) (in English) Cvd = Fc ([TMP] - Rc [COP- IOP])

Positive CVD corresponds to capillary transfer to the tissues. Negative, it corresponds to drainage of interstitial fluid to the capillary.

On the venous side of the capillaries, the Transmural pressure TMP is the mechanical pressure that pushes the fluids and components of the capillaries towards the interstitial liquid spaces of the tissues, thus against the direction of drainage.

We see in this formula the relative part of each of the 7 factors to be considered to understand and correct venous drainage defects.

We see, for example, that venous drainage of fluids and small proteins is favoured by

the increase of:

- the filtration coefficient Fc,

-the interstitial hydrostatic pressure IHP, itself dependent on EVP factors,

-the reflection coefficient Rc, and

-the capillary oncotic pressure (POC) of the plasma and

by the decrease of:

the capillary hydrostatic pressure PHC

the TMP and

the oncotic pressure of the interstitial tissue fluid IOP.

The inverse evolution of one or more of these 7 factors will degrade the quality of the tissues, particularly the skin.

We can also see how <u>insufficient drainage of macroproteins by the lymphatics increases the Interstitial Oncotic pressure IOP and reduces venous drainage.</u>

Conversely, any <u>deficit in venous drainage overloads the lymphatic system</u> not only with fluid but also with products of tissue suffering and inflammation resulting from insufficient venous drainage.

The capillary filtration coefficient Fc and the reflection coefficient Rc depend on the quality of the capillary walls.

<u>The resulting drainage filtration pressure = 18 mmHg (TMP) - 25 mmHg (oncotic pressure) = 7mmHg.</u>

When the TMP is too high and prevents drainage, whatever the cause, treatment based on this law is effective. <u>It is a matter of reducing the TMP to values lower than the Oncotic pressure gradient</u>, <u>either by reducing the Intra-Venous pressure or by increasing the Extra-Venous pressure</u>

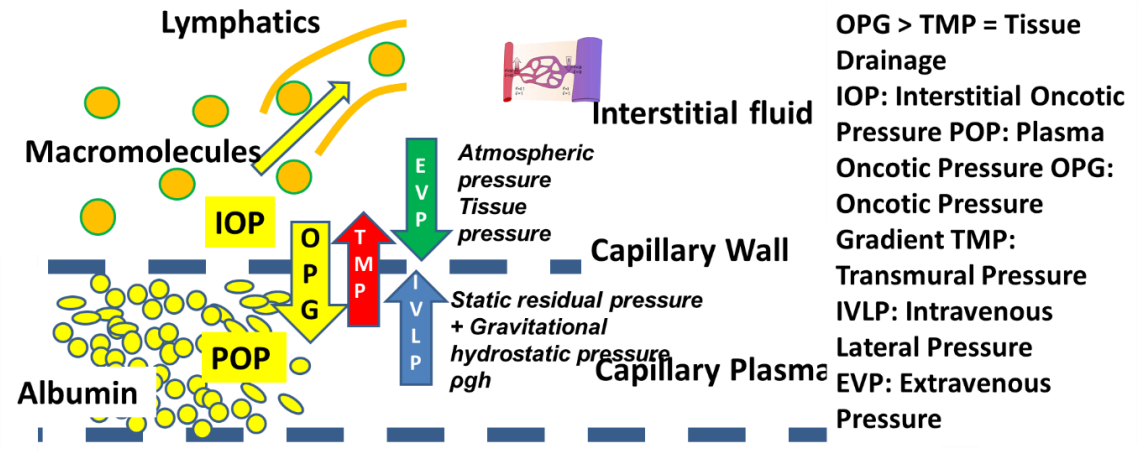
TMP and OPG.

<u>When the OPG is too low</u>, or even reversed, the TMP predominates, and drainage is reduced.

OPG is reduced either by hypoproteinaemia or by accumulation of products and macromolecules in the interstitial fluid (inflammation and/or lymphatic insufficiency). Inflammation is most often responsible for so-called "veno-lymphatic" oedema. On the other hand, an increase in POP can compensate for an excessively high TMP.

<u>Lymphatic insufficiency is indirectly aggravated</u> by venous insufficiency. The accumulation of non-drained products leads to an inflammatory reaction (hypodermitis, ulcer), thus to lymphatic overload

.



32- *The Glycocalyx model.*

Starling's model is being questioned in favour of the Glycocalyx model, which is schematically as follows:

Here is what I understand. The glycocalyx-clefts model defines the glycocalyx as a semi-permeable layer with large and narrow pores, applied to the endothelial cells and the intercellular slits of the capillaries where the oncotic pressure would be lower than in the interstitial liquids. The capillaries would no longer be divided into arterial and venous sides, but only arterial because they would let fluids and proteins pass to the tissues but would not reabsorb them. The drainage of fluids, proteins, cellular debris, and nutrients from the interstitium prevented by the hydrophobicity of the glycocalyx would be done only by the lymphatics.

Levick and Michel showed in animals that fluid passing from the arterial end of the capillary into the interstitium does not return to its venous end. This would be since the hydrophobic glycocalyx, modifies the pressures and prevents the fluid from being reabsorbed by the capillary. Therefore, the fluid cannot return to the venous system. If the low-pressure lymphatic system that becomes the drainage site for fluid, cellular debris, and nutrients from the interstitium, ***why and how does excess intravenous pressure cause fluid retention in the tissues in the absence of lymphatic insufficiency?***

The Glycocalix model still needs to be confirmed in all respects, and Starling's model remains valid for describing the microcirculation of the lower extremities, as it remains consistent with hemodynamic and clinical data.

33- *Oedema, hypodermitis, ulcer.*

Oedema, hypodermitis and ulcers are associated with defects in tissue drainage, mostly due to excess transmural pressure TMP.

331-Oedema is simply related to TMP excess that opposes fluid evacuation.

Nevertheless, it is aggravated by chronic or transient abnormalities of capillary wall permeability.

332- Hypodermitis is a chronic inflammation of the skin and subcutaneous layer secondary to the accumulation of waste products and catabolites, as shown by hemosiderin deposits.

It also reflects tissue fragility to trauma and infection.

It usually starts in the lower third of the leg, opposite a re-entry perforator, in a cutaneous area in direct contact with fascia and bone, without a muscular intermediary. This particularity combines 3 favourable conditions.

- Distality increases the height of the column of gravitational hydrostatic pressure.

- The hemodynamic trauma of the re-entry thrust (water hammer) of the closed shunt

- the escape point outwards thrust due to deep venous obstacle is increased by the proximity of the calf pumps.

This is a particularly fragile area and is known to be difficult to heal, even in healthy subjects. It is the starting point for ulcers.

This last point is remarkable. *A a matter of fact, there is no starting point of venous ulcer at the calf or thigh. Though distal, the foot is not affected by venous ulcers (except for certain malformations). This can be explained by the thickness of the skin of the sole and the presence of perforators drained by Léjars pump compressed by the body weight when support when walking.*

333-Venous ulcers.

An ulcer is a wound that does not heal in the usual time. There are many causes: arterial, venous, capillary, infectious, paraneoplastic, neoplastic.

As we have just seen, the starting point of the venous ulcer is usually located at the ankle, where drainage conditions are the most precarious, opposite the re-entry perforators.

It then extends mainly due to superinfection.

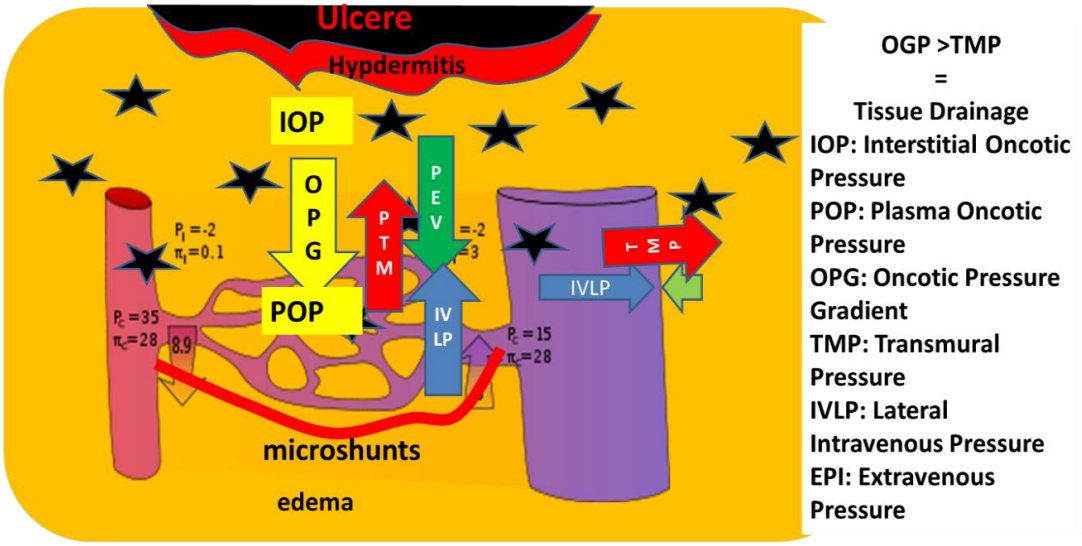
It is also remarkable that the bleeding is "red" with a normal or even elevated venous oximetry, which could be due to an opening of the micro shunts that steal the capillaries, so easing necrosis.

Note again that most large perforators located at the bottom of the ulcers are re-entry are not in themselves the cause of the ulcer, but only the point of excessive pressure/flow overload of the closed shunts. Ablating them leaves behind an obstacle to the physiologic drainage of the damaged tissues. Therefore, CHIVA disconnection of the escape point , spares the re-entry perforator, suppresses the flow overload and the water hammer, while it preserves the drainage, which ensures a good and lasting healing of the ulcer.

Ref: P. Zamboni and all: Minimally Invasive Surgical management of primary venous Ulcer vs. Compression Eur J vasc Endovasc Surg 00,1 6 (2003)

Discussions and controversies on the physiopathology of venous ulcers (peri-capillary fibrin sleeve (Browse and Burnand theory, release of inflammation mediators by white blood cells.), have no practical meaning for the treatment, when we understand that any venous ulcer occurs only if the TMP is excessive and heal if the TMP is normalized and the infectious complication is effectively treated. By the way, the inflammatory released by the incompetent veins is NOT due to the reflux direction of the flow, as demonstrated after CHIVA shunts disconnection, but to the flow- pressure overload. Ref: P. Zamboni and al.

Oscillatory flow suppression improves inflammation in chronic venous disease journal of surgical research s e p t e m b e r 2 0 1 6 (2 0 5) 2 3 8e2 4 5.



Drainage failure due to Transmural Pressure TMP Excess
Edema, accumulation of toxic catabolites, Hypodermitis
Opening of micro-shunts

Chapter 4

Each chapter includes some of the elements of the previous chapters and anticipates those of the following chapters.

4- The venous network: calibres, walls, drainage hierarchy, valvular incompetence, anatomical and functional topography of the shunts.

41-Calibre and intrinsic hemodynamic properties of the venous wall

411-The compliance (inverse of elasticity) is the elongation capacity of the wall.

412-T-Tension T is the stretching force transmitted by the transmural pressure TMP as a function of vessel radius r.

413-Hooke's law and Young's modulus describe the variation of the elastic compliance of the vessel.

414-Visco-elasticity delays the response time of the calibre

415- Parietal shear stress is the applied force F that moves the blood sheet tangentially

416- Vasomotricity:

417- Venous remodelling

418- The reservoir effect decreases the intravenous lateral pressure IVLP

42- Hierarchy of the networks and drainage

43- Anatomy of the key hemodynamic, diagnostic, and therapeutic points of the venous network

431-Abdominal-Pelvic Veins

4311-Left Renal Vein, Nutcracker Syndrome NTS Syndrome, Left Gonadal Vein and Varicocele

4312- Varicocele is a dilatation of the left gonadal vein

43121- Varicocele reflux due to open deviated shunt ODS

43122- Non-refluxing compensating varicocele (non-refluxing OVS).

43123-Left common iliac vein and May Thurner syndrome or Cockett's syndrome

43124-Pelvic veins and pelvic escape points.

431241- Parietal pelvic veins

4312411- Gluteal veins.

4312412- Obturator vein.

431242- Parietal pelvic escape points.

4312421-The obturator point

4312422-Superior gluteal point

4312423-Inferior gluteal point

4312443 - Visceral pelvic vanishing points

4312431-Internal Pudendal Vein

4312432-The vein of the round ligament of the uterus

4312433-External hemorrhoidal vein and hemorrhoidal disease ("haemorrhoids")

431244- Pelvic visceral escape point

4312441-The perineal point

4312442-The clitoral point

4312443-The inguinal point

432- Veins of the lower extremities

4321- Femoral veins

43211- The superficial femoral vein, single or double

43212- Congenital stenosis of the superficial femoral vein

4322- surfaceveins of the lower extremities

43221-Saphenous veins and vein of Giacomini

432211- Great saphenous vein

43222-Small saphenous vein

43223- Giacomini's vein

4323-Valves

43231-Complete closure of the valve occurs after a brief time of reflux.

43232- Valvular incompetence

4324- Connections between the different networks

43241- Saphenofemoral SFJ and saphenopopliteal SPJ connections.

432411-SFJ and SPJ incompetence are points of escape from closed shunts.

432412--SPJ is usually found in the popliteal fossa between the gastrocnemius muscles.

43242- Perforators

43243- Anastomoses

43244-Escape points

432441- Open vicarious shunts OVS

432442- Leak points of closed shunts

432443- Escape points opened deviated shunts by the ODS

432444- Mixed escape points

432445- Re-entry points

4325. Shunts. Detailed classification.

43251-Superficial shunts

432511- Closed shunts CS.

432512- Shunts 0 without diastolic escape point.

432513- Combined superficial diastolic shunts.

432514- Systolic shunts OVS

432515- Mixed shunts: MS

432516- Classification of deep diastolic reflux

43252. Practical and theoretical shunts

4- The venous network: calibres, walls, drainage hierarchy, valvular incompetence, anatomical and functional topography of the shunts.

41-*Calibre and intrinsic hemodynamic properties of the venous wall.*

The calibre of veins depends on the physical characteristics of their wall (Hook's, Young's and Laplace's laws, Compliance) subjected to transmural pressure TMP but also to shear stress.

The venous wall is made up of 3 layers: intima, media, and adventitia, where elastic structures dominate the muscle layer, *which differentiates it from the arterial wall.*

This explains the high compliance C *which confers* *great capacitive properties* *to the venous bed, which contains 70% of the total blood volume.* ***The pressure varies little with respect to the volume variation thanks to the reservoir effect which dampens it.***

The wall is fed by the vasa vasorum. Its innervation participates in the regulation of its tension. ***This could explain why harvesting the great saphenous vein with its surrounding tissue improves their graft quality of the coronary bypasses.*** **Ref:** 1-No touch technique of saphenous vein harvesting: Is great graft patency rate provided? Papakonstantinou NA J Thorac Cardiovasc Surg. 2015 Oct;150(4):880-8. doi: 10.1016/j.jtcvs.2015.07.027. Epub 2015 Jul 15.

2-The no-touch saphenous vein for coronary artery by-pass grafting maintains a patency, after 16 years, comparable to the left internal thoracic artery: A randomized trial. Samano R1, ClinicalTrials.gov NCT01686100.Copyright © 2015.

411-*Compliance C (inverse of elasticity) is the elongation capacity of the wall.*

It is proportional to the variation Δ in the calibre Ca of the vein as a function of the Tension T (force applied to stretch), Compliance C = ΔCa/T.

412-*Tension T is the stretching force transmitted by the Transmural pressure TMP as a function of the radius r of the vessel.*

It responds to Laplace's law. T= TMP /r. ***This explains the counterintuitive effect whereby the smaller the calibre of a vein, the greater the pressure required to dilate it and vice versa. Similarly, to obtain the same venous compression, the thigh bandage must be tightened more than the ankle bandage.***

413 *Hooke's law and Young's modulus describe the variation in compliance*

according to the degree of wall stretching within characteristic limits of each type of wall.

Thus, the dilatation of the calibre does not vary linearly but in the form of a long, sloping S.

414-*Visco-elasticity* ***delays the compliant response of the calibre to pressure by creep Cr, and then maintains the achieved calibre even if the pressure drops a little by Relaxation R.***

1-The delayed volumetric response Q to the transmural pressure TMP is creep Cr.

2- Relaxation R is the phenomenon by which the transmural pressure TMP required to reach the venous calibre C, is then lower to maintain it during the following relaxation R.

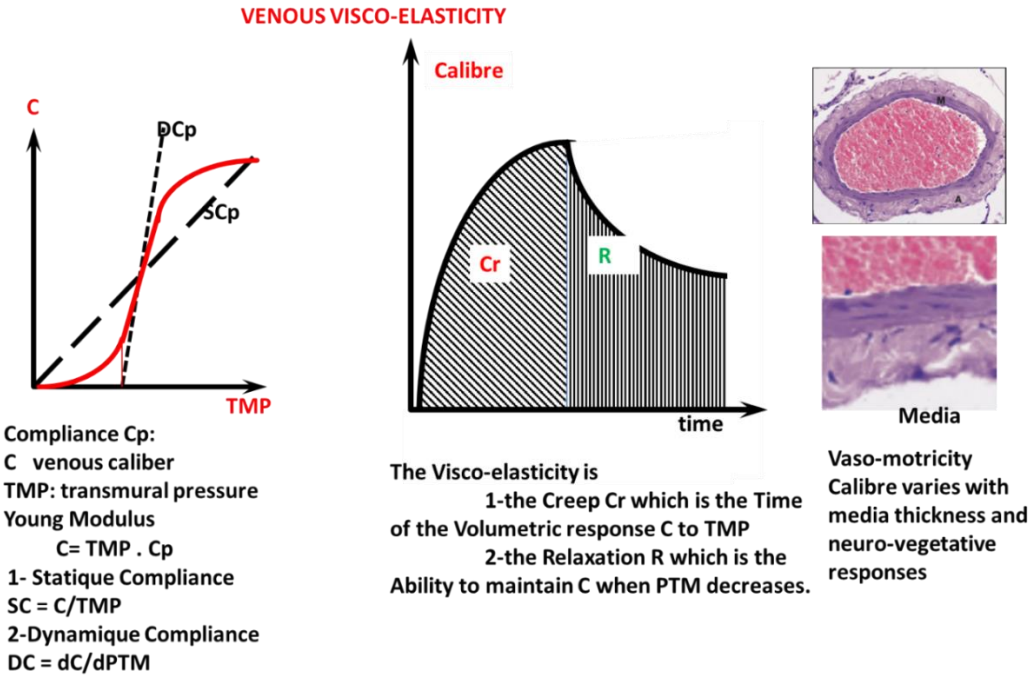
415- *Parietal shear stress is the applied force F* ***that mobilizes tangentially to the venous wall, in addition to forces applied perpendicularly to it, such as the Intravenous lateral Pressure IVLP of the Transmural Pressure TMP.***

*The resulting velocity and deformation of the **boundary blood layer** depend on its viscosity. This tangential friction exerts a stress on the intima which is minor in the laminar regime and becomes very important in the turbulent regime.*

In addition to their mechanical effects, these stresses trigger chemical, neuro-hormonal and structural reactions in the walls, particularly in varicogenesis.

416- *Vasomotricity:*

***The calibre of the veins varies according to the constitution of its muscular media and its responses to neuro-humoral solicitations** such as in thermoregulation and hormonal solicitations such as during pregnancy, the effects of which on the appearance and/or evolution of varicose veins of the pelvis and lower extremities are well known.*



Compliance Cp:
C venous caliber
TMP: transmural pressure
Young Modulus
C= TMP . Cp
1- Statique Compliance
SC = C/TMP
2-Dynamique Compliance
DC = dC/dPTM

The Visco-elasticity is
1-the Creep Cr which is the Time of the Volumetric response C to TMP
2-the Relaxation R which is the Ability to maintain C when PTM decreases.

Vaso-motricity
Calibre varies with media thickness and neuro-vegetative responses

417- *Venous remodelling*

Venous remodelling is the matching of calibre to transmural pressure (TMP).

When the TMP rises for long periods, the calibre gradually increases as the muscular media thickens (equivalent to Starling's law for the myocardium) until it stabilizes when it reaches a section that, by reducing velocities, sufficiently decreases parietal stresses and turbulence while maintaining flow. Conversely, a previously dilated vein with a thickened wall, such as the great saphenous vein subjected to excess TMP, will see its calibre and wall thickness

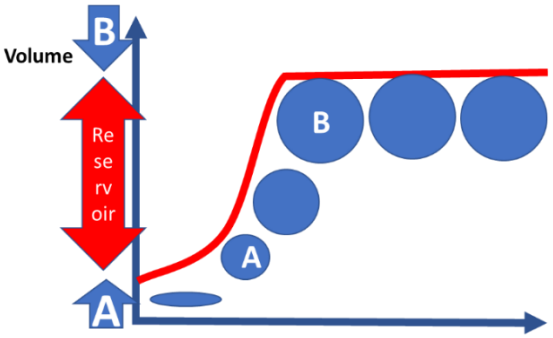
*progressively reduced in a few weeks when the constraints have been corrected by the reduction of flow and pressure**. This is notably the case after the CHIVA treatment where the regression of the calibre of varicose veins to normal requires time for remodeling. This demonstrates how the control of TMP and circulatory regimes corrects the biological disorders secondary to hemodynamic disorders:*** DELFRATE R.: Thanks to the CHIVA strategy may the histoarchitecture of great saphenous vein-sparing, make it suitable as graft for bypasses Veins and Lymphatics 2019; volume 8:8227

418-*The Reservoir Effect lowers Intravenous lateral pressure IVLP as long as the passive and active elastic compliance of the venous walls can offer little resistance to the increase in veins volume (according to the pressure/volume/resistance ratio).*

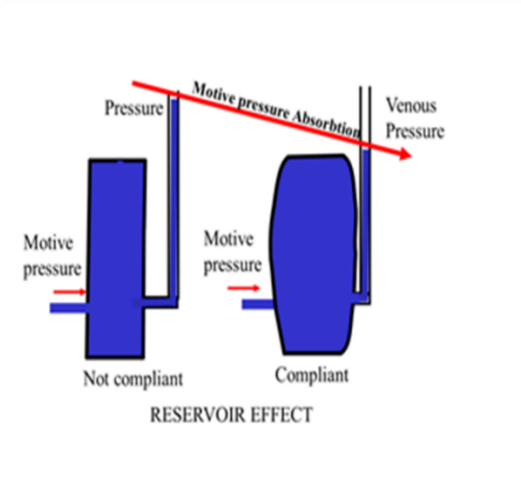
Thus, the reservoir effect reduces TMP, jerks and allows preloading of the right ventricle at quasi-constant pressure and "on demand" flow.

Contrary to what may be claimed, varicose veins are passive, regardless of their calibre, and therefore do not aspirate reflux by "reservoir effect", any more than a glass aspirates water from the bottle that fills it.

Varicose veins are overloaded veins, which are subjected to a shunt effect, of which they are the victims and not the cause.



Reservoir effect ensures a volume and pressure according to the needs of the right heart preload: The venous pressure varies little for a large variation of volume progresses thanks to the compliance between the limits A and B of viscoelasticity particular to each vein according to its histological structure
Limits of the reservoir effect:
A: Minimum volume
B: Maximum volume



42- *Hierarchy of networks and drainage*

I have classified the venous networks of the lower extremities into N1, N2, N3, N4 (for original R1, R2, R3, R4 in French) which drain into each other according to a precise physiological hierarchy, running throughout 4 specific compartments delimited by facias.

Ref :**1) FRANCESCHI C. (1988) Théorie et Pratique de la Cure Conservatrice et Hémodynamique de**

l'Insuffisance Veineuse Ambulatoire, Précy-sous-Thil: L'Armançon.2 FRANCESCHI C.: The conservative and hemodynamic treatment of ambulatory venous insufficiency Phlebologie. 1989 Nov-Dec;42(4):567-8.

It is not the reflux in a vein but the drainage hierarchy inversionwhich indicates the hemodynamic pathology. Restoring it is the hemodynamic rational therapeutic response, whatever the flow direction in each network.! So, reflux in a superficial vein is not pathologic if it connects to the other networks according to the physiological hierarchy. Moreover, a normal direction may be pathologic if it connects contrary to the physiologic hierarchy. We will see below many examples. So, normal flow direction (antegrade) or reflux is not, contrary to the still current mistake, pathologic or not IF the source and the goal of the flow are not identified!

N4, N3 and N2 drain superficial tissues, i.e., the skin for the most part, into the N1 network.

"Phlebosomes" are the drainage territories of a vein. Thus, the occlusion of a vein will impair the drainage of its phlebosome

The venous network is made up of successive hierarchical conduits.

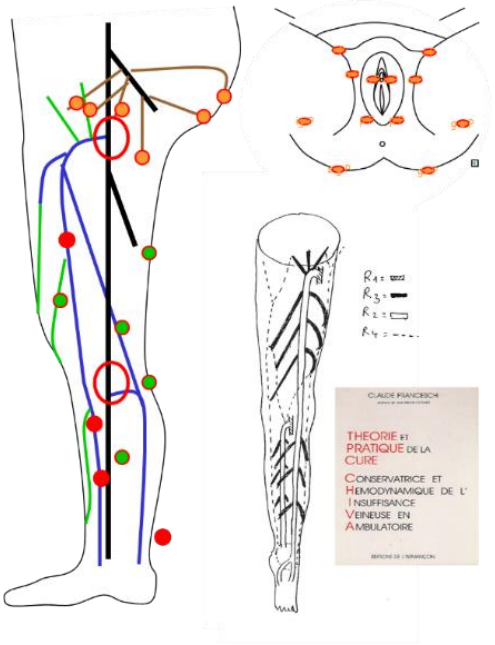
Intradermal venules fed by the microcirculation are drained by the N3 network.

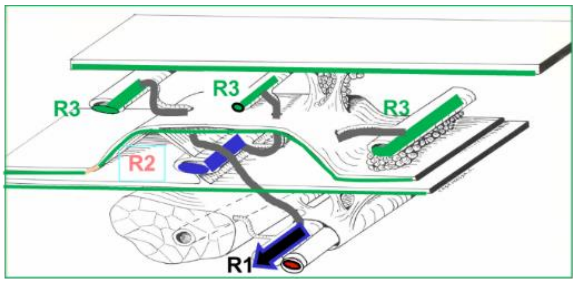
N3 network, runs inside the supra-fascial compartment, *connects to the N2 network by junctions or into the N1 network via perforators.*

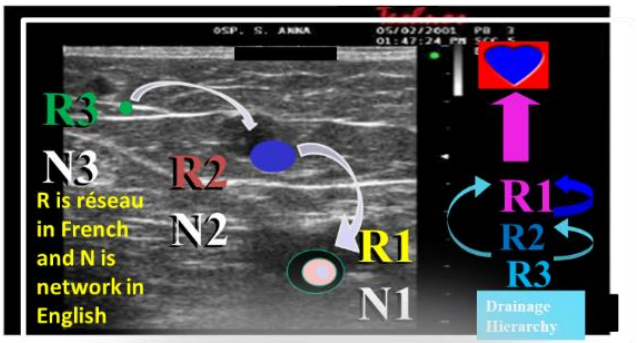
The N2 network is in a compartment formed by a fascial separation. *It consists of the trunk of the Greater saphenous Vein (GSV), which can be recognized in the "Egyptian eye" delimited by the fascial separation of the thigh, the Giacomini's vein and the small saphenous Vein (SSV). It does not directly drain the microcirculatory units, from which it receives blood via the veins of the N3 network.* ***It drains into the deep N1 network via perforators, the saphenofemoral junction SFJ and saphenopopliteal junctions SPJ. Giacomini's vein joins the arch of the small saphenous vein SSV to the trunk of the great saphenous vein GSV, which drains into N1. It can also drain directly into N1 via a perforator. The calibre of these veins varies greatly from one subject to another.*** *They are sometimes partially aplastic* ***but without any hemodynamic pathological impact. It is therefore not the calibre alone that can be considered pathological***

N4 networks connect segments of N2, N2>N4>N2. N4 Longitudinal N4L connects 2 levels of the trunk of the great saphenous vein and Transversal N4 N4T connects the trunk of the great saphenous vein with that of the small saphenous vein.

The N1 network consists of all deep veins located in the subfascial compartment. It drains superficial tissues indirectly via N2 and N3. Its flow drains all tissues and muscles towards the heart (cardiopetal) .





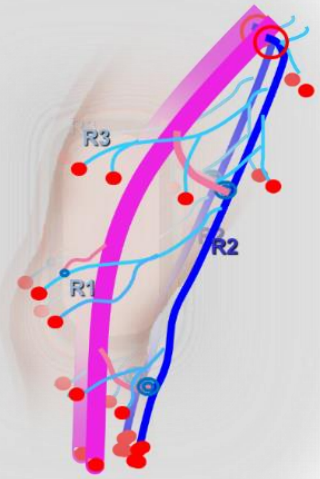


Potential escape points

- **Saphenofemoral**
- **Saphenopopliteal junction**
- **Pelvic leakage points**
- **Numerous perforators**

Networks in 4 hierarchical levels of drainage

- **R1, R2, R3, R4 according to their topography and hemodynamic function**



This hierarchy of drainage, N3>N2>N1 (R3>R2>R1) and N3>N1 (R3>R1), is functionally physiological so that it does not matter which direction the flow in the vein of a network is, if it does not contradict the hierarchy. For this reason, a reverse flow in a vein, called reflux, does not necessarily have pathogenic significance if it respects the hierarchy.

A retrograde flow ("reflux») in a segment of the trunk of a great saphenous vein N2 draining into N1(N2>N1) is not pathogenic but physiological if it does not receive flow from N1. It is pathological if it receives a flow from N1, thus in the opposite direction to the hierarchy: N1>N2>N1.

On the other hand, a N2 or N3 flow can be of normal orthograde (antegrade) direction but pathological. This is the case, for example, of the epigastric vein flow normally descending

from an N3 towards the arch of the great saphenous vein but which <u>becomes pathological without changing direction when it is overloaded by N1</u> pelvic blood via a N1>N3>N2>N1 <u>pelvic escape point</u>. This is also the case for the trunk of the <u>great saphenous vein</u> N2 whose antegrade ascending flow, therefore <u>not refluxing, is pathological</u> because it is overloaded by a systolic escape point N1>N2 to by-pass a deep obstacle N1: N1>N2>N1 = Open Vicarious Shunt OVS.

Thus, contrary to what is still too often the case, <u>it is not the direction of the flow recorded in the trunk of veins N1, N2 or N3 that determines venous insufficiency</u> . I call true pathological reflux, any drainage contrary to the physiological hierarchy. It occurs when the incompetence of communications between networks causes abnormal overload of one network by another superficial one. N2>N3, N1>N2, N1>N3.

These hierarchy inversions occur at <u>junctions or perforators that we call EP escape points</u>.

These hierarchically reversed <u>flows N1>N2, N2>N3, N1>N3 then drain into N1 by crossing what I have called the RP re-entry points</u> (saphenofemoral and saphenopopliteal junctions, perforators).

<u>The pathophysiological relevance of hierarchical flow inversions changes according to the conditions of their occurrence:</u>

at rest,

during systole or diastole of:

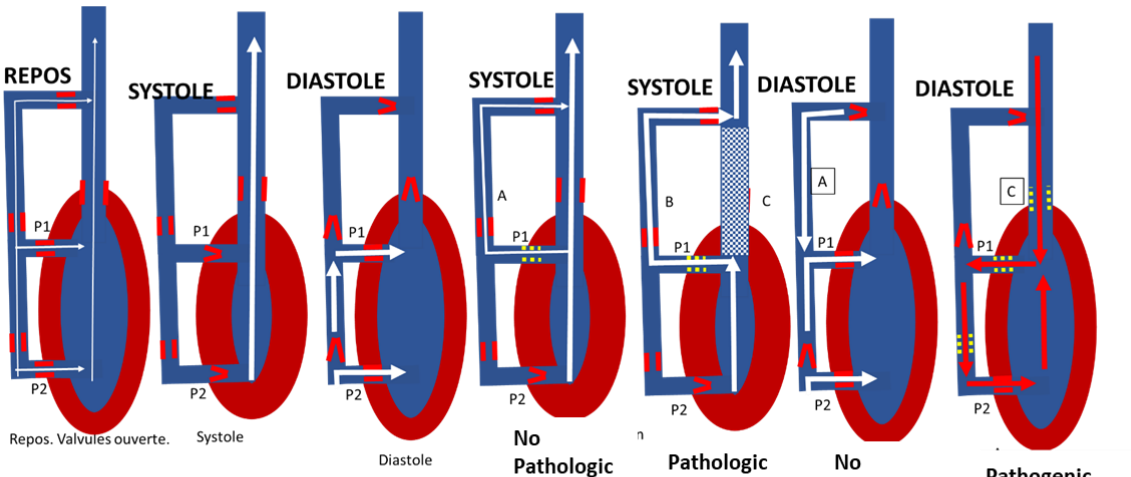
heart

thoracoabdominal pump

and especially valvulomuscular pumps.

Competent valves and perforators

Pathogenic and non pathogenic incompetents valves and perforators



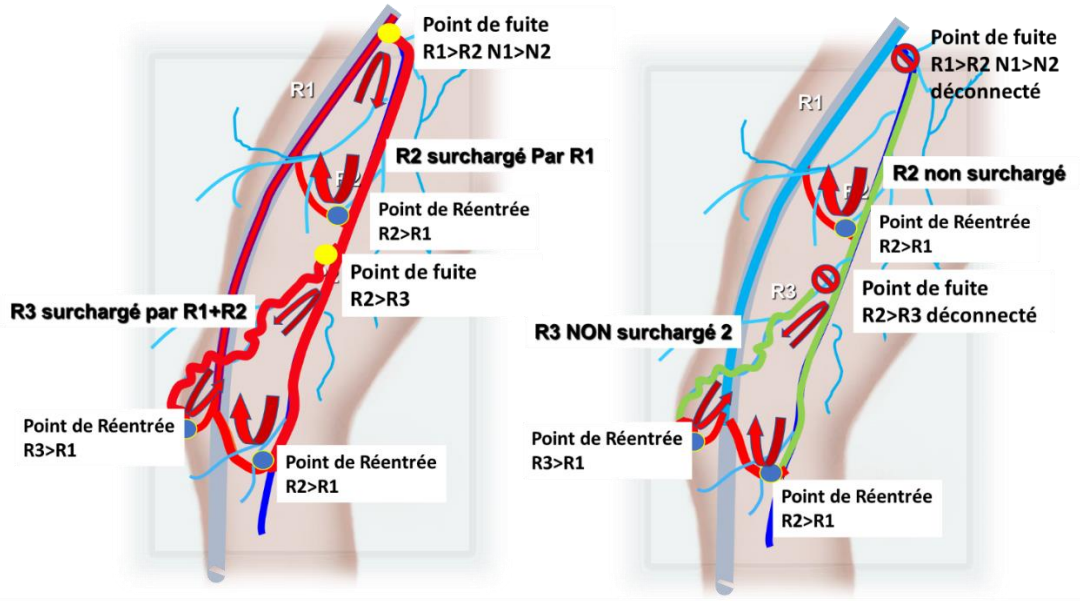
Perforantes et Valvules continentes

No Pathologic systolic reflux

Pathologic systolic reflux OVS Perforator Escape point

No Pathogenic Saphenous vein reflux : no hierarchical inversion systolic

Pathogenic reflux deep veins into saphenous: Hierarchical inversion



French into English translation: R, Point de réentrée is reentry point, point de fuite is escape point , surchargé is overloaded , déconnecté is disconnected.

Example of N1>N2>N1 closed shunt + N2>N3>N1 Open deviated shunt overloaded by hierarchic inversion . CHIVA Shunts disconnection, achieves R2 and R2 no more overloaded reflux , so no longer pathogenic. N3 and N2 collapse to normal caliber. We call them shunt 0.

43- *Anatomy of hemodynamic, diagnosis, and therapeutic key points of the venous network*

The anatomic and topographic variability of deep and superficial veins creates hemodynamic conditions that require specific diagnostic and therapeutic procedures.

431-Abdominal-Pelvic Veins

4311-Left Renal Vein, Nutcracker Syndrome NTS syndrome, Left Gonadal Vein and Varicocele.

Nutcracker Syndrome (NTS), The left renal vein may be compressed by the superior mesenteric artery against the aorta. This stenosis is anatomically very frequent, but it is not necessarily pathogenic nor hemodynamically significant. *When it is hemodynamically significant, it can be compensated by the Reno-azygo-lumbar system and/or by a reflux in the left gonadal vein (ovarian in women and spermatic in men) called varicocele. The horizontal supine posture can cause this stenosis.* ***For this reason, its pathological character can only be affirmed if it persists in a half-sitting position.***

When it is poorly compensated, it can lead to renal venous hypertension with impairment of the function, which results in proteinuria and haematuria. It requires treatments that are not yet fully evaluated, such as stenting or transposition of the left renal vein, or renal transposition or gonadal vein-inferior vena cava anastomosis.

The association of left renal vein stenosis and left varicocele does not necessarily mean that the varicocele is compensatory (Deep Open Vicarious Shunt OVS).

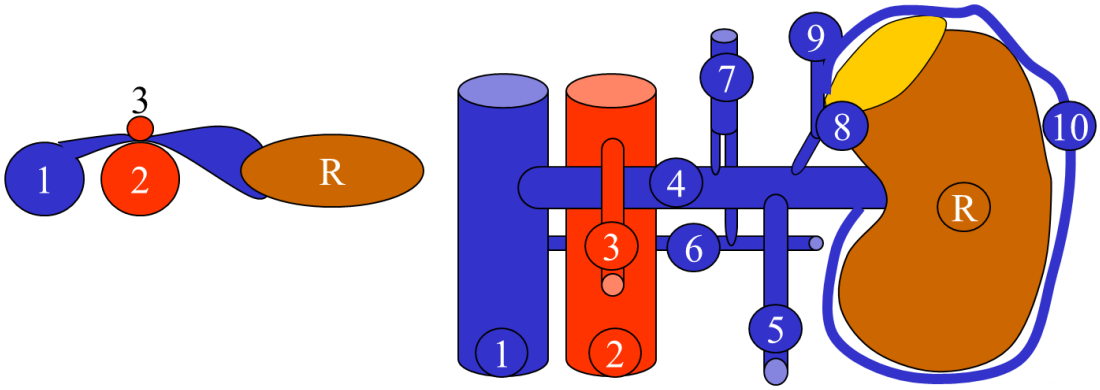
This varicocele is compensatory only if the reflux of the varicocele persists in anti-Trendelenburg decubitus (feet higher than the head) during echodoppler or phlebography.

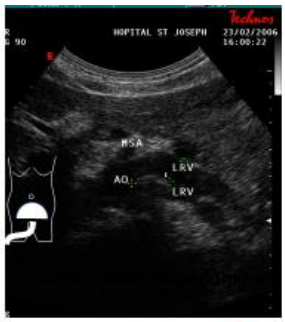
When well compensated by a varicocele, without manifestation of pelvic congestion, the varicocele is considered an open vicarious shunt to be preserved.

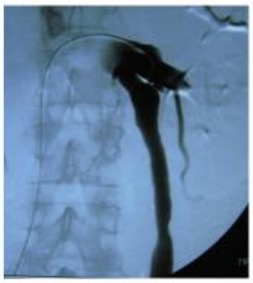
When it is ***well compensated by a varicocele, but is complicated by a pelvic congestion syndrome****,* ***embolization/ligation of the varicocele*** *of this open vicarious OVS shunt can only be considered* ***in association with the treatment of the left renal vein stenosis****.*

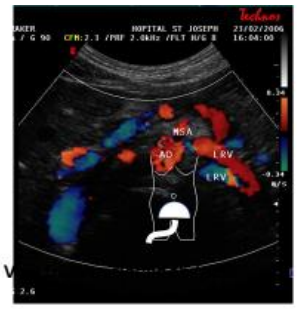
When it is ***well compensated by a varicocele, and is not complicated by a pelvic congestion syndrome****, but is associated with* ***varicose veins of the perineum and/or the lower extremities, treatment should be limited to the treatment of the pelvic escape points, unless the latter reflux not only during dynamic tests, but also at rest, which would indicate a compensation pathway for the renal vein stenosis: Ref.*** *Delfrate, M. Bricchi, C. Franceschi. Minimally invasive procedure for pelvic escape points in women Veins and Lymphatics 2019; volume 8:7789*

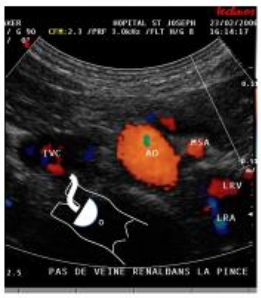
Compression of the left renal vein (4) against the Aorta (2) by the superior mesenteric artery (3) can generate a dangerous hemodynamic stenosis for the left kidney (R) due to a drainage deficit and a vicarious overload of the hemi-azygos (7), lumbar (6) adrenal (8) and phrenic (9) veins, the perirenal venous circle (10) (hemorrhagic risk) and the genital vein (5) whose reflux and pressure generate a left varicocele (spermatic in men and ovarian in women) sometimes responsible for a pelvic congestion syndrome.







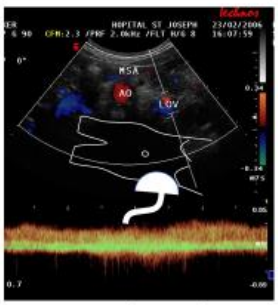




Trans-abdominal scan in Trendelenburg position.

Aorto-Mesenteric clip.
Venous flow = 0

No reno-azygo-lombar compensation
PERMANENT left ovaric vein reflux in Trendelenburg position (head lower than the feet).

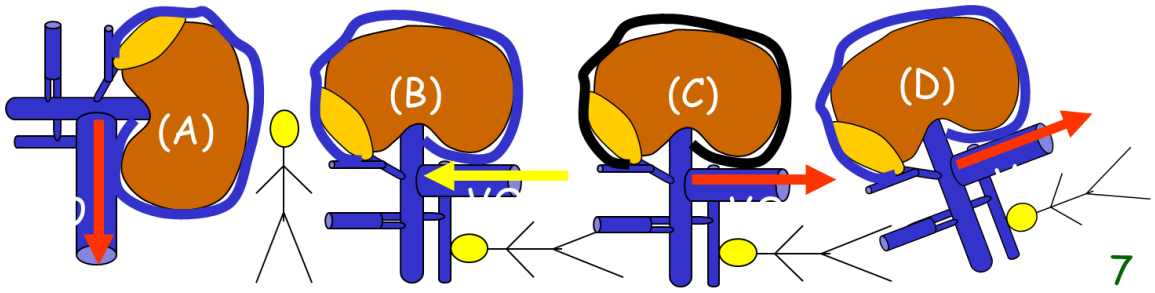


Trans-abdominal scan in Trendelenburg position.

How to assess the risk? Measure the pressure in the renal vein, spontaneously and during the occlusion of the refluxing ovarian vein. By echodoppler: the reflux in the ovarian vein (VO)

-perceived in standing, in the sitting and half-sitting position and modulated by respiration, it can be due to the force of gravity alone without needing residual renal venous pressure (A). It is normalized in decubitus (B)

-If it is perceived as permanent and in decubitus, it can no longer be related to the force of gravity but to a saving vicarious effect (C). This test is made more sensitive by tilting the patient into Trendelenburg where the reflux is seen to persist (D). This pressure can be approximated by measuring the inclination necessary to stop this reflux.



4312-*The varicocele is a dilatation of the left gonadal vein*

Depending on its cause, it may be a refluxing or orthograde OVS open vicarious shunt or an open deviated shunt.

43121- Varicocele refluxing by open deviated shunt ODS

Gonadal (ovarian) veins are much more frequently dilated and refluxed (varicocele) in single and especially multiparous women.

When they do not compensate for renal vein stenosis, they create an open deviated shunt ODS that ***requires treatment only when it is responsible for the clinical syndrome of pelvic congestion.***

W*hen it is related to perineal or lower limb varicose veins, without a clinical syndrome of pelvic congestion,* ***treatment of the pelvic escape points alone is sufficient.*** **Ref:** R. Delfrate, M. Bricchi,C. Franceschi. Minimally invasive procedure for pelvic escape points in women Veins and Lymphatics 2019; volume 8:7789

The knowledge of the precise location of these escape points and their specific treatment avoids unnecessary associated treatments of embolization of varicoceles. ***Ref:*** 1. Franceschi C,

Bahnini A. Points de fuite pelviens viscéraux et varices des membres inférieurs. Phlébologie 2004;57:37-42.2. Franceschi C, Bahnini A. Treatment of lower extremity venous insufficiency due to pelvic escape points in women. Ann Vasc Surg 2005;19:284-8. 3. Franceschi C. Anatomie fonctionnelle et diagnostic des points de fuite bulboclitoridiens chez la femme (point C). J Mal Vasc 2008;33:42.3-

In male, varicocele is benign, requiring treatment only if it is responsible for fertility or pain.

43122- N*on-refluxing compensator varicocele (non-refluxing OVS).*

Unlike varicocele, which performs a refluent open vicarious sshunt of left renal vein stenosis, the open vicarious shunt of ilio-caval occlusion is anterograde (non-refluent).

43123-*Left common Iliac Vein and May Thurner Syndrome MTS named also Cockett Syndrome*

The left common Iliac Vein passes through a clamp formed by the spine and the right common Iliac Artery to reach the Inferior Vena Cava. May Thurner syndrome (also called Cockett syndrome) is a stenosis due to this clamp associated with endoluminal synechiae.

The pinch rate, which varies according to posture, is traumatic for the vein and may explain the clear predominance of left iliac thrombophlebitis, particularly in women during pregnancy. It requires treatment when it causes phlebitis or when it is both hemodynamically and clinically significant in the left lower limb. <u>However, this clamp can be misleading when it is stenosing only in horizontal supine position.</u> A study showed that May Thurner Syndromes diagnosed by phlebography (thus in this position) <u>were mostly asymptomatic</u>

Ref: van Vuuren TM, Kurstjens RLM,Wittens CHA, et al. lllusory angiographic signs of significant lliac vein compression in healthy volunteers. Eur.J Vasc Endovasc Surg 2018;56:874-9.

<u>I have shown by echodoppler that these asymptomatic cases are probably those that we have called Pseudo MTS, which is to say, a complete obstruction in horizontal supine position which disappears in half sitting.</u> That explains the assessment of "illusory" MTS in horizontal supine phlebography in the young asymptomatic subjects of the study.

Therefore, the signs and symptoms falsely reported to these illusory pseudo-MTS (postures not very frequent in everyday life) must make us look for other causes.

Ref: Paolo Zamboni, Claude Franceschi, Roberto Delfrate. The overtreatment of illusory May Thurner syndrome Veins and Lymphatics 2019; volume 8:8020. Vdeo: Pseudo MTS : https://www.youtube.com/watch?v=h931XXo2hdk&t=23s

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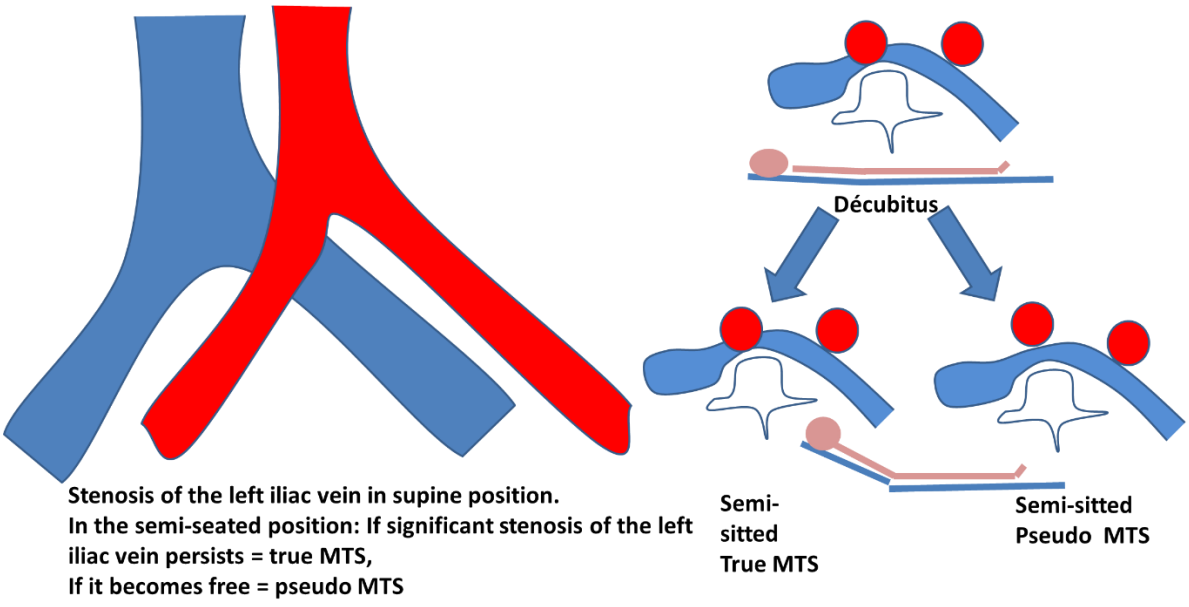
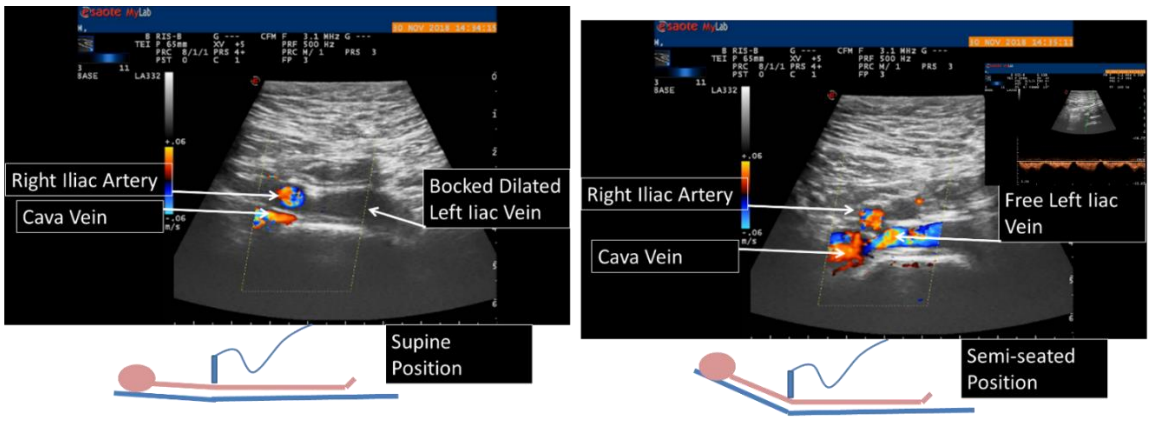




May Thurner Syndrome at phlebography but
Pseudo May Thurner Syndrome on Echodoppler: only postural

43124-*Pelvic Veins and Pelvic escape points*

The trunk of the hypogastric vein is constitutionally incompetent. It receives competent parietal and visceral tributary.

Incompetence and varicose dilatation of these veins are like varicocele very frequent and asymptomatic in mono or multiparous women. *The placenta functioned as a physiological arteriovenous fistula that dilated the visceral pelvic veins, some of which remained dilated and incompetent after pregnancy. Most of these varicose veins are asymptomatic.* ***However, some women suffer from them in the form of "pelvic congestion syndrome" which can be very debilitating and should be recognized and treated. It should not be confused with other causes of pelvic pain. Yet too much overlooked in the past, it tends to be overdiagnosed since some years, maybe due to the" trendy" embolization.***

During pregnancy, the combination of the effects of hormones on venous tone and hypertension of the pelvic veins (placental hyper flow and compression by the gravid uterus)

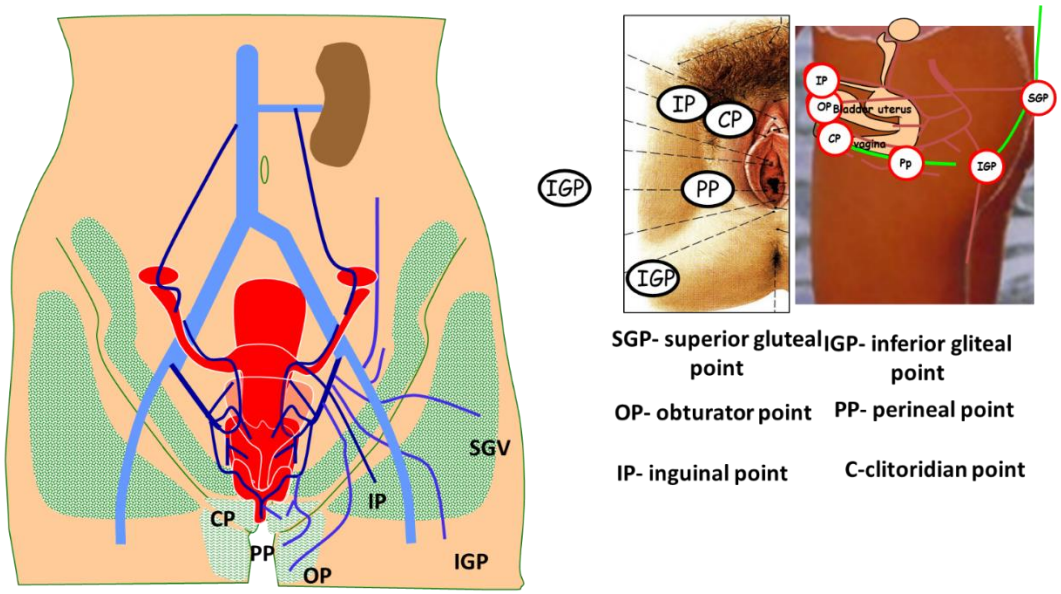
*forces the superficial pelvic tributaries to escape, resulting in varicose veins of the perineum, labia majora, and lower extremities, homo and/or contralateral**. Some escape points close spontaneously in the months following pregnancy, but others may persist.***

Phlebography showed the leaks but could not specify their topography. <u>The echodoppler allowed me to describe and precisely locate these escape points (in particular points I, O, P) and to treat them electively without having to resort to embolization of the pelvic veins.</u> .

Ref: 1. Franceschi C, Bahnini A. Points de fuite pelviens viscéraux et varices des membres inférieurs. Phlébologie 2004;57:37-42.2. Franceschi C, Bahnini A. Treatment of lower extremity venous insufficiency due to pelvic escape points in women. Ann Vasc Surg 2005;19:284-8. 3. Franceschi C. Anatomie fonctionnelle et diagnostic des points de fuite bulboclitoridiens chez la femme (point C). J Mal Vasc 2008;33:42.3. 4: R. Delfrate, M. Bricchi,C. Franceschi. Minimally invasive procedure for pelvic escape points in women Veins and Lymphatics 2019; volume 8:7789

The latter was only proposed in case of associated clinical pelvic congestion syndrome.

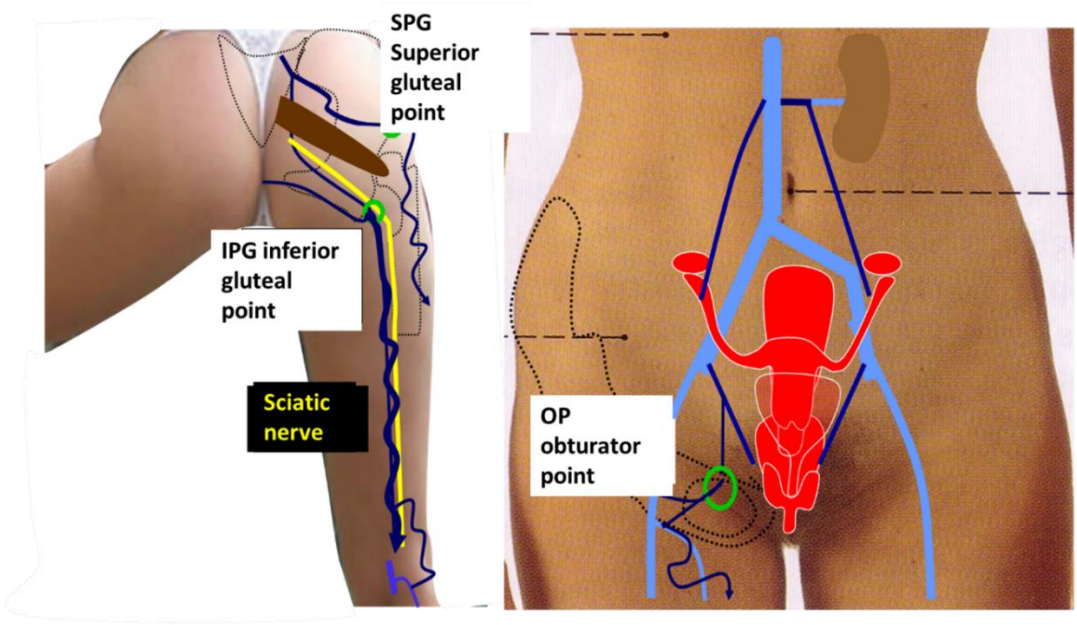
Note that the pelvic escape points assessment by Echodoppler requires a specific technique (see chapter 7).



Schematic venous anatomy of the pelvis of women, the pelvic and parietal veins receive their extra-pelvic afferents through connection points of the pelvic and abdominal walls

Leak points = penetration of superficial veins to the pelvis

431241- *The parietal pelvic veins*



Pelvic leak points of the hypogastric tributaries. Gluteal and obturaror veins.

43124111- Gluteal Veins.

The parietal pelvic veins connect to hypogastric vein. The superior and inferior gluteal veins are incompetent most often in venous malformations and less often in post-thrombotic syndromes. Their Valsalva reflux feeds closed shunts via Superior Gluteal and Inferior Gluteal escape points (GS points and GI point).

4312412- Obturator Vein

The Obturator Vein can flow back into the Femoral Vein and/or the arch of the great saphenous Vein via the Obturator Escape point (O point).

431242 - *Parietal Pelvic escape points*

3 on the right and 3 on the left, the parietal Pelvic Escape points fed by the reflux of 3 parietal tributaries of the hypogastric vein. ***Unlike the visceral escape points, they do not communicate with each other.***

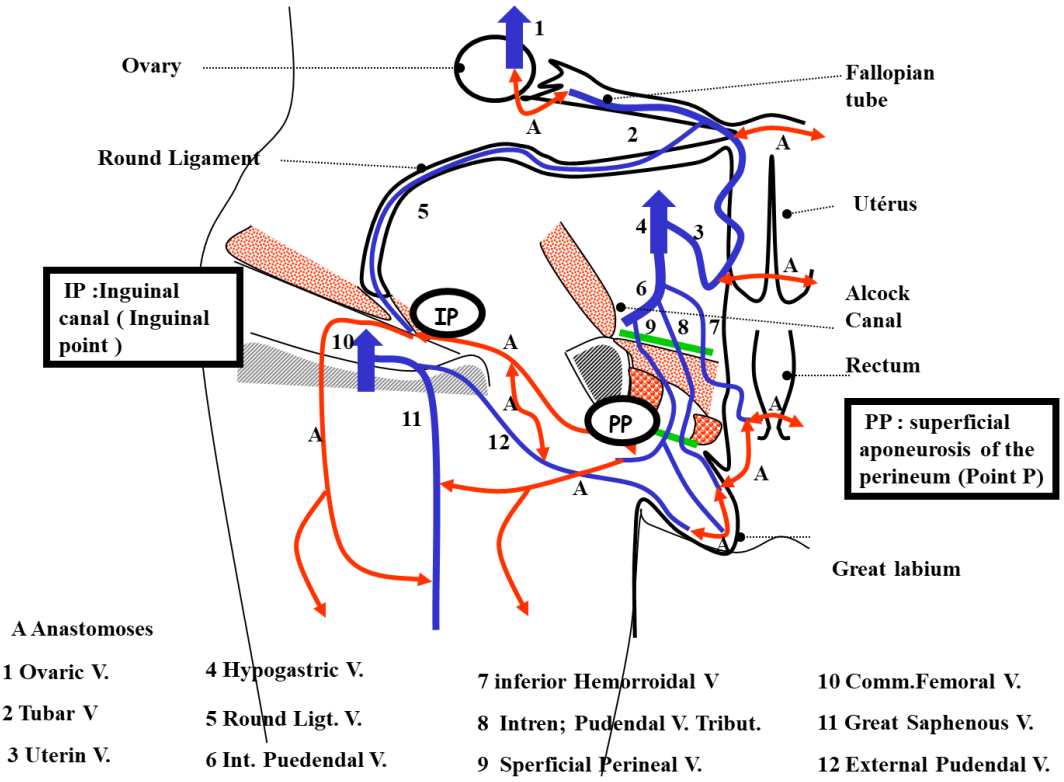
4312421-*The Obturator point (point O) is located t the Obturator Hole from which the Obturator Vein anastomoses with the femoral and/or great saphenous vein.*

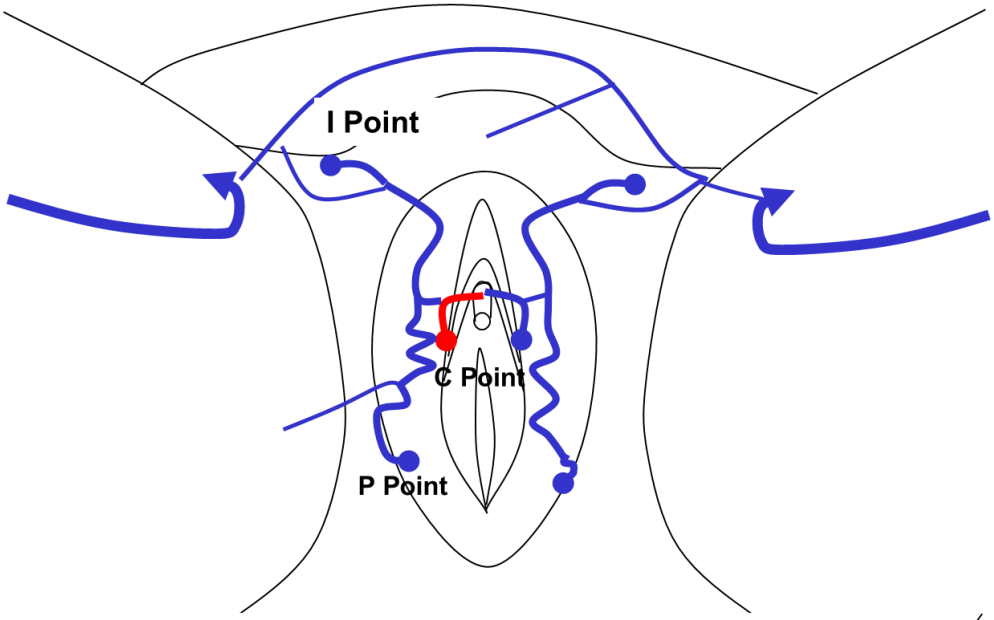
4312422-The superior gluteal point (GS point) ***receives tributaries of the superior gluteal vein, which passes at the superior border of the Pyramidal muscle***

4312423-*The inferior gluteal point (GI point)* ***receives tributaries of the inferior gluteal vein (also called Ischiatic vein) which passes under the Pyramidal muscle along with the sciatic nerve.***

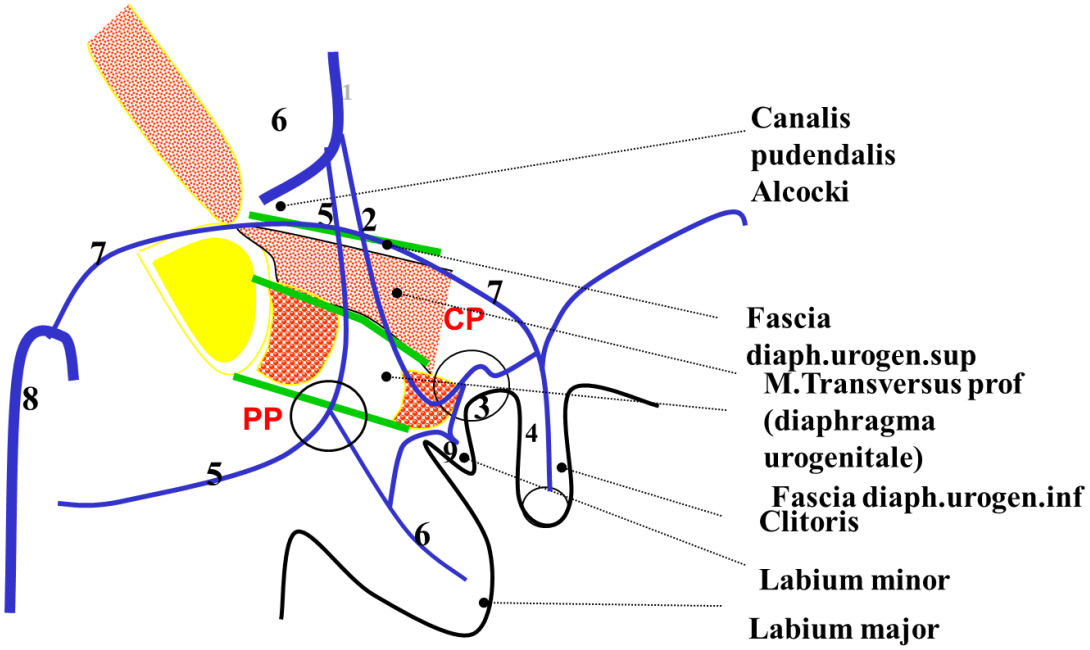
431243-*Visceral pelvis veins*

Ref: 1. Franceschi C, Bahnini A. Points de fuite pelviens viscéraux et varices des membres inférieurs. Phlébologie 2004;57:37-42.2. Franceschi C, Bahnini A. Treatment of lower extremity venous insufficiency due to pelvic escape points in women. Ann Vasc Surg 2005;19:284-8. 3. Franceschi C. Anatomie fonctionnelle et diagnostic des points de fuite bulboclitoridiens chez la femme (point C). J Mal Vasc 2008;33:42.





Plexus ven. Commnicans (Clit. Et Bulbus vest.): During Valsalva maneuvre, reflux from Internal Pudendal vein into Anterior Saphena varicose vein through retrograde flow in bulbar vein, Plexus Ven. Communicans (Clit. Et Bulbus vest.), Dorsal Clitoris Vein and External Pudendal



1 Vena pudenda interna
2 Vena Bulbi Vestibuli
3 Plexus ven. Commnicans (Clit. Et Bulbus vest.) Clitoris point cP
4 Vena clit. subcutanea
5 Vena perinei
6 Vena labialis ant.
7 Vena pudenda externa
8 Safena magna
9 Plexus labialis

CP: Clitoral point

PP:Perineal Point

431244 - *Visceral Pelvic escape points*

The Visceral Veins are valvulated but communicate with each other transversely and longitudinally by non-valvulated plexuses.

In women, there are, from top to bottom, the hemorrhoidal, pudendal, round ligament, uterine, vaginal, bladder and periurethral plexuses. ***This explains why a right visceral reflux sometimes communicates with left visceral escape points and vice versa.***

They also communicate with the ovarian veins from which they may receive reflux via the plexuses.

4312441-Internal Pudendal Vein

The Internal Pudendal Vein refluxes into the Perineal and Labial veins via the Perineal point (point P) and into the dorsal vein of the clitoris via the Bulbar Vein (Clitoral point C).

4312442-The Vein of the Round Ligament of the Uterus communicates with the ovarian veins via the tubar vein.

4312433. *External hemorrhoidal vein and hemorrhoidal disease ("haemorrhoids")*

The superior rectal veins drain into the inferior mesenteric vein. The middle and lower rectal veins drain into the hypogastric vein via the internal pudendal vein. The inferior rectal veins drain the rectum via their internal hemorrhoidal tributaries and the anal canal via the external hemorrhoidal veins.

These veins communicate with each other via ***the submucosal hemorrhoidal plexus which constitutes a porto-caval anastomosis. The internal plexus is in the upper part of the anal canal and the external plexus is located at the anus.***

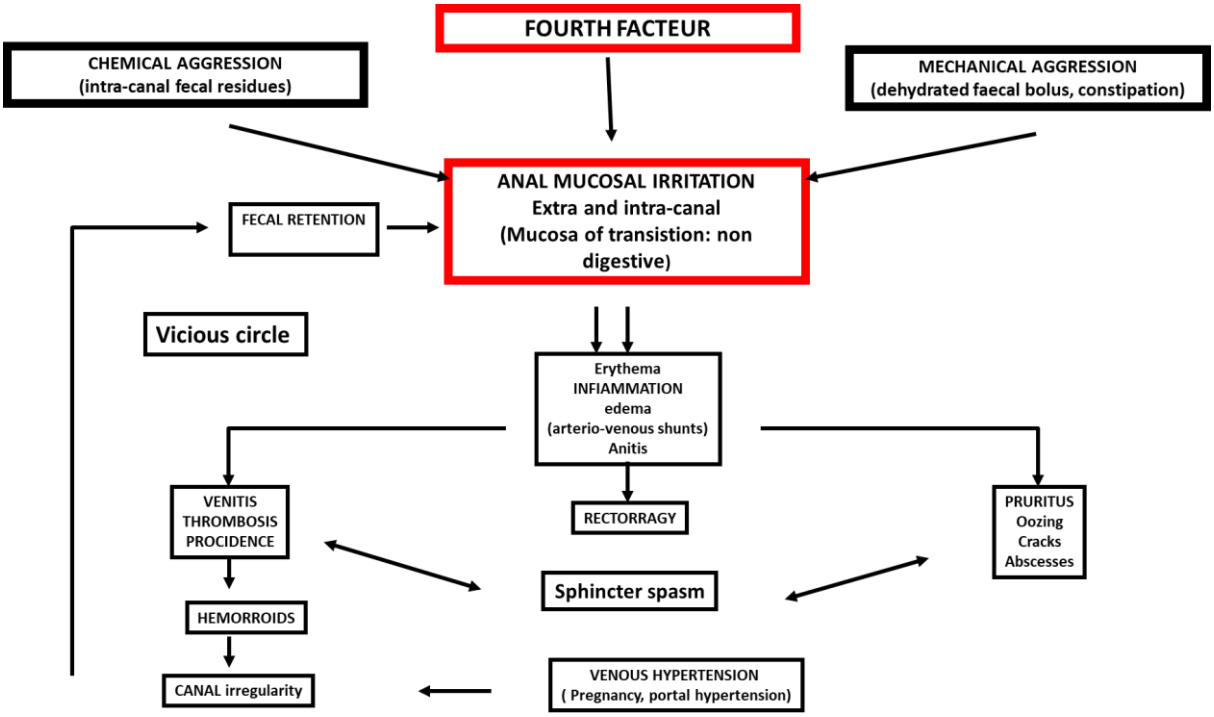
Dilated sometimes by portal hypertension, but most often independently of this pathology, they produce the disease called "haemorrhoids", internal in the anal canal and external in the anus.

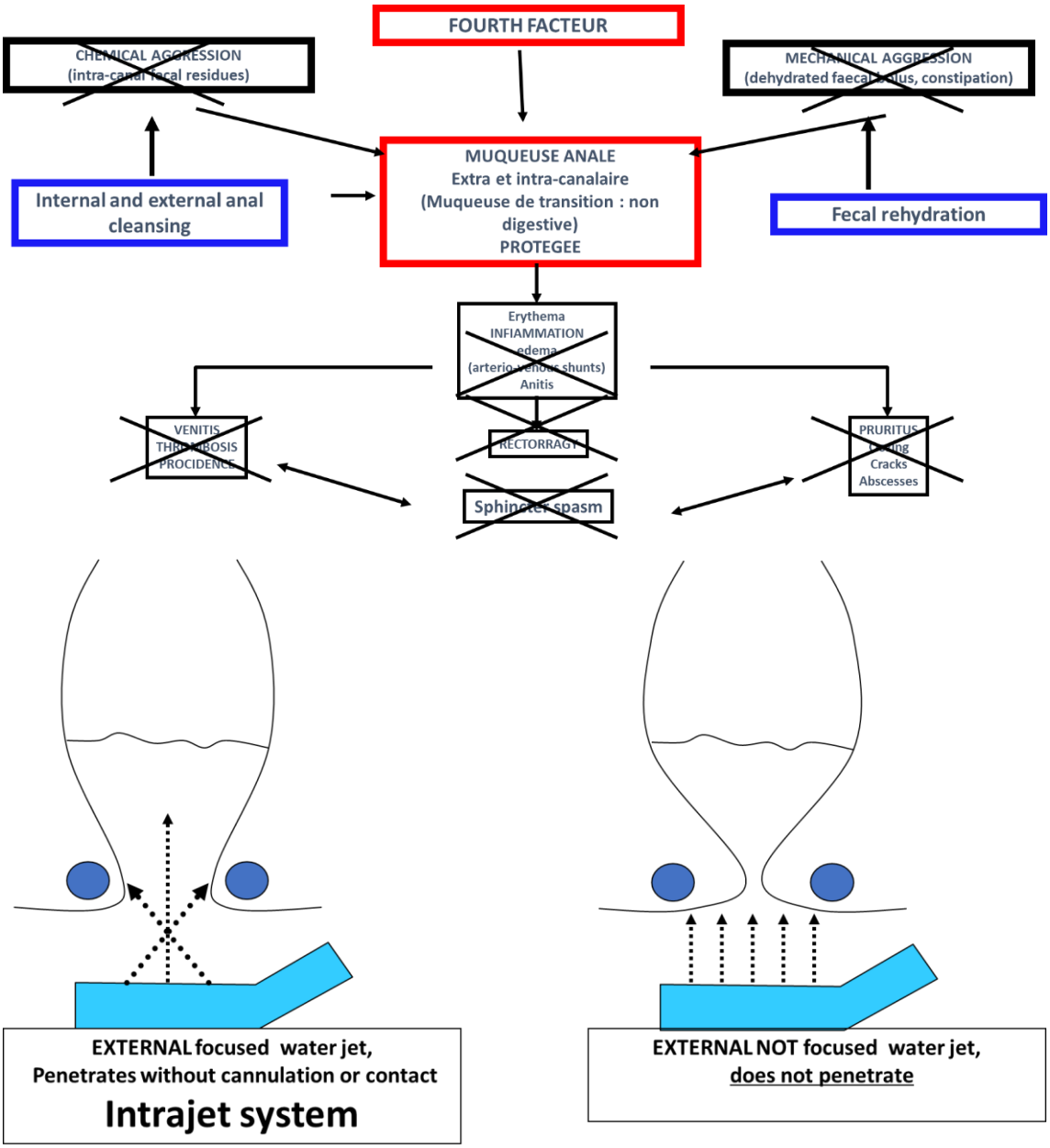
Several physiopathological theories *have been proposed, of which 3 factors are most often cited. Vascular: decrease in venous return due to abdominal thrust during defecation and modification of pelvic and digestive vasomotricity: erythema, bleeding. Mechanical: submucosal laxity and supporting tissues + sphincter constipation: sphincter hypertonicity: Prolapse, rectal bleeding, strangulation, venous dilatation*

The fourth factor that I have proposed

Ref: **C.**Franceschi. Hémorroïdes : maladie des veines ou d'un quatrième facteur. Essai d'analyse physiopathologique. Conséquences thérapeutiques. Actualités Médicales Internationales. Angiologie (8), n° 145, décembre 1991 VIDEO https://youtu.be/1FoYynLlb98**.**

is consistent with the fact that it combines the first three as complications of the fourth. It is the intolerance of the mucosa of the anal canal to stool and to trauma during defecation because it is a transitional mucosa in the same way as the mucosa of the oropharynx. The complications are local irritation, inflammation, transmitted to the hemorrhoidal veins which thrombose and dilate, bleeding by mucosal inflammation etc. The ***treatment consists of facilitating non-traumatic defecation and cleaning the residues after defecation****. A special water jet allows water to penetrate the lower rectum, without contact with the body because the device is 5 to 7 cm away. Before defecation, it allows without cannulation a micro-lavage which facilitates a non-traumatic evacuation and without excessive push. After defecation, it allows a rinsing of the macro and micro residues of fecal matter. An independent RCT study has demonstrated its effectiveness. Eliminates pain, pruritus, bleeding and stops the evolution of the disease.* **Ref**: B.Vergeau,R.Clément,M.Massoneau,C.Franceschi. Evaluation de l'efficacité et de la tolérance d'un nouveau procédé de traitement des hémorroïdes symptomatiques : Intrajet. Med.Chir.Dig. 1995 -24- 109-111 Video: Intrajet 4th factor https://www.youtube.com/watch?v=1FoYynLlb98&t=16s





431244- *Pelvic Visceral escape points*

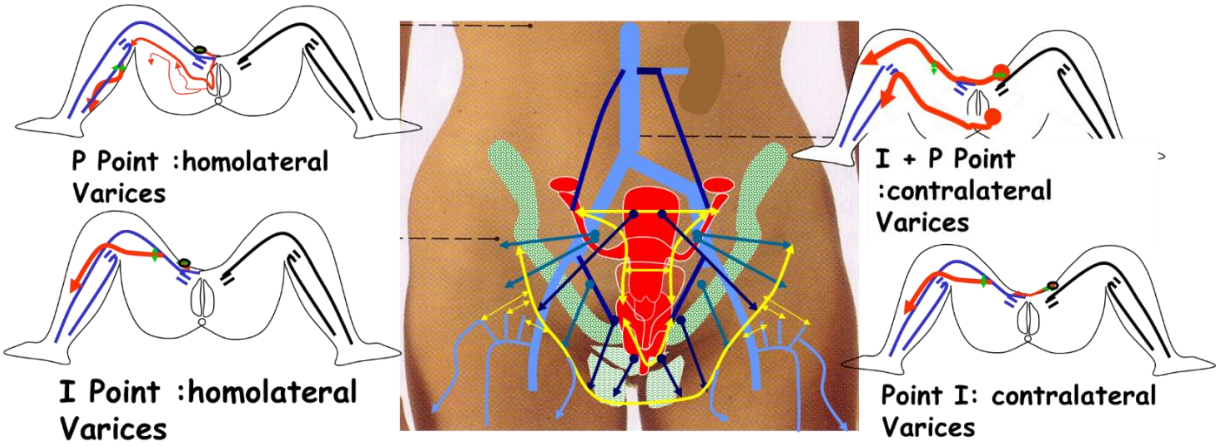
I have located and described with the echodoppler 6 escape points responsible for varicose veins of the perineum and the lower extremities, fed by the visceral tributaries of the Hypogastric Vein. 3 on the right and 3 on the left, they communicate by homolateral but contralateral anastomoses, so that a right escape point can feed varicose veins of the left lower limb and vice versa. They are almost always found in mono or multiparous women.

Ref: 1. Franceschi C, Bahnini A. Points de fuite pelviens viscéraux et varices des membres inférieurs. Phlébologie 2004;57:37-42.2. Franceschi C, Bahnini A. Treatment of lower extremity venous insufficiency due to pelvic escape points in women. Ann Vasc Surg 2005;19:284-8. 3. Franceschi C. Anatomie fonctionnelle et diagnostic des points de fuite bulboclitoridiens chez la femme (point C). J Mal Vasc 2008;33:42.

4312441-The perineal point (P point) ***is located at the union of the ¾ anterior and ¼ posterior of the vulva-perineal fold, at the orifice of the perineal fascia crossed by the perineal vein, which ascends between the deep and superficial muscles of the perineum and then joins the internal pudendal vein in Alcock's canal.***

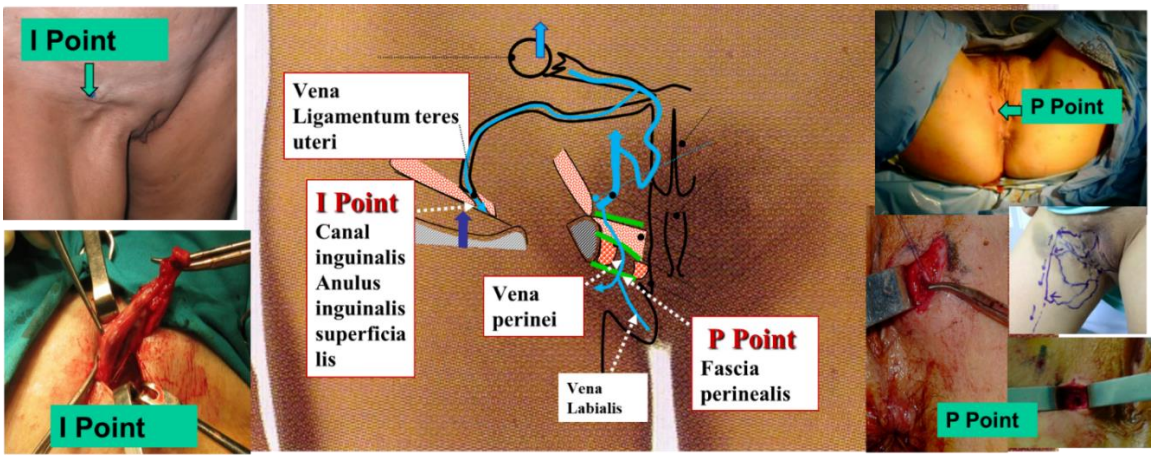
4312442-The Clitoral point (C point) ***is located at the base the clitoris at the plexus which joins the superficial dorsal vein of the clitoris to the bulbar vein which drains into the internal pudendal. This point can exist in men, but much more rarely.***

4312443-The Inguinal point (I point) ***is located at the superficial orifice of the inguinal canal, close to the pubis spine. It is crossed by the Vein of the Round Ligament of the Uterus, which is anastomosed to the tubal and ovarian veins, from which it can transmit reflux. The equivalent of this point may also exist in men with a varicocele.***



These 12 tributary branches (6 on each side) can flow back into each other on the same side or on the other side via the plexus and/ or open the lower limbs and the perineum via the pelvic leakage points which can also communicate with each other on the same side and on the other side via the superficial anastomoses

Creating as many leakage points towards the perineum and the lower limbs and can communicate between them (rich and anarchic anastomoses) which can in their turn reflux into: The saphenous or extra-saphenous networks

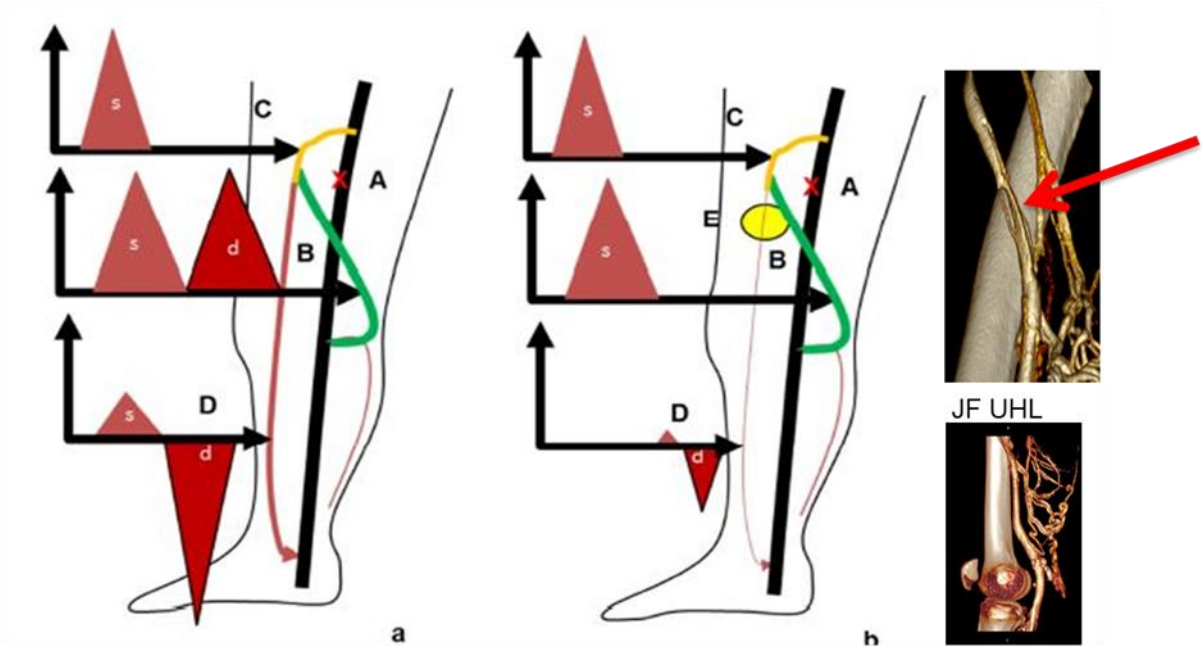


432- *Veins of the lower extremities*

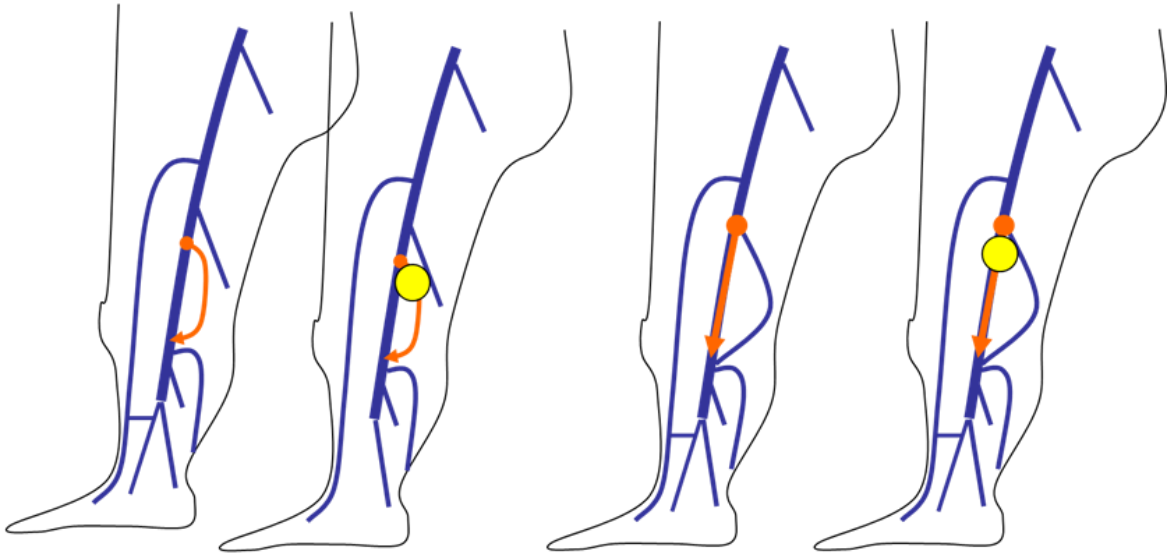
4321- Femoral veins

43211- The single or double superficial femoral vein is necessary to recognize because it is decisive for the hemodynamic treatment of deep venous insufficiency by deep CHIVA (CHIVP) when only one of the two is incompetent.

43212- Congenital superficial femoral vein stenosis is quite common in the Hunter ring (Uhl). A few years before, I had described this obstacle hemodynamically because of the systolic reflux of the saphenopopliteal junction, witnessing an open shunt vicariant of a superficial femoral obstacle. It explains the spontaneous and post-crossectomy cavernomas of the small saphenous vein junction as well as the mixed shunts its level.



Obstacle of the Superficial Femoral Vein diagnosed hemodynamically by echodoppler that I called vicarious open shunts and mixed shunts and treated by CHIVA before anatomical confirmation by Dr JF UHL



Double superficial femoral vein with an incontinent collateral is a closed shunt corrected by CHIVA

Incontinent superficial femoral vein and continuous deep femoral vein connected to the popliteal vein is a closed shunt corrected by CHIVA

The variability of the anatomy of the femoral veins is crucial for the treatment of venous insufficiency

43213- The deep femoral vein communicates sometimes with the popliteal vein. This communication is particularly important in cases of occlusion of the superficial femoral vein. It allows CHIVP treatment by ligation of an incompetent superficial femoral vein in the event of a deep closed shunt.

4322- Superficial veins of the lower extremities

43221-Saphenous veins and Giacomini veins

The trunks of the Great and Small saphenous veins are N2 and their tributaries N3. Giacomini's vein is N2, but its course and location in relation to the fascia are not constant.

432211- The great saphenous vein Is located along the medial aspect of the thigh and leg. Its calibre, very variable in healthy subjects as well as in varicose subjects, does not prejudge its pathology.

It is especially interesting to know this in case of need for arterial by-pass surgery. It usually varies between 4 and 5 mm. *It sometimes presents segmental hypoplasia or aplasia which are to be considered for the CHIVA strategy.* ***Its arch may be double or may by-pass the femoral artery from behind.***

The descending tributaries are the external pudendal veins, the superficial epigastric vein, and the superficial iliac circumflex. The Doppler normal "orthograde descending"

direction of their flows cannot be distinguished from a reflux fed by a pelvic escape point by calf compression or Paranà manoeuvres, but only by the Valsalva test. They often communicate their reflux to contralateral descending tributaries through an OVS in case of iliac and or ilio-caval occlusion, or CS by pelvic escape.

The saphenofemoral junction SFJ is often the escape point EP of closed shunts but also sometimes of open vicarious shunt OVS in case of iliac and/or ilio-caval obstruction. It represents the so called "spontaneous Palma" when the OVS connects the right and left crosses by anastomoses between their descending tributaries.

Its calibre is not measured at the level of the arch but 10 cm below the saphenofemoral junction.

Finally, competent, or not, it remains the best arterial by-pass material, especially below the knee, and is equivalent to the internal mammary by-pass when it is harvested according to the no-touch method. **Ref:** 1-No touch technique of saphenous vein harvesting: Is great graft patency rate provided? Papakonstantinou NA J Thorac Cardiovasc Surg. 2015 Oct;150(4):880-8. doi: 10.1016/j.jtcvs.2015.07.027. Epub 2015 Jul 15.

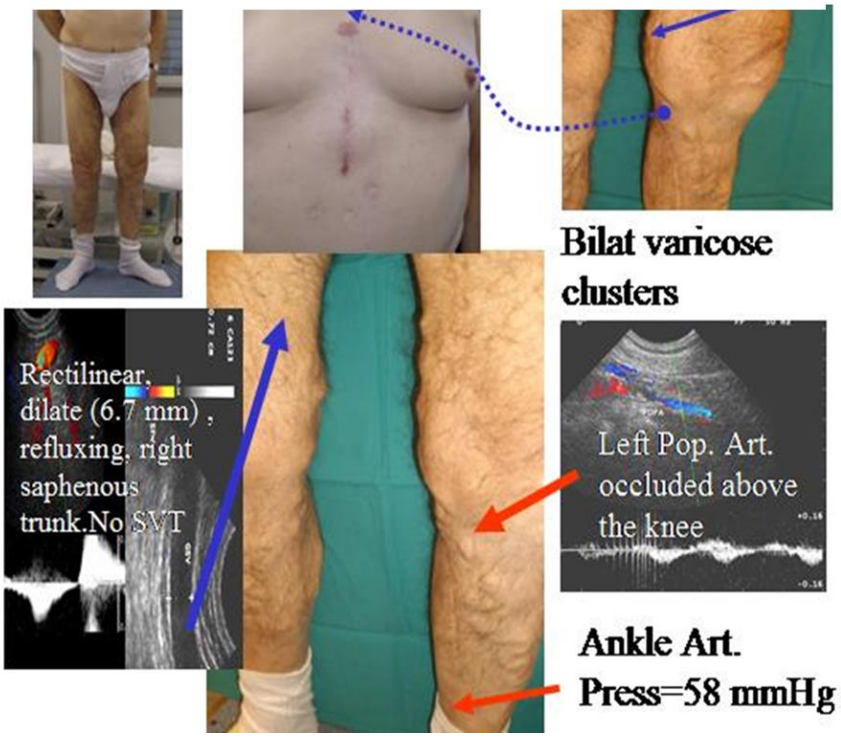
2-The no-touch saphenous vein for coronary artery by-pass grafting maintains a patency, after 16 years, comparable to the left internal thoracic artery: A randomized trial. Samano R1, ClinicalTrials.gov NCT01686100.Copyright © 2015

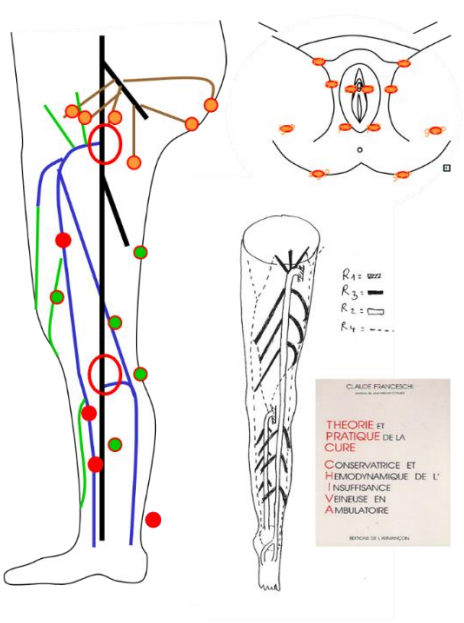
In this regard, Bioprotec Lyon saint Priest (https://bioprotec.fr) recovers stripped saphenous veins from varicose patients and sells them as grafts for arterial bypass surgery.

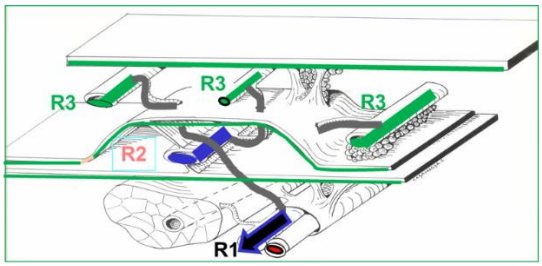
The Saphenous vein can save life, even in varicose people. Why destroy it without warning the patient? Especially since it can be treated effectively without destroying the saphenous vein by hemodynamic methods

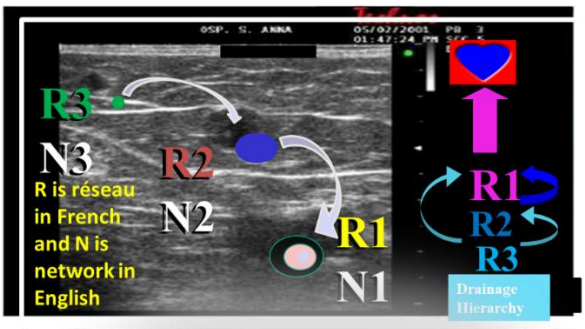
Male 78 y

To-day:
-left leg limp
-Bilat varicose clusters
10 years ago
-5 coronary by-passes (3 left GSV) +
-Right GSV crossectomy for SVT







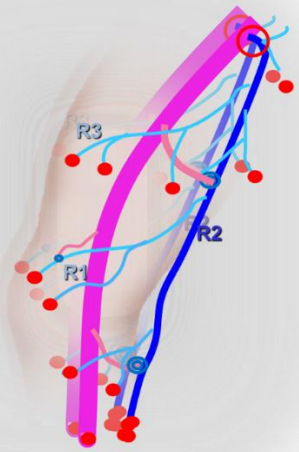


Potential escape points

- Saphenofemoral
- Saphenopopliteal junction
- Pelvic leakage points
-Numerous perforators

Networks in 4 hierarchical levels of drainage

-R1, R2, R3, R4 according to their topography and hemodynamic function



43222-*The small saphenous vein runs along the posterior aspect of the calf inside a fascia separation.*

It is usually of small calibre, but a large calibre is not necessarily pathological.

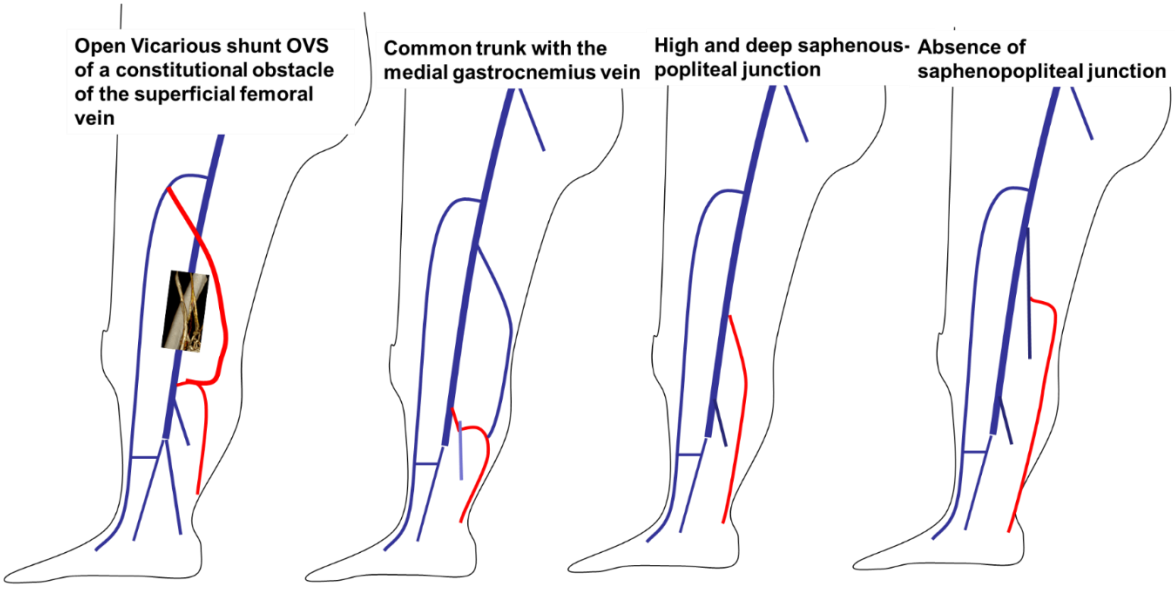
Two Particularities are important to consider for treatment, especially surgery.

-First, it terminates directly via its junction at variable levels in the femoral vein or indirectly via a common trunk with the internal gastrocnemius vein.

-Secondly, its junction is close to the sciatic nerve and its trunk runs along the short saphenous nerve, which is a source of postoperative neurological complications.

The saphenopopliteal junction is often the escape point of a closed shunt CS. It is also, quite often, the escape point of an open vicarious shunt OVS, or even of a mixed shunt MS related to a femoral superficial obstacle. This obstacle, which I

described hemodynamically because of the presence of this OVS, was later described anatomically by Uhl.



Anatomical and hemodynamic features of the Small Saphenous Vein relevant for hemodynamic treatments

43223- *Giacomini's Vein usually connects the junction of the of the small saphenous vein to the trunk of the great saphenous vein.*

Its course and structure often vary, without pathological value.

It presents 4 useful points to recognize.

- ***Its function as Open vicarious shunt,***
- ***First segment of a mixed MS shunt,***
- ***Descending drainage from a perineal EP escape point (P point),***
- ***To disconnect closed CS just below the junction with Giacomini's vein.***

4323-*Valves*

Venous valves are watertight flaps usually in the form of two diametrically opposed flexible swallow's nests, staggered, and arranged in variable numbers according to the individual, inside the deep and superficial veins.

<u>They open and close according to the direction of the force vector of the pressure that directs the flow</u>.

They <u>remain open at rest</u> due to the Residual Flow/pressure of tissue drainage from the microcirculation, <u>regardless of posture</u>.

The <u>inversion of the force vector</u> reverses the pressure gradient and leads to their <u>closure</u>. These inversions are <u>transient</u>, the time of diastole of the valvulo-muscular pumps during walking.

43231-*The complete valve closure occurs after a brief reflux time.*

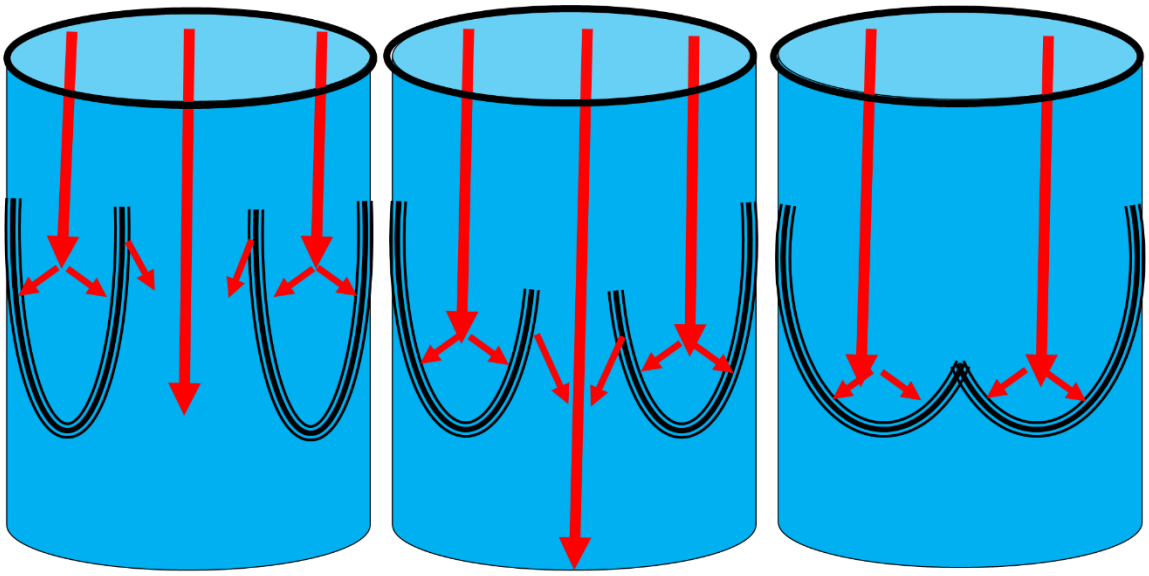
<u>The quicker and powerful the inversion, the shorter the time of reflux</u> *(just as a door closes quicker and stronger according to the draft power that pushes it)**. This is the so-called physiological reflux, usually less than 500ms, but which can vary according to the closing conditions.***

Normal valves can reflux more than 500 ms, in 2 conditions:

A slow inversion of the pressure gradient slows down the closing time.

-In lying position, due to the lack of Gravitational Hydrostatic Pressure GHSP, the veins are partially collapsed so that the time to refill them delays the closure time.

They are said to be competent (watertight) when they can prevent extra physiological reflux.



Valve closure: velocity proportional to the aspirative pressure gradient by positive pressure in the valve nest and Venturi effect in the center.

43232- *Valvular incompetence.*

Valve incompetence can be constitutional/congenital but is more often acquired during life. Valve destruction is sometimes secondary to deep and superficial thrombosis.

But <u>most often, it would be due to inflammation caused by blood stasis in the valve nests of the superficial veins</u>. Thus, it would <u>be favoured by the living conditions</u> prolonged immobile postures sitting and standing.

The variability of the number of valves according to the individuals, could explain for part of varicose veins in the families.

The upward or downward progression of valve incompetence does not appear to be systematic and has no impact on the hemodynamic treatment strategy.

Incompetence of one or more valves is pathogenic only when their topography impairs the dynamic fractionation of the FDPHG hydrostatic pressure DFGHSP.

Valve incompetence is the most frequent physical cause of venous insufficiency. *It creates hemodynamic conditions that, when walking, excessively dilate the veins (varicose veins) and reduce tissue drainage (oedema, trophic disorders, ulcers)* ***because it hampers the dynamic fractionation of the gravitational hydrostatic pressure FDPHG and activates shunts****.*

On the other hand, it is ***not pathogenic at rest****, in a lying or sitting position with the legs raised, because the column of hydrostatic gravitational pressure is short, and the shunts are inactivated****.***

It is neither more nor less pathogenic than prolonged standing or sitting in a healthy subject in whom immobility keeps the valves open, which does not fraction the column. This means that standing still conditions are alike between venous competent and incompetent subjects.

The closures occur successively from top to bottom during the thoraco-abdominal pump systole cough (effort of defecation, Valsalva manoeuvre) and cardiac in case of tricuspid reflux.

On the contrary, they follow one another from bottom to top during diastole of the valvulo-muscular pumps of the calf (walking) and the compression and Paranà manoeuvres. Video; sequential valves closure ***https://www.youtube.com/watch?v=XVt7RwitPkw&t=2s***

It is understood here that the more distal the valves that close, the more the pressure at the ankle is reduced. This explains why treatments that restore continence (CHIVP, valve repair, neo-valve, prosthesis) only at the femoral level are hemodynamically less effective than those that restore it below the knee.

Pseudo-incompetence

Reflux does not always mean incompetence.

This is the case, for example, of reflux from a popliteal vein between 2 competent valves that leaks into an incompetent sapheno-popliteal junction.

This is also the case of segmental positive Paranà and negative Valsalva non-overloaded reflux of the great saphenous vein. It is either a spontaneous shunt 0 or the expected good result after N1>N2 CHIVA disconnection

It can also escape just because of an incompetent N2>N3 escape in an incompetent tributary N2>N3>N1. This is the type II shunt case which requires a simple N2-N3 disconnection.

Degrees of incompetence.

Incompetence is proportional to the size of the valvular gap.

-If it is small, the reflux time is long, but the flow velocity is low: I call it "partial reflux."

-If it is large, the time is shorter, and the overlying column is incompetent over its entire height: I call it "total reflux."

-If the overlying valves are competent: I call it "segmental reflux".

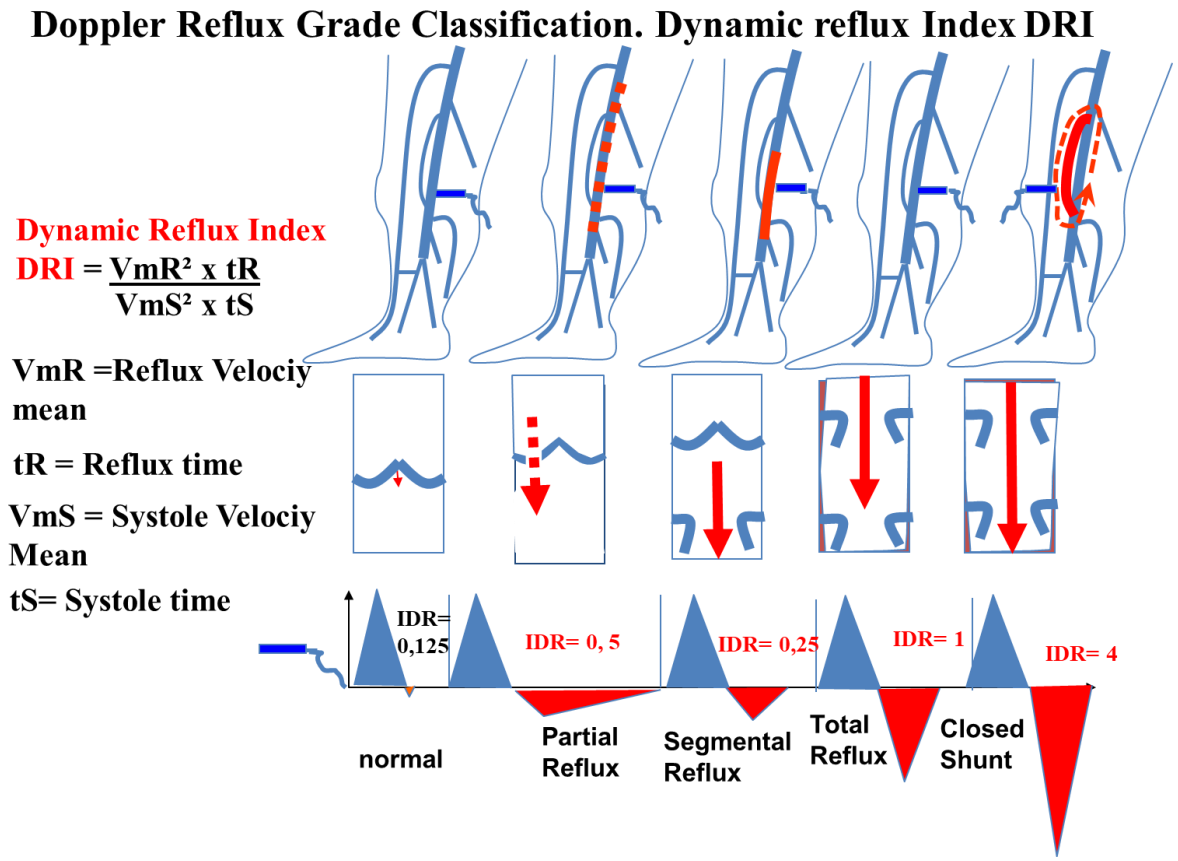
If the amount and time of reflux is greater than the systolic flow, I call it "shunt diastolic reflux".

Taking this in consideration, I have proposed a measure of reflux called the Dynamic Reflux Index (DRI) to evaluate the hemodynamic incidence of reflux using Doppler

Journal des Maladies Vasculaires (Paris)
Masson, 1997, 22, 2, 91-95

MESURES ET INTERPRÉTATION DES FLUX VEINEUX LORS DES MANŒUVRES DE STIMULATION. COMPRESSIONS MANUELLES ET MANŒUVRE DE PARANA. INDICE DYNAMIQUE DE REFLUX (IDR) ET INDICE DE PSATAKIS

C. FRANCESCHI



4324- *Connections between networks*

The connections between the various networks are variable in number, topography, and functionality. They consist of N2>N1 junctions (Saphenofemoral SFJ and Saphenopopliteal SPJ junctions) and N2>N1 or N3>N1 perforators, but also anastomoses.

In accordance with the physiological drainage hierarchy, venules drain into N3 tributaries which empty into the collecting trunks of the Great and Small saphenous veins N2 (N3>N2) which then empty into the popliteal and femoral collecting trunks (N3>N2>N3. Perforators are inconstantly valvulated venous segments of variable length and composition arranged along the course of superficial veins N2 and N3, which they connect to the deep veins by "perforating" the fascia and aponeurosis N2>N1 and N3>N1).

The anastomose is connection between the different superficial territories called phlebosomes.

These connections must be known because they are all potentially the place of inversion and pathological transmission of flows and refluxes between the different phlebosomes and compartments.

43241-Spahenofemoral junction SFJ and Saphenopopliteal junction SPJ

The Saphenofemoral and Saphenopopliteal junctions are the N1>N2 escape points of the most frequent closed CS shunts, but also of open vicarious OVS shunts compensating for deep obstacles, or mixed MS shunts. *Only dynamic tests and manoeuvres can differentiate them to establish the best hemodynamic strategy.*

432411-SFJ and SPJ incompetence as escape points of closed shunts when they show reflux activated by thoracoabdominal pump systole (positive Valsalva manoeuvre) and valvulomuscular pump diastole (compression or Paranà manoeuvre).

Systolic reflux followed by diastolic reflux reflects a mixed shunt MS escape point associating an open vicarious shunt OVS with a closed shunt CS that will be described later.

The topography of the SFJ is almost constant, in the groin. It can rarely be double and exceptionally absent. It most often comprises a valve called the ***terminal valve followed downstream a pre-terminal valve.*** *Their respective degree of continence is* ***of decisive*** *physiopathological and* ***therapeutic value***

When the terminal valve is competent, the pre-terminal incompetence shows reflux during the compression release and the Paranà manoeuvre, fed by the descending tributaries of the arch.

These manoeuvres do not allow us to say whether these flows are normal or overloaded by pelvic escape points. Only the Valsalva Manoeuvre can provide the differential diagnosis. It causes systolic Valsalva reflux (during thrust) only if there is a pelvic escape point. Then, the latter is localized by ascending the echodoppler probe along the positive Valsalva tributary vein.

432412--The SPJ is usually found at the popliteal fossa between the gastrocnemius muscles, under the popliteal fold.

It can also be found higher and deeper, under the thigh muscles when it connects to the Popliteal Vein, and below when the small saphenous vein connects to the internal gastrocnemius vein with that it form a common trunk that connects to the Popliteal vein..

SPJ can also be absent; the Small saphenous Vein being then prolonged directly by the Vein of Giacomini.

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4324133- The networks exchange flows via their anastomoses according to their pressure gradients.

43242- *Perforators.*

Perforators are irregularly valvulated venous segments of variable length that connect the superficial N2 and N3 veins to the deep N1 veins by "perforating" the fascia and

aponeurosis. *Their number is very high according to anatomists, but it is* ***much lower*** *if only those which are* ***visible and active on echodoppler*** *are retained.*

Therefore, ***they must be sought everywhere, including where they are not usual****, as long as the escape or re-entry point of a reflux has not been identified.*

They are said to be

- direct when they are connected to deep intermuscular veins and

-indirect when they connect to intramuscular veins.

They may also ***be connected to the bone marrow via the periosteum****, particularly those of the Tibia****.***

Thanks to echodoppler, they can be located at all levels of all superficial veins N2 and N3.

The most frequently encountered are located from top to bottom at the level of the perineum, the buttock, the posterior and medial aspect of the thigh, the popliteal fossa, the medial aspect of the leg and the medial gastrocnemius muscle.

Some are named after the anatomist who described them.

Dodd's perforator *at the union of the middle third/lower third of the long saphenous vein of the thigh.*

Boyd's perforator *at the subcondylar great saphenous vein*

Cockett's perforators *connect the great saphenous vein to the posterior tibial vein.*

In practice, it is the echodoppler that finds the perforators of the EP escape and RP re-entry points by systematically following the dilated and/or refluxing superficial veins during tests and dynamic manoeuvres.

Competent valves prevent reflux when the pressure at N1 is greater than N2 or N3 (reversed pressure gradient).

As we will see later, ***the absence of a valve in the perforators does not cause reflux when the pressure gradient is oriented inwards R>N1 or N3>N1 by the dynamic manoeuvres***

On the other hand, they allow reflux when the gradient is oriented towards N2 or N3, N1>N3, N1>N2. Thus, ***the valves are passive. They do not create the flows, but when competent, they oppose them when the pressure gradient direction is outwards.***

The pathological or pathogenic significance of reflux depends on the conditions of its occurrence, *at rest and according to the systolic or diastolic phases of the pumps, particularly the thoracoabdominal and valvular pumps. Thus,* ***a systolic reflux is not always pathogenic, whereas a diastolic reflux between 2 compartments is always a pathological escape point.***

Indeed, systolic reflux is not always due to the escape point of a vicarious shunt compensating for a deep obstacle.

It is most often due to an incompetent perforator that allows a small part of the systolic ejection flow of the valvular pump to pass through.

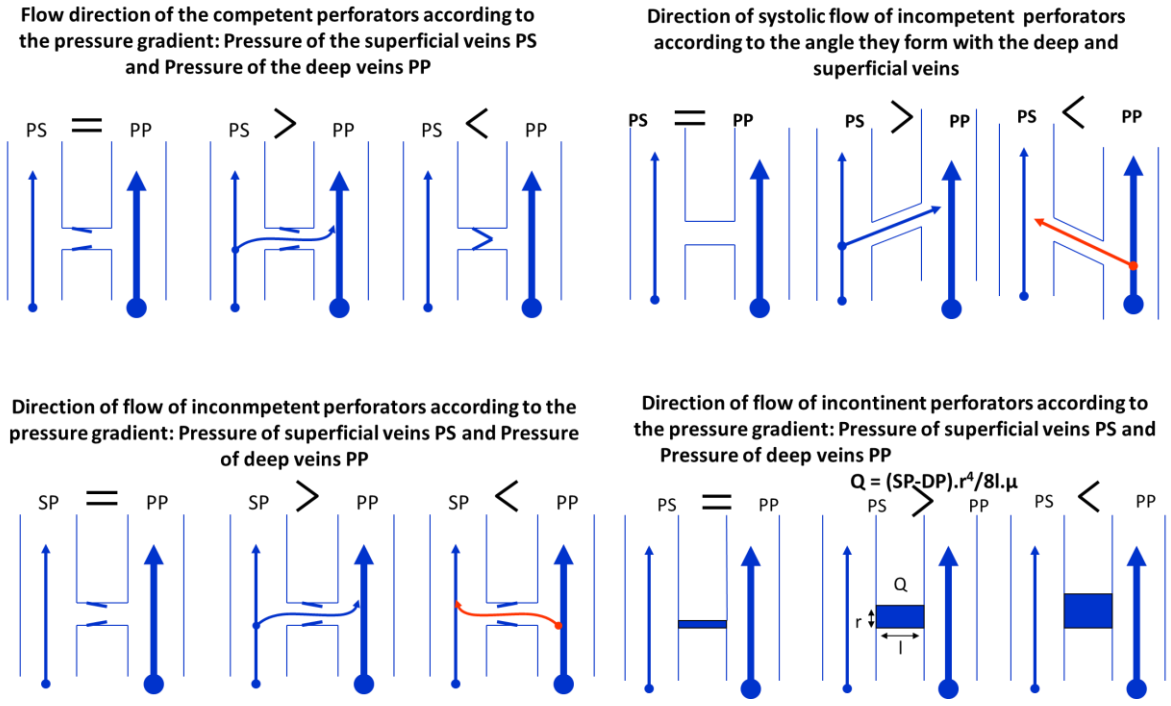
These <u>systolic refluxes of negligible pathological incidence</u> are favoured by the geometry of the incompetent perforators, which form an upward angle with the deep vein (see figure below).

These benign systolic refluxes <u>can be neglected</u>, *especially when they are associated with a diastolic re-entry flow that must be preserved.*

Nevertheless, it can be <u>associated with diastolic reflux when it is the escape point of a mixed shunt MS.</u>

<u>The calibre of the perforators, small or large, does not prejudge their pathological function. Thus, an escape point may be small and must be eliminated and a re-entry point of large calibre must be respected</u>.

The plantar perforators are constitutionally incompetent, which explains the saphenous flow supplied by the Léjars pump via the N1>N2 reflux of the perforators when the foot presses the ground.



43243- *Anastomoses.*

Anastomoses between the veins of the different phlebosomes are extremely numerous, so that they allow compensatory flow exchanges when one is absent or occluded or destroyed by a destructive treatment (varicose recurrence).

They can also transmit to a network a reflux which they have received from another network, as when they transmit to the saphenous networks reflux from tributaries of the pelvic veins.

This is notably the case for labial, perineal and dorsal veins of the clitoris and penis, which transmit their reflux to the superficial networks of the lower extremities.

The same is true of the refluent veins of the Venus mount supplied by the veins of the round ligament of the uterus, which transmit their reflux to the descending tributaries of the saphenofemoral junction. This is also the case of reflux of the male varicocele in the tributaries of the Great Saphenous Arch.

The Obturator Vein is a special case, which can connect and reflux into the great saphenous arch without a perforator as such.

43244-Escape points

As we have seen, all connections and perforators can be escape points that reverse the hierarchical direction of the flow.

432441- Open vicarious shunts OVS (shunts bypassing Obstacles).

The reflux of the escape points EP of the Open vicarious Shunts OVS by-passes, at rest and/or at stress, according to the resistance of the obstacle.

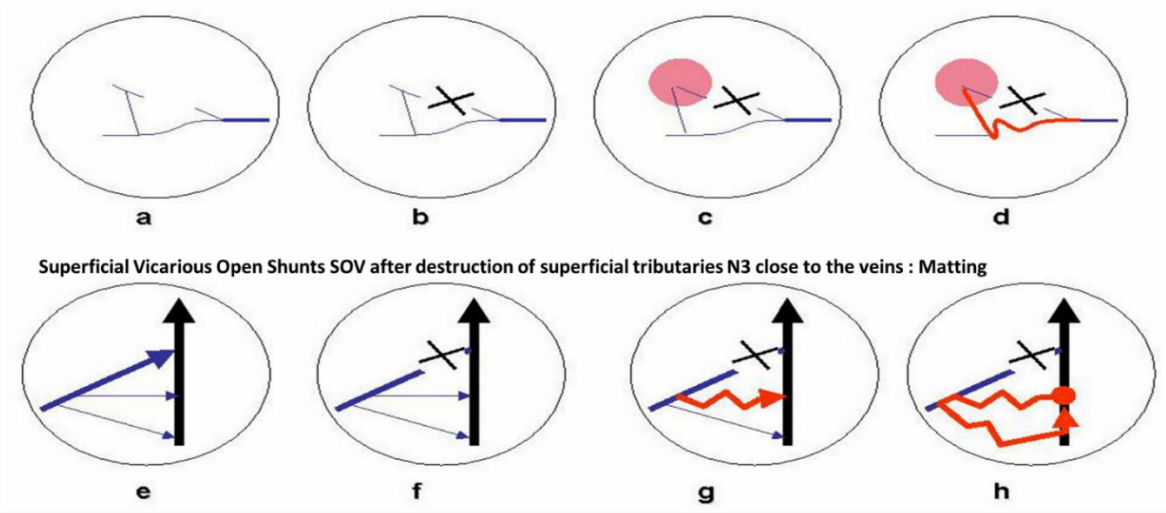
At rest it drains the physiologic flow from the microcirculation, pushed by Residual pressure. During effort, as walking, it flows the systolic blood of the valvulo-muscular pump.

They are most often located at the level of the saphenofemoral junctions (iliac and cava obstacles) and popliteal junctions (femoral obstacle) and leg perforators (popliteal obstacle).

A venous stenosis is hemodynamically significant when it increases the upstream P1 pressure, thus the TMP and its harmful consequences on tissue drainage. For the same geometric structure of stenosis given by static imaging, namely its radius r and length L, the hemodynamic significance resistance R = P1/Q can be null and increase according to the flow Q and the upstream pressure P1. ***The resistance R is equivalent to the pressure drop P1-P2= Q.8Lμ/πN4 (Q=Flow rate, L=Length of the stenosis, μ = viscosity, r = radius of the Poiseuille stenosis calibre)****. For the same anatomic stenosis of radius r and length L for a fluid of viscosity μ, the resistance R increases with the flow rate Q, which increases with P1. This explains why the significance of a venous stenosis increases residual pressure and valvulo-muscular pump pressure, which force and rupture the valves of perforators and tributaries to form open vicarious shunts. These shunts reduce the upstream pressure the lower their downstream resistance (larger r). The value of this compensation is measured by the venous pressure upstream of the shunt, which avoids treating the obstacle when a normal pressure*

attests to the perfect compensation of the obstacle by the OVS. ***This is also why the measurement of the upstream venous pressure at rest and during exercise is essential to evaluate the pathological degree of a stenosis and the value of its compensation to establish rational hemodynamic therapeutic strategy.***

Most are the recurrent varicose veins and telangiectasias that play the role of OVS after extensive ablation of the superficial veins.



Superficial Vicarious Open Shunts SOV after destruction of superficial tributaries N3 distant from the venules: varicose recurrences

432442- *Escape points of closed shunts CS. N1>N2, N1>N3.*

1-The refluxes caused by the diastole of the valvulo-muscular pump (especially of the calf) and the systole of the Valsalva manoeuvre are most often located at the level of the pelvic escape points, saphenofemoral and popliteal junctions, the pelvic and thigh escape points, more rarely at the level of the plateau and the medial face of the tibia. Exceptionally at the level of the perforators of the tibial and gastrocnemius veins.

They are activated by valvulo-muscular pumps by the aspirative diastole of the valvulo-muscular pumps (walking, compression and Paranà).

Sometimes, the expected diastolic reflux from the SFJ and trunk of a dilated great saphenous vein whose valves are destroyed does not occur. This is due to 2 possible causes.

Either there is a hemodynamic obstacle to the re-entry.

Competitive" deep reflux. This is the case when a major deep diastolic reflux refills massively and very quickly the valvulo-muscular pump so that it leaves no room for the volume of saphenous reflux. I call this reflux, "competitive" deep reflux because, although the saphenous valves are incompetent, the reflux cannot occur, the pump being already "occupied" by the deep reflux.

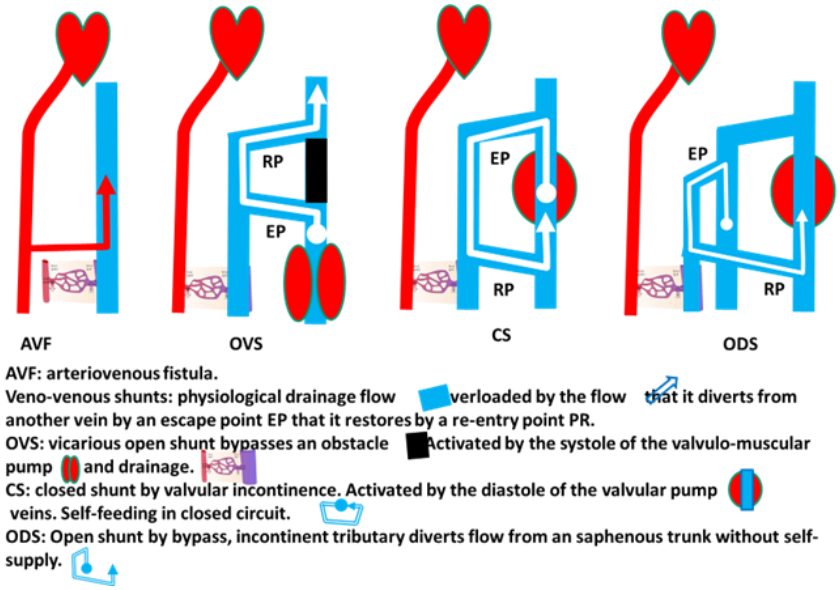
Either there is an <u>organic obstacle</u> in the re-entry pathways (thrombosis/ligation of perforators, deep veins great saphenous segment) i.e., between the great saphenous vein and the valvulo-muscular pump. *That unfortunately exists after non-hemodynamic occlusive treatments that leave the <u>illusion of a "cure" when in fact it is an obstacle to drainage</u>.*

<u>2-Systolic Valsalva Reflux.</u> Refluxes from the CS escape points are also activated but by the downwards repulsive systole of the thoracoabdominal pump (Valsalva) and <u>only through the escape points N1>N2, N1>N2>N3 (not N2>N3 when N1>N2 is competent)</u>. The effectiveness of the Valsalva manoeuvre is reduced from top to bottom.

432443- Escape points of opened deviated shunts ODS N2>N3

The escape points of Open Deviated shunts ODS N2>N3 are activated like those of closed CS shunts by residual pressure and diastole of the valvulo-muscular pumps.

<u>But they are not activated by thoracoabdominal pump systole, namely the Valsalva manoeuvre</u>*. This means that a flow caused by the systole of the thoracoabdominal pump (Valsalva +) affirms a reflux of closed shunts CS N1>N2>N1, N1>N3>N1, N1>N2>N3>N1, N1>N3>N2>N1 etc....* ***A diastolic reflux caused by the valvulo-muscular pump but not reproduced by the systole of the thoracoabdominal pump (Valsalva -) affirms the presence of limited reflux N2>N3>N1 by open deviated shunt or shunt 0, N2>1 or N3>N1 .***



432444- Mixed Escape points.

I call mixed shunt MS <u>the combination of an open vicarious shunt OVS with a closed shunt CS.</u> They share the <u>same escape point</u> EP but their re-<u>entry points are different</u>.

A mixed escape point associates a <u>systolic reflux</u> that feeds an <u>open vicarious shunt OVS</u> followed by a <u>diastolic reflux</u> that feeds a <u>closed shunt CS</u> during the <u>systo-diastolic action of the valvulo-muscular pump</u> (Paranà or Compression-relaxation).

The EP escape point and the first segment of the MS that exhibits systolic plus diastolic reflux are common to the CS and OVS .

Then first segment divides into 2 branches. One flows only the systolic flow of the OVS to its specific downstream re-entry perforator RP. The other drains only the diastolic flow from the CS to its specific upstream re-entry perforator. RP

432445- Re-entry points RP

I call "re-entry points" RP, the perforators, and junctions through which the N2 and N3 flows of the shunts return to N1.

RPs of CS and ODS are most often located below the knee.

RPs of SOVs are most often located above the knee.

In SMs, the escape point is common, but its RPs are different, the OVS RP is usually above, and the CS RP is below the common escape point EP.

These re-entry points RP that should be preserved because of their positive hemodynamic function are still too often confused with EP escape points, and therefore sadly removed.

The error is often due to the small innocent systolic refluxes that precede the beneficial diastolic re-entry flow are mistakenly considered pathogenic whereas they are victims of the shunts!

4325. Shunts. Detailed classification.

Why know about veno-venous shunts?

Knowing, understanding, and recognizing each veno-venous shunt means getting to the heart of the physiopathology.

It allows us to take advantage of the essential diagnostic possibilities of the echodoppler to establish the tailored hemodynamic mapping specific to each patient.

Then, this mapping allows to elaborate the best possible therapeutic strategy followed by a specific minimally invasive tactic in terms of approach technique and material.

Recognizing closed CS and open deviated shunts ODS allows precise disconnection of escape points and preservation of re-entry points, without destroying the incompetent veins, because they will return to normal calibre within a few weeks.

Recognizing the open vicarious shunts is to preserve them, thus avoiding an aggravation of the disease.

My first classification in 1988 was extended at the CHIVA meeting in Teupitz, Germany.

The groups are differentiated by their EP escape points and the subgroups by their paths. This classification is not only descriptive. It is useful for studies and therapeutic trials.

Indeed, the hemodynamic conditions are specific to each group and must be specified in the diagnostic and therapeutic evaluations, whatever the method or technique applied.

Reminder:

A veno-venous shunt is a vein whose physiological drainage flow is overloaded by a flow that its "steals" from another vein through an EP escape point and that it restores through a re-entry point. These shunts have the common characteristic of reversing the physiological hierarchy of drainage between the various networks and compartments. They differ according to the conditions of their activation (rest, dynamic diagnostic manoeuvres (Paranà, Valsalva) and their effects on the Transmural pressure TMP

43251-Superficial shunts

432511- Closed shunts CS.

Closed shunts CS are activated by the diastole of the valvulo-muscular pump and Valsalva (systolic Valsalva reflux +).

Escape points.

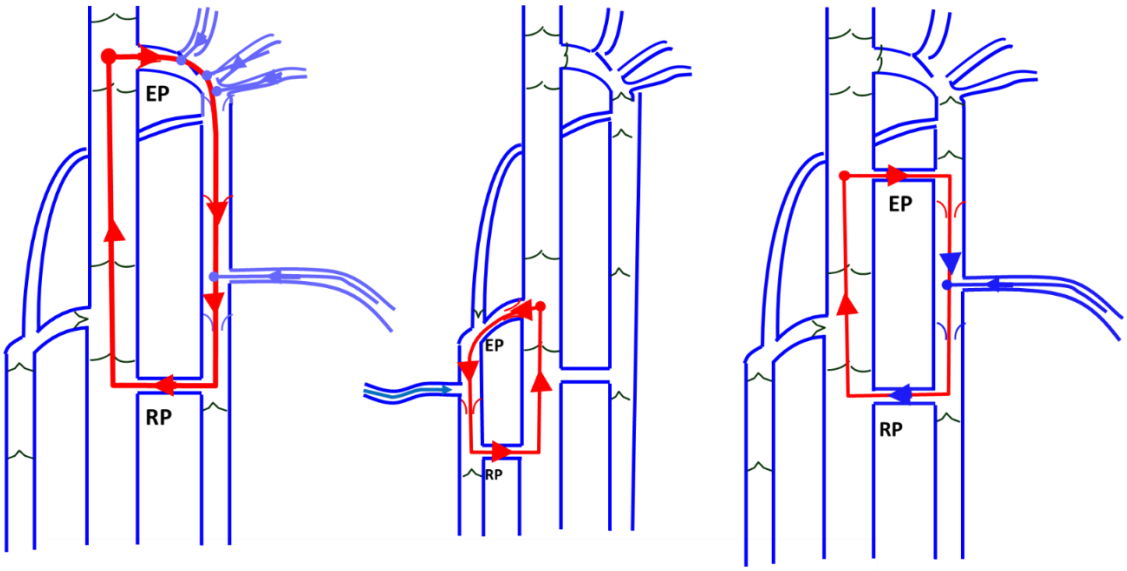
The escape points are fed by the deep network N1. They first overloads N2 (N1>N2) or N3 (N1>N3) to return into N1 through a re-entry point (N2>N1 or N3>N1) after a variable pathway depending on the shunt type (Shunts I, III, IV, V, VI and MS).

SHUNT TYPE I.N1>N2>N1

-Escape point EP N1>N2: Saphenofemoral junction or perforating vein of the thigh or leg trunk.

-Pathway: N2

-Re-entry: N2>N1



SHUNT 1:Closed Shunt N1>N2>N1: 3 examples of closed SHUNT CS . Recirculation. N2 Overload by N1
3 examples
EP:Escape Point, SFJ, SPJ and Thigh Perforatot. RP: Reentry Points

SHUNT TYPE III:

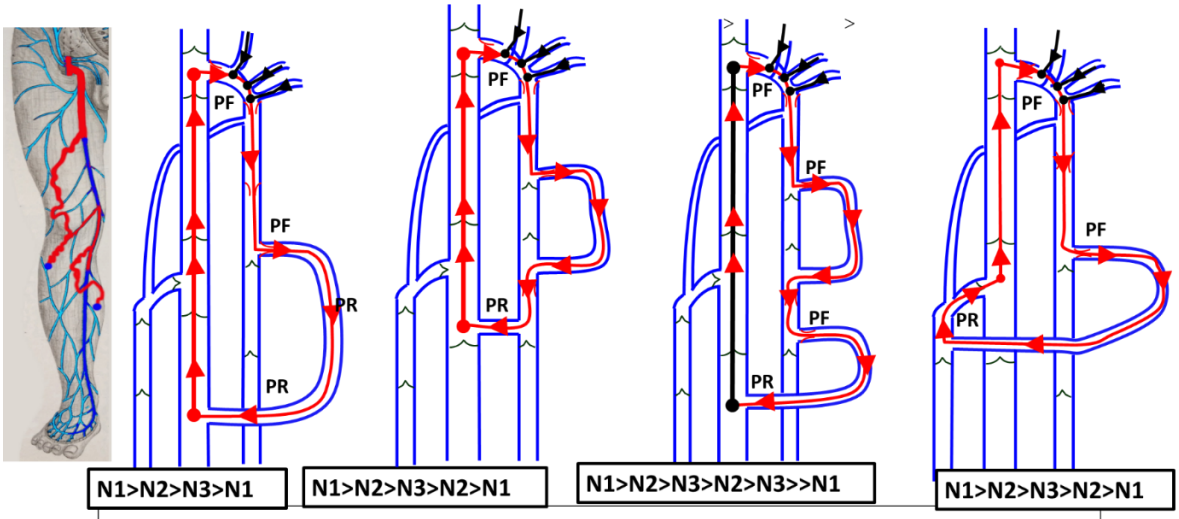
- ***Escape point EP N1>N2: Saphenofemoral junction or thigh or leg trunk perforator.***

-Pathway: N2>N3.

-Re-entry N1

Note that there is <u>no N2>N1 re-entry point on the N2 saphenous trunk</u>, i.e., between N1 and N3.

Shunt III subgroups according to the pathway: N1>N2>N3>N1, N1>N2>N42>N2>N1, N1>N2>N4L>N3>N1, N1>N2>N4T>N2>N1.



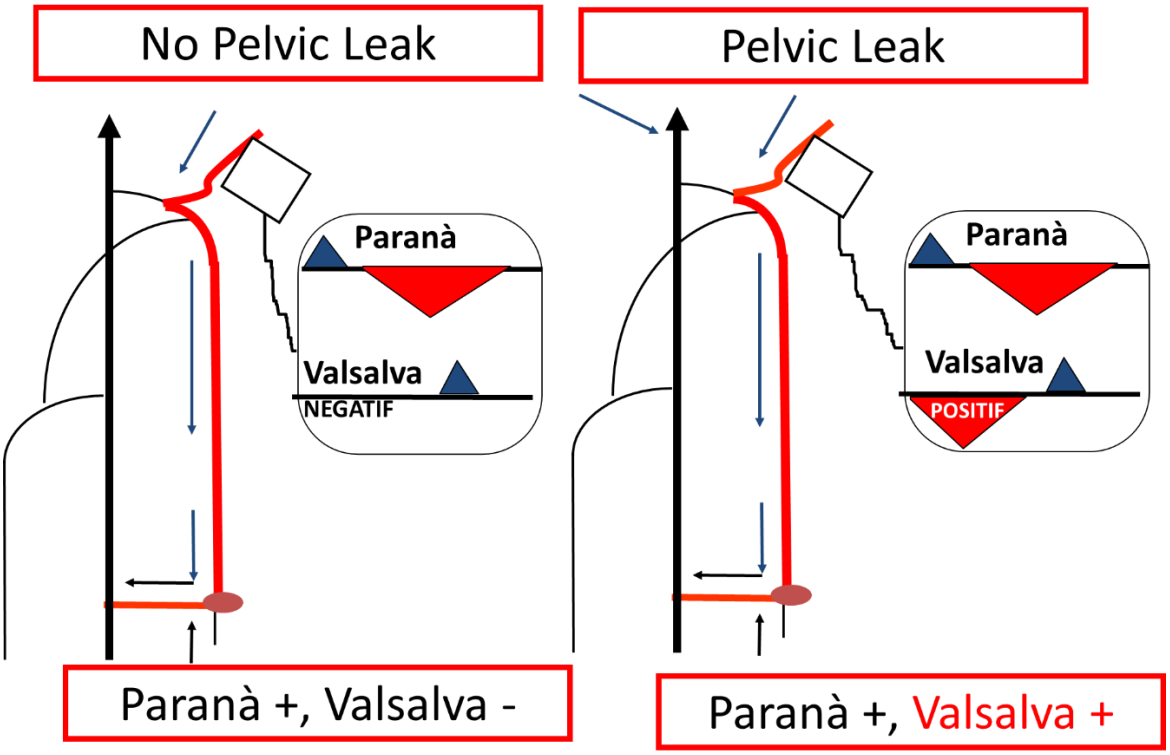
SHUNT Type III closed CS . Recirculation. Type III N1>N2>N3>N1: Overload by N1 . No Re-entry N2 between N1 and N3.

SHUNT TYPE IV: N1>N3>N2>N1

-Escape point EP: N1>N3 Pelvic escape points.

-Pathway: N3>N2

-Re-entry: N2>N1

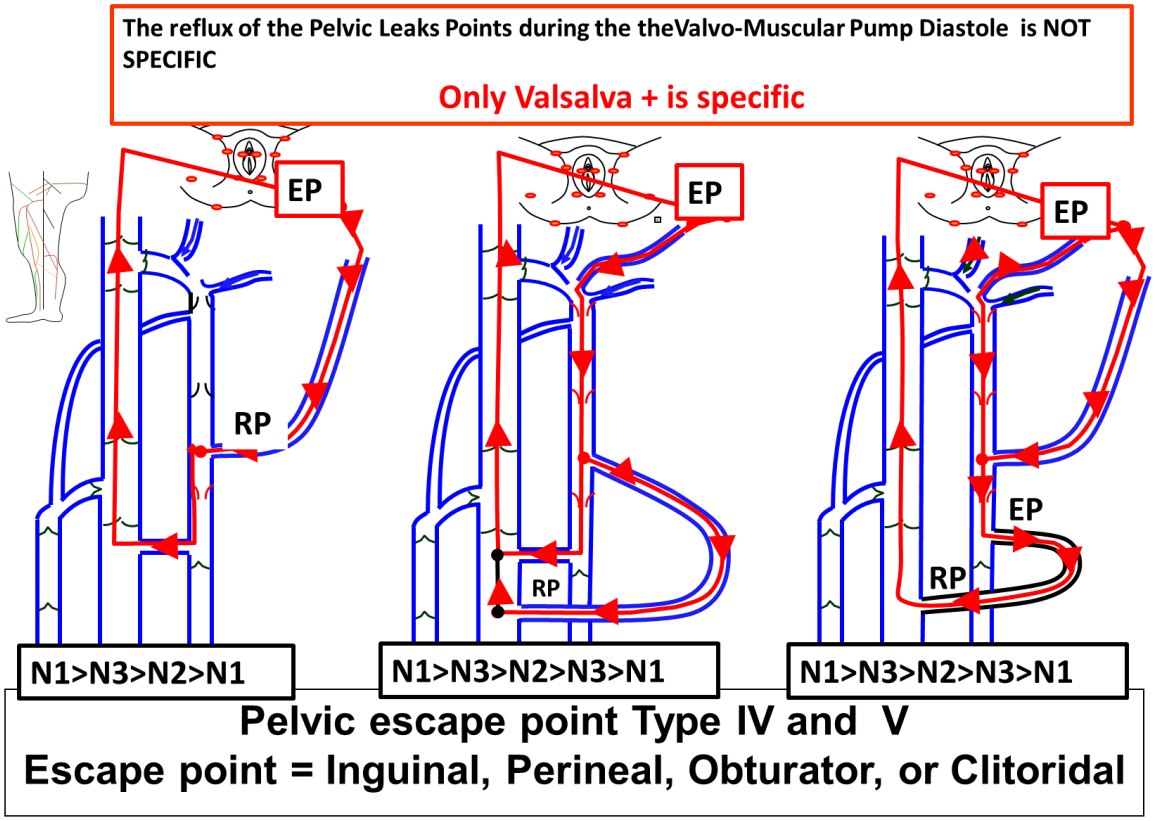


SHUNT TYPE V:N1>N3>N2>N3>N1

-Escape point N1 EP: Pelvic escape points.

-Pathway: N3>N2>N3

-Re-entry: N3> N1

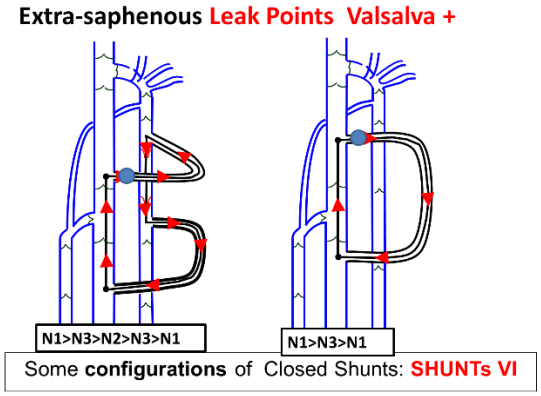


SHUNT TYPE VI

Escape point EP: Extra-saphenous perforator.

Path: N3 or N3>N2

Re-entry N1



SHUNT Type II. Open Deviated Shunt ODS

Superficial escape point N2>N3 TYPE II SHUNTS, not supplied by N1, are shunts opened by ODS by-pass and more rarely closed CS:

<u>Activated by diastole of the valvulo-muscular pump but negative Valsalva (not systolic Valsalva reflux)</u>.

Escape point EP:-N2-N3 junction.

Pathway: N2> N3.

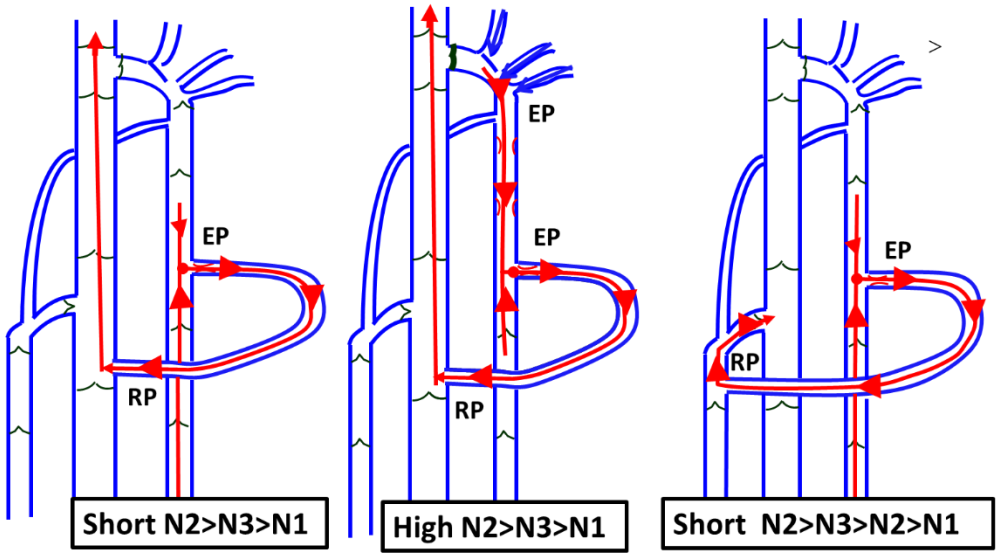
-Re-entry RP: N3>N1.

Subgroups:

SHUNT Type II a N2>N3>N1 without N2 incompetence.

SHUNT Type II B N2>N3>N1 with N2 incompetence, but without EP N1>N2 nor RP N2>N1.

SHUNT Type II C: N2>N3>N1 : common N2 but different N2>N1 and N3>N1 re-entries.
SHUNT N4L closed but without deep escape point: EP escape point: N2-N3 junction. path: N4L. Re-entry N2: N2>N4L



SHUNT open by deviation ODS. No Recirculation.
Type II N2>N3>N1 and N2>N3>N2>N1 : N3 Overload by N2 3 examples
EP = Escape point N2>N3 PN = Re-entry points N3>N1 and N3>N2

432512- *Shunts 0 with no diastolic escape point:*

By definition, a shunt without an escape point cannot be called shunt because it is not overloaded and does deviate any flow. However, we have called Shunt 0 the retrograde flows not overloaded and obeying the hierarchical direction of the drainage.

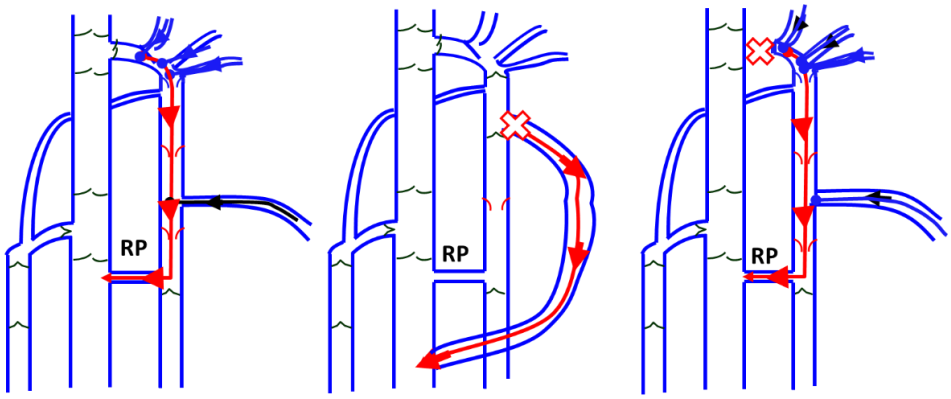
SHUNT TYPE 0:

-No escape point EP:

-Pathway: N2 or N3

-Re-entry RP: N2>N1 or N3>N1

***Activated by diastole of the valvulo-muscular pump and <u>Valsalva negative</u> (no systolic Valsalva reflux*).



SHUNT O : N2>N1 segmental reflux not overloaded

SHUNT O : N3>N1 post disconnection N2>N3 of an open deviated shunt type II (CHIVA)

SHUNT O : N2>N1 post disconnection N1>N2 of a closed shunt Type I (CHIVA)

Shunt Type 0 NON pathological
No EP= Leakage points N1>N2 nor N2>N3 nor N1>N3 nor N2>N3
PR = Reentry points N2>N1 and N3>N1

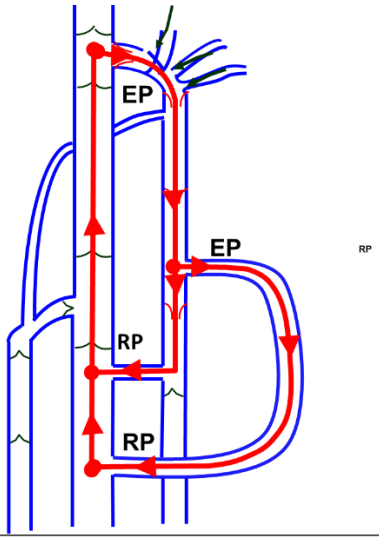
432513- *Combined superficial diastolic shunts:*

Combined superficial diastolic shunts connect with staggered escape points and common segment of pathway.

SHUNT I + II: N1>N2>N1 + N2>N3>N1, N2 common

SHUNT I + IV.: N1>N2>N1 + N3>N2>N1, N2 common

SHUNT III + V.: N1>N2>N3>N1 + N3>N2>N3>N1, N2 and N3 common



Combined Shunt Closed Type I + Open by Deviation Type II
EP= Escape points N1>N2 and N2>N3 PN = Re-entry points N2>N1 and N3>N1

432514- *Superficial Open Vicarious Shunts OVS*

1- Superficial Open Vicarious Shunts OVS by superficial obstacle N2(constitutional or acquired or iatrogenic):

N2>N3>N2>N1,

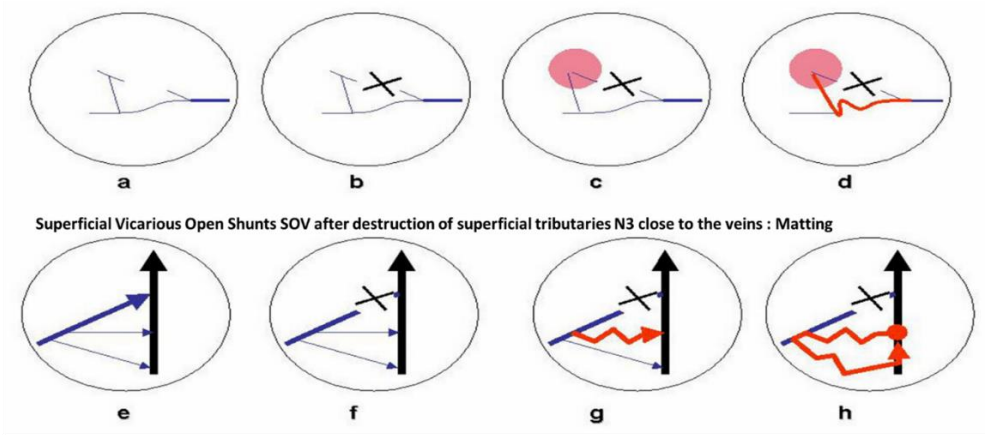
Micro-circulation>venules>N3>N1,

Micro-circulation>venules>N3>N2>N1.

Note that the escape point is not N1>N2 nor N2>N3 <u>but N1 is replaced by microcirculation and venules.</u>

These shunts are essentially seen <u>in post venous destructive treatments</u> that trigger collateral pathways.

when the obstacle is close to the microcirculation, the resistance is such as to open micro arterio-venous shunts represented by <u>"matting"</u>



Superficial Vicarious Open Shunts SOV after destruction of superficial tributaries N3 distant from the venules: varicose recurrences

2-Superficial Open Vicarious Shunts OVS by deep obstacle N1:

N1>N2>N1, N1>N2>N3>N1, N1>N3>N1 etc. according to the succession of vicarious superficial veins.

432515- *Mixed Shunt: MS. N1>N2 common systolic and diastolic escape point EP and different re-entry points.*

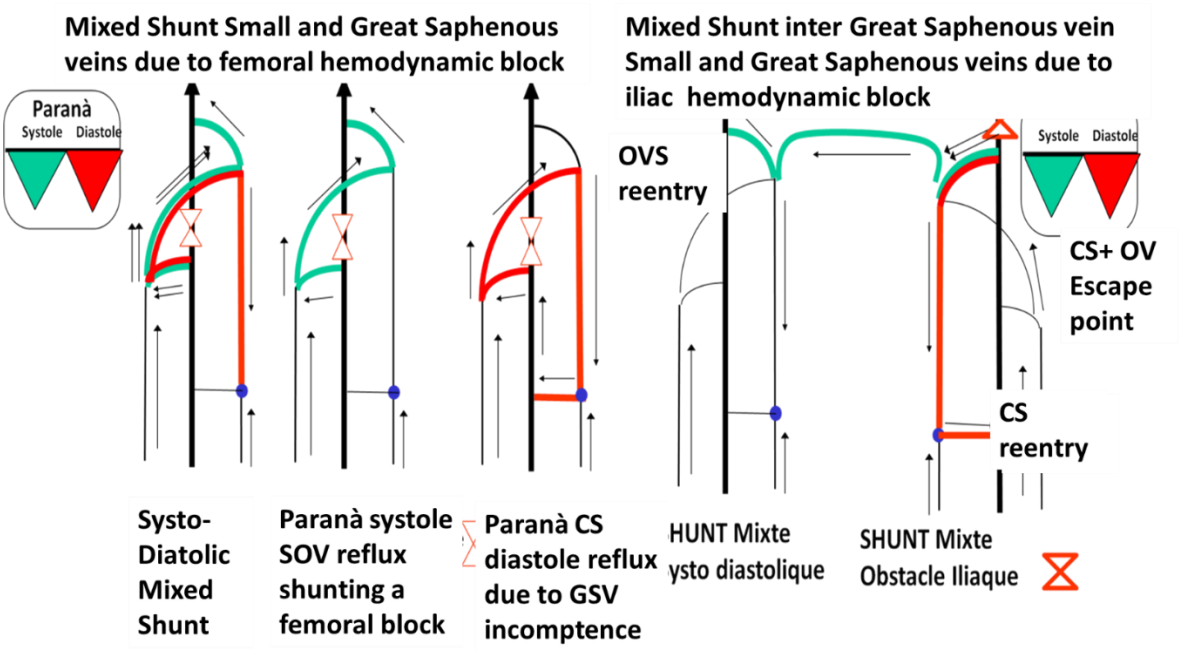
A venous shunt is mixed MS when it combines an open vicarious shunt OVS with a closed shunt CS.

OVS and CS have in common an escape point N1>N2 and a first segment N2 that reflux in both systole and diastole. Then N2 divides into 2 veins A and B (N2 and/or N3) that connect two different re-entries C and D (N2>N1and/or N3>N1). During the systole, the vein A flows upwards only the OVS to the re-entry C. During the diastole, the vein B flows backwards the CS to the re-entry D. As a matter of fact, OVS one is activated only in systole and the CS only in diastole.

They are mainly seen in 2 cases of hemodynamic obstruction.

- ***<u>Obstacle of the superficial femoral vein</u>, acquired or congenital (stenosis at the level of Hunter's canal that I had defined with the Doppler then confirmed later in the cadaver (Uhl).***

-<u>Iliac obstruction compensated by a spontaneous Palma</u> via the arch of the right and left great saphenous veins, combined with a Great Saphenous V CS.



432516- *Classification of diastolic deep shunts*

Deep veins present closed shunts when an <u>incompetent deep venous segment A is connected by its two ends to a competent deep vein B. A sucks in B during diastole</u>.

This is often the case for closed shunts of <u>the superficial femoral vein</u> overloaded during diastole either by its competent collateral or by the deep femoral vein. This is also the case for closed shunts of the <u>posterior tibial vein</u> overloaded either by its competent collateral or by the competent peroneal (fibular) vein.

43252. Practical and theoretical shunts

This detailed classification is useful to define each type of shunt and the hemodynamic feature <u>in addition to the clinical CEAP</u> in studies and trials.

In clinical practice, we just must <u>remember and apply the basic principles</u>:

1--to look for

-the escape point that overloads the veins,

-the pathway of the shunt,

-the re-entry point where they drain flow in,

-according to the systolic and diastolic phases of the Valsalva and Paranà manoeuvres.

2-- Then report this topographic and hemodynamic data on a map.

-This cartography (mapping) will be crucial as well as for the strategy (what to do) as for the tactics (how to do) of the hemodynamic treatment as CHIVA according to the shunts

-closed shunts CS,

-open deviated shunts ODS

-open vicarious OVS

-mixed shunts MS

Chapter 5

Each chapter includes some of the elements of the previous chapters and anticipates those of the following chapters.

5- Hemodynamic Pathophysiology of Venous Insufficiency

51- Venous insufficiency due to valvular incompetence

511-Physiologic venous insufficiency due to impaired Dynamic Fractioning of gravitational hydrostatic pressure DFGHSP

512-Pathological venous insufficiency due to lack of Dynamics Fractioning of gravitational hydrostatic pressure DFGHSP

5121- Incompetence of the deep femoro-popliteal veins and leg veins

5122-Incompetence of superficial or collateral veins

5123-Incompetence of superficial and deep veins

5124-Pelvic incompetence

51241-Varicocele

511411-Female varicocele

512412-Male varicocele is rarely associated with Nutcracker

51242-Incompetence of the visceral tributaries of the hypogastric vein

512421-Internal Pudendal vein:

5124211-In females, Internal Pudendal Vein.

5124212-In males, internal pudendal vein.

512422-The vein of the round ligament of the uterus.

512423--Varices of the broad ligament

512424--Hemorrhoidal vein and "haemorrhoids

51243- Incompetence of the parietal tributaries of the hypogastric vein

512431--The vein of the round ligament

512431--The obturator vein,

512432- The superior gluteal vein

512433--The inferior gluteal vein (also called ischiatic vein).

52- Venous obstructions

521-surfacevenous obstructions

5211-surfacedermo-hypodermal venous obstructions

5212-surfacevenous obstructions due to venous destruction

522- Deep venous obstructions

5221-Pelvic venous obstruction

52211-Nutcracker syndrome or aorto-mesenteric clamp

52212- May Thurner Syndrome MTS

5222- Iliac and/or vena cava thrombosis or agenesis

5223-Deep venous obstructions of the lower extremities

5224- Associated deep and superficial shunts

523-Thoracoabdominal obstruction

524-Cardiac obstruction

525- Reflux and inflammation

526- Veno-lymphatic insufficiency

5261- Impaired lymphatic drainage due to venous insufficiency

5262- Venous drainage altered by lymphatic insufficiency

527-Varicogenesis

528-Remodeling

53-Venous ulcer

54-Venous malformations

55-Hierarchy of networks and derivations

551- Hierarchy of networks

552- Venous-venous shunts

5521- -Superficial shunts

55211- Closed superficial shunts CS

55212- Open shunts for open shunts ODS

55213- Shunts O

55214 - Combined diastolic Superficial shunts

55215 - Combined diastolic Superficial shunts

55215- Superficial systolic shunts OVS

55216 - Mixed superficial shunts: MS

55217 - Classification of deep diastolic shunts

55218.Perforators

552189 Practical and theoretical shunts

5- *Hemodynamic Pathophysiology of Venous Insufficiency*

Transmural Pressure TMP is the core of pathophysiology

TMP= IVLP-EVP

IVLP : Intravenous lateral Pressure, EVP : Extravenous Pressure.

The aetiology of venous disease is multiple,

but the hemodynamic causes are limited:

to 2 conditions:

1.Valvular incompetence and/or

2.Obstruction

And one effect:

Increase of the Transmural pressure (TMP).

That is the case for congenital, hereditary diseases such as venous malformations, or acquired diseases such as thrombophlebitis, or even "family" diseases such as so-called essential varicose veins.

Any accident or iatrogenic medical or surgical act that occludes or destroys a vein can also disrupt venous function, as demonstrated by varicose recurrences after ablative non-hemodynamic ablative procedures.

The hemodynamic bases of the production and effects of venous flows and pressures on venous function and its insufficiencies are amply detailed and explained in their clinical and therapeutic context in the preceding chapters. The reader can simply refer to them. These basics are repeated more succinctly in this chapter

***1-Hemodynamic venous insufficiency** is the consequence of <u>a permanent or transient excess of Transmural Pressure TMP</u> due to <u>dysfunction</u> of one or more components of <u>the venous system</u>.*

Normal TMP = no hemodynamic venous insufficiency.

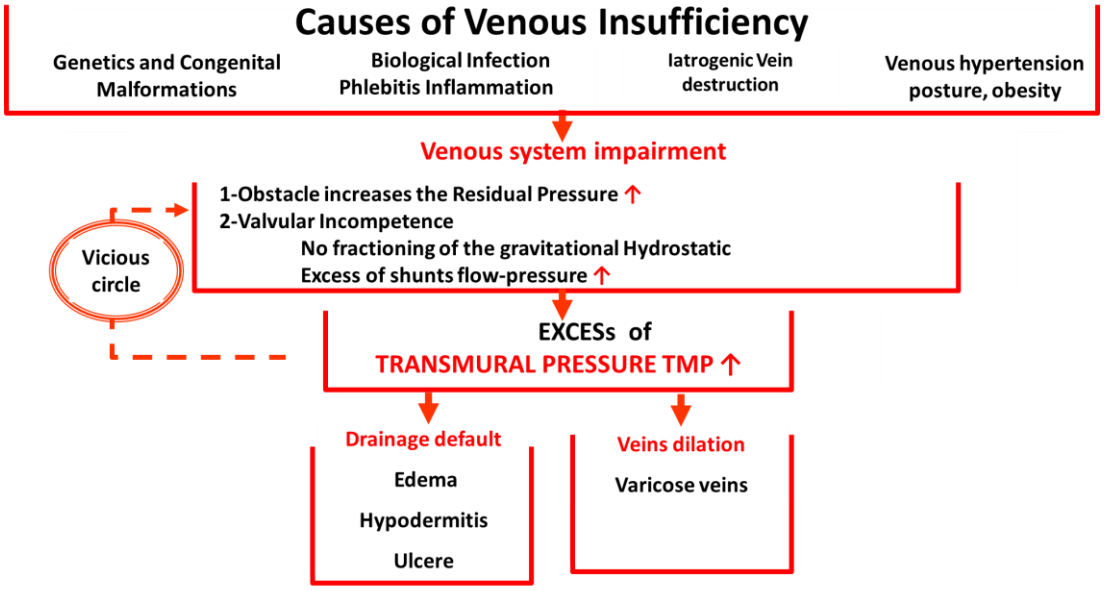
High TMP = Hemodynamic venous insufficiency.

***2-Diagnosis**: Find the causes*

***3-Treatment**: Treat the <u>causes WITHOUT degrading the functions</u>, especially drainage.*

***4-Necessary conditions**:*

To know and understand the mechanisms that regulate of the TMP



Although identical, the signs of venous insufficiency are related to different alterations of the venous system that must be diagnosed and treated specifically

The normal transmural pressure TMP of the veins and microcirculation is about 10-15 mmHg when lying down along the lower limbs, and 90 mmHg when standing still and 35 mmHg when walking at the ankle...

-This condition is the necessary and sufficient condition to ensure the venous functions.

*-It is the result of the **balance between the lateral intravenous pressure (IVLP) and the extravenous pressure (EVP)**.*

The Intra-Venous Lateral pressure (IVLP) is the sum of

Pressure produced by gravity

- Hydrostatic Gravitational pressure (GHP) and

-Pressures produced by the

-Cardiac pump CP

-Thoracoabdominal pump TAP and

-Valvulomuscular pumps VMP.

Pumps can aggravate or correct excessive Gravitational Hydrostatic Pressure GHSP

Veno-venous shunts

-Open vicarious shunts OVS

reduce TMP

-Closed shunts and Open deviated shunts

Increase TMP

Diagnosis according to the pumps phases

Closed shunts

- Valsalva positive

-Paranà positive

-Open deviated shunts

- Valsalva negative

-Paranà positive

--Mixed shunts

Systo-diastolic shunts (Mixed shunts) increases TMP by its CS shunt and reduces it by it OVS

Lifestyle also affects Transmural pressure (TMP):

-Pregnancy,

- Body position,

- Physical activity,

-Ambient heat and

-Work ergonomics.

The Extra-Venous pressure EVP depends on

-Tissues surrounding the veins,

- Microcirculation oncotic pressure gradient OPG

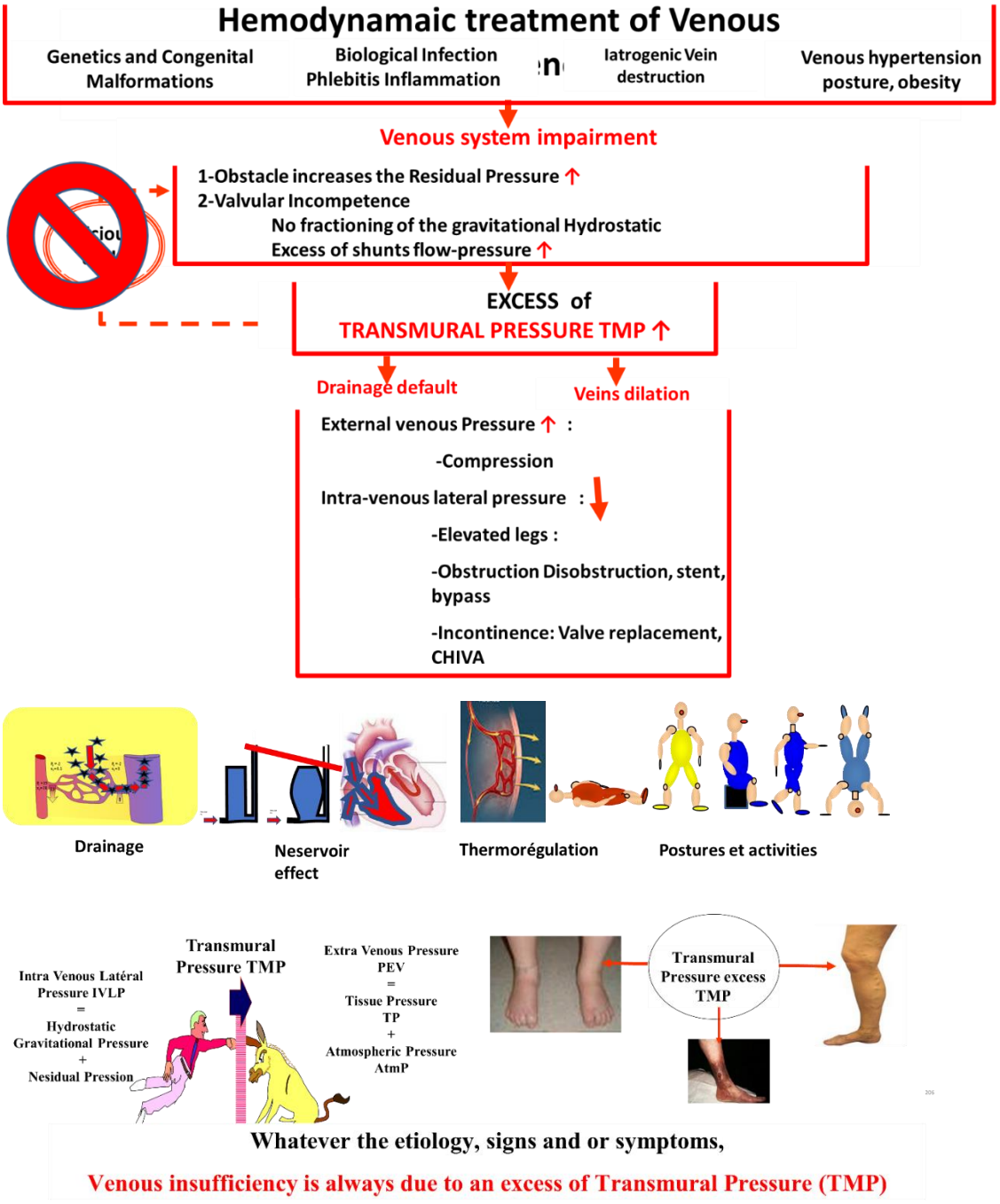
- Atmospheric pressure.

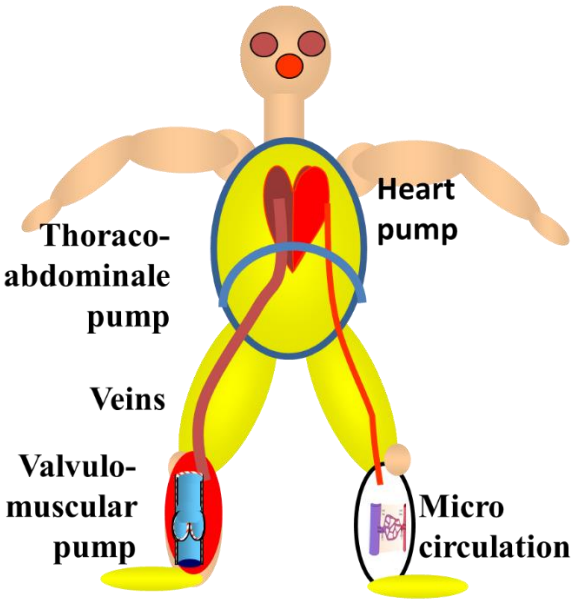
***The selective diagnosis** of each cause depends essentially on*

-Understanding and knowledge of venous hemodynamics

-Rational and method of echodoppler mapping.

The therapeutic strategy can then focus on the correction of TMP.





5 Organs of the venous system

LIVP

1-Gravitational hydrostatic pressure: GHSP Proportional to the height of the blood column.

2- Static component

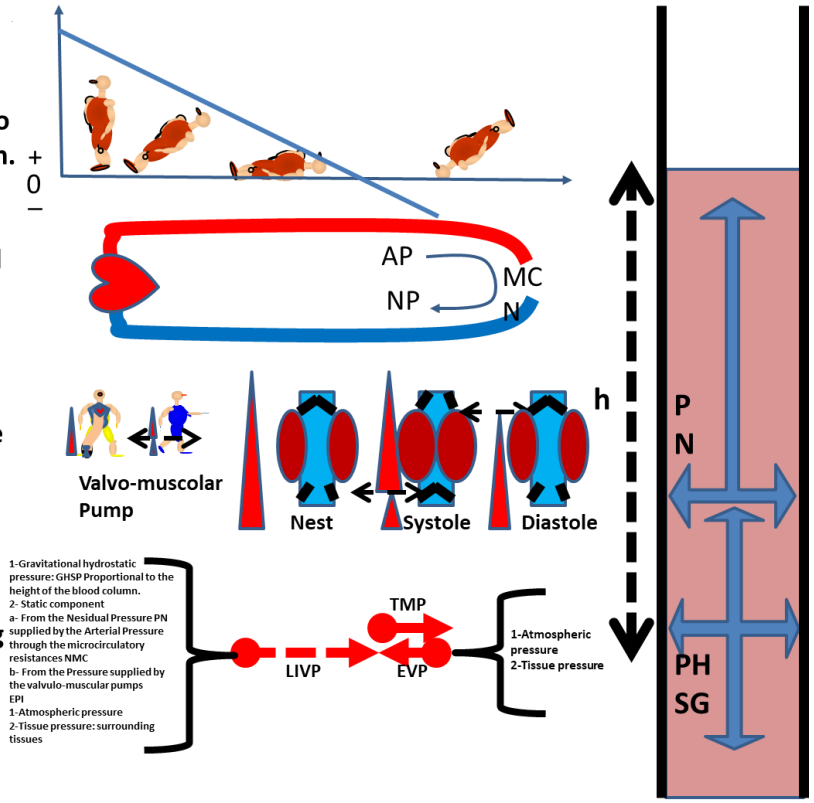
a- From the Nesidual Pressure NP supplied by the Arterial Pressure through the microcirculatory resistances MCN

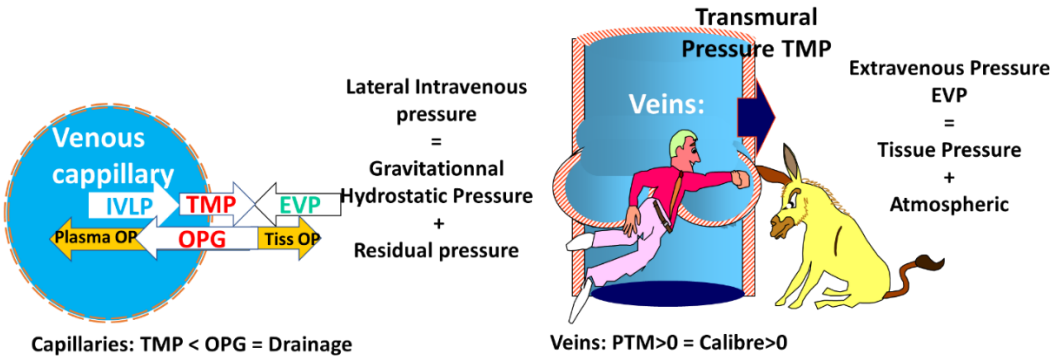
b- From the Pressure supplied by the valvulo-muscular pumps

EVP

1-Atmospheric pressure

2-Tissue pressure: surrounding tissues





TMP and Tissue Drainage
Tissue Drainage requires a low TMP, lower than the Oncotic Pressure Gradient (OPG) between the interstitial tissue fluids and the plasma

In this chapter I will describe the causes of excess Transmural pressure (TMP):

1. valvular incompetence

-Physiological valvular incompetence

- Deep valve incompetence

Superficial Valvular incompetence

-Closed shunts CS and open deviated shunt.

2. resistance to flow

-venous obstructions and open vicarious shunts OVS

-right atrioventricular and thoracoabdominal hypertension

3 Valvular incompetence associated with flow obstructions

4-Capillary and venous hemodynamics

5-Deactivation of the cardiac pump

6-Venous malformations

7-Lifestyle

51- *Venous insufficiency due to valvular incompetence*

511-*Physiological venous insufficiency due to the impairment of Dynamic Fractioning of Gravitational Hydrostatic Pressure DFGHSP*

In the healthy subject, *in the sitting or standing position, the physiological non-closure of the valves maintains a* ***too high column*** *of gravitational hydrostatic pressure GHSP.*

Fortunately, walking fractions this column *by alternate* ***systo-diastolic closure of the valves of the valvulo-muscular pumps****, in particular those of the calf that I called* ***Dynamic Fraction of the Gravitational Hydrostatic pressure DFGHSP****.*

If the immobile sitting or standing *position is maintained for* ***too long****, it causes the* ***clinical*** *manifestations of* ***venous insufficiency****.*

The gravitational "intravenous haemorrhage" *in the veins of the lower extremities steals blood volume/pressure of the cava vein,* ***exceeds the corrective possibilities of the reservoir effect which disables the cardiac pump****. (Tlt test aims to assesses this cause of brief loss of consciousness by setting the patient in a still vertical position).*

If the lifestyle, which includes prolonged standing and sitting postures unfavourable to the DFGHSP, *is prolonged over several months and years, the* ***clinical signs of excess TMP*** *appear according to the microcirculatory predispositions. (See microcirculation chapter)*

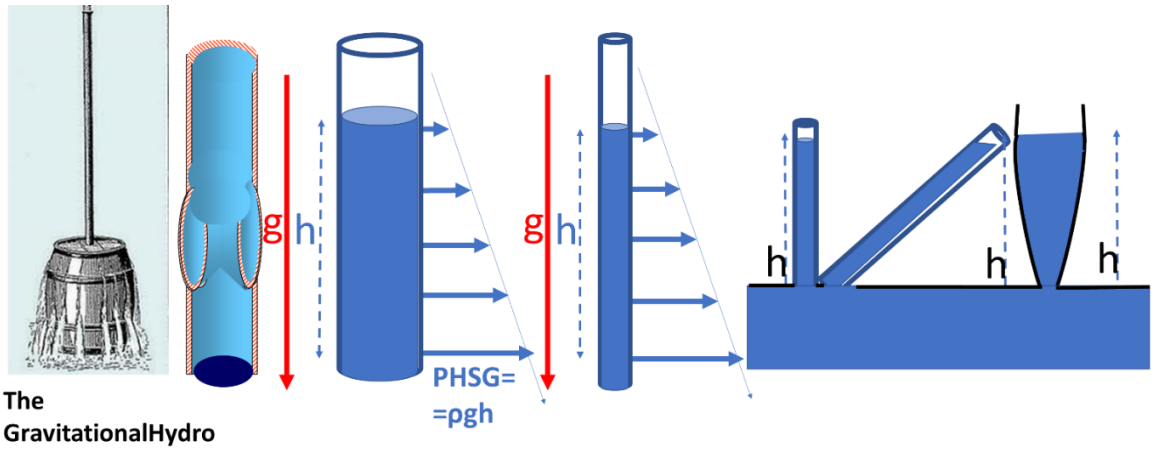
Moreover, if blood stasis destroys the valves *by inflammation, the subject evolves towards the "pathological" form of venous insufficiency by valvular incompetence****.***

512-*Pathological venous insufficiency due to a lack of Dynamic Fractioning of Hydrostatic Gravitational Pressure DFGHSP* Ref: C Franceschi, M Cappelli, JM Escribano, E Mendoza - Fractionnement dynamique de la pression hydrostatique gravitationnelle Vasculab Journal of Theoretical and Applied Vascular Research (page 5) - JTAVR 2020;5(2) 3.3.2 **FRANCESCHI C.: The conservative and hemodynamic treatment of ambulatory venous insufficiency Phlebologie. 1989 Nov-Dec;42(4):567-8.**

The DFGHSP defect may be due to:

- to ***deep incompetence*** *of the inlet and outlet veins of the valvulo-muscular pumps*

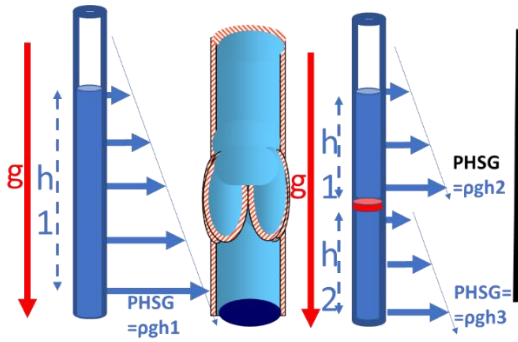
-but also, to incompetence of a deep collateral or ***superficial veins*** *which, by maintaining an unfractionated column in parallel, "shunts" the deep competent veins of the pump.*



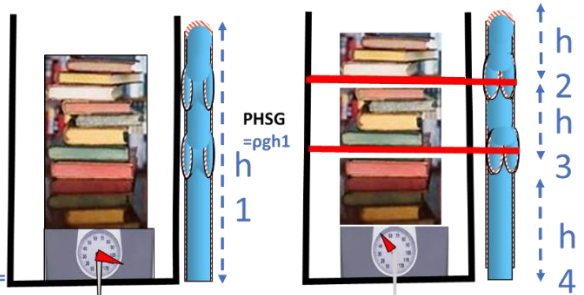
The GravitationalHydrostatic Pressure GHSP= =ρgh depends only on the height of the pipe whatever the gauge and "bursts" the barrel.

Gravitational Hydrostatic Pressure GHSP = =ρgh whatever the vein size

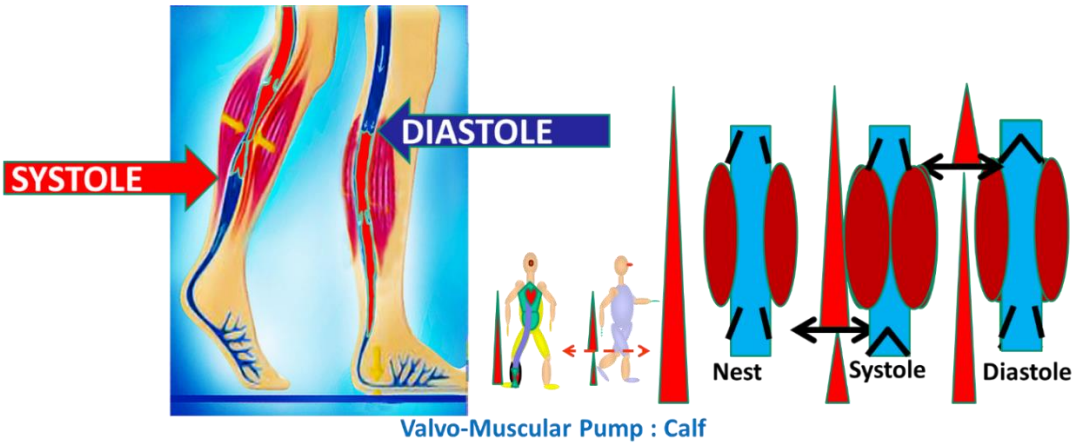
GHSPρgh depends only on the height h independently of the shape , size and volume



Gravitational Hydrostatic Pressure PHSG= =ρgh reduced because the height is split and GHSP 1 and GHSP 2 by closure of the valve or ligation

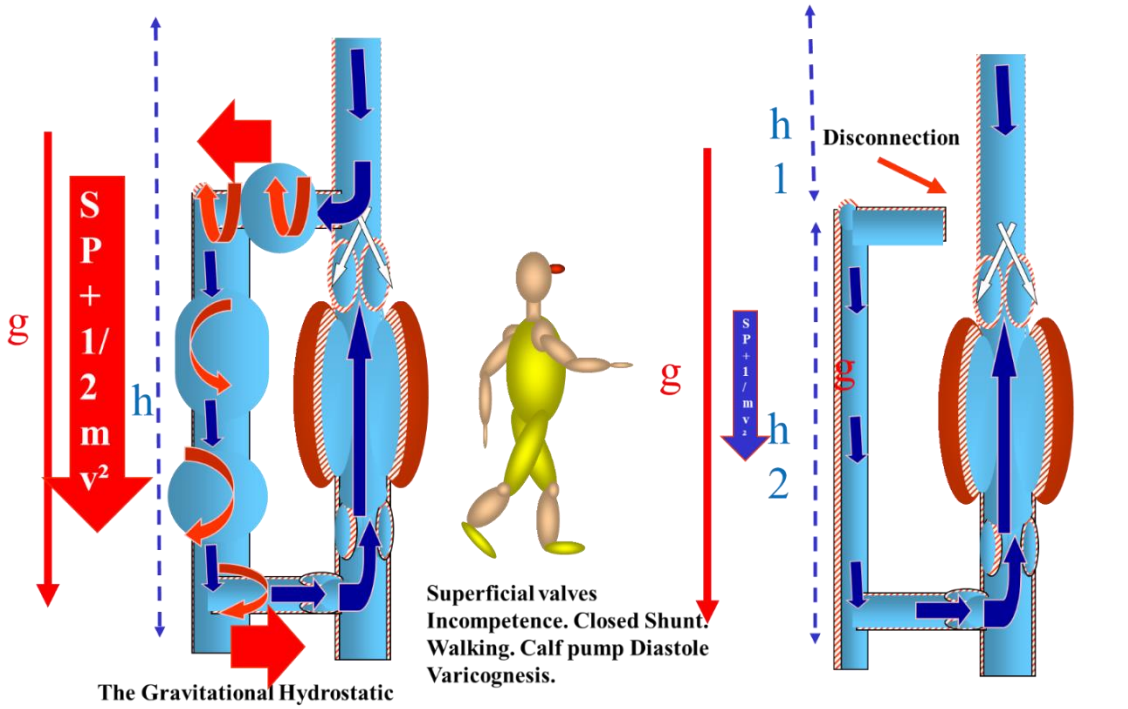


Dynamic Fractionation of GHSP , DFGHSP. Diastolic valve closure splits PHSG ρgh1 (open valves) into ρgh 2,3, and 4 like shelves split the weight of books



Valvo-Muscular Pump : Calf

Dynamic Fractionation of Gravitational Hydrostatic Pressure (DFGHSP): Successive and alternate closure of the upstream and downstream valves of the muclar pumps during walking.



The Gravitational Hydrostatic Pressure GHSP =ρgh + Closed shunt diastolic load (Psat+ ½ mv²) with turbulence more varicogenic than single GHSP

Disconnection of the closed shunt.
Splitting of theGHSP h into h1 and h2 + suppression of the closed shunt overload. Caliber normalization

Dynamic Hydrostatic Gravitational Pressure Fractionation DFGHSP

The DFGHSP defect may be due to:

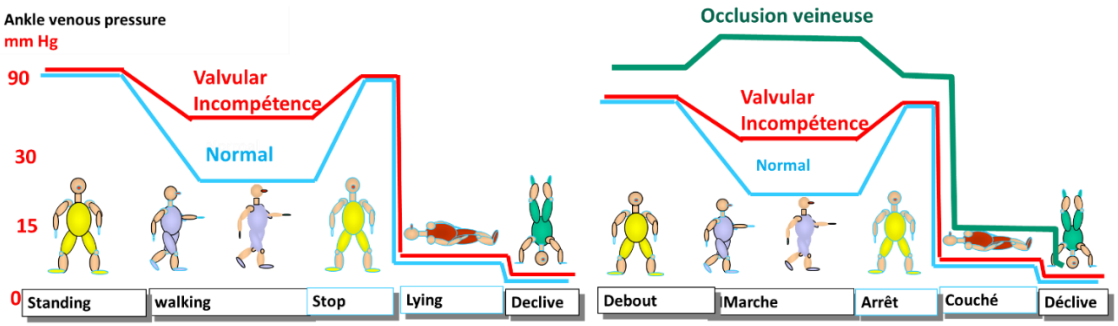
- to deep <u>incompetence of the inlet and outlet veins of the valvulo-muscular pumps</u>

-but also, to <u>incompetence of a deep collateral or superficial veins</u> which, by maintaining an unfractionated column in parallel, "shunt" the deep competent pump veins

*Absence or **incompetence of the outlet the valves** of the valvulomuscular pump produces diastolic reflux that **prevents diastolic fractionation** at its level.*

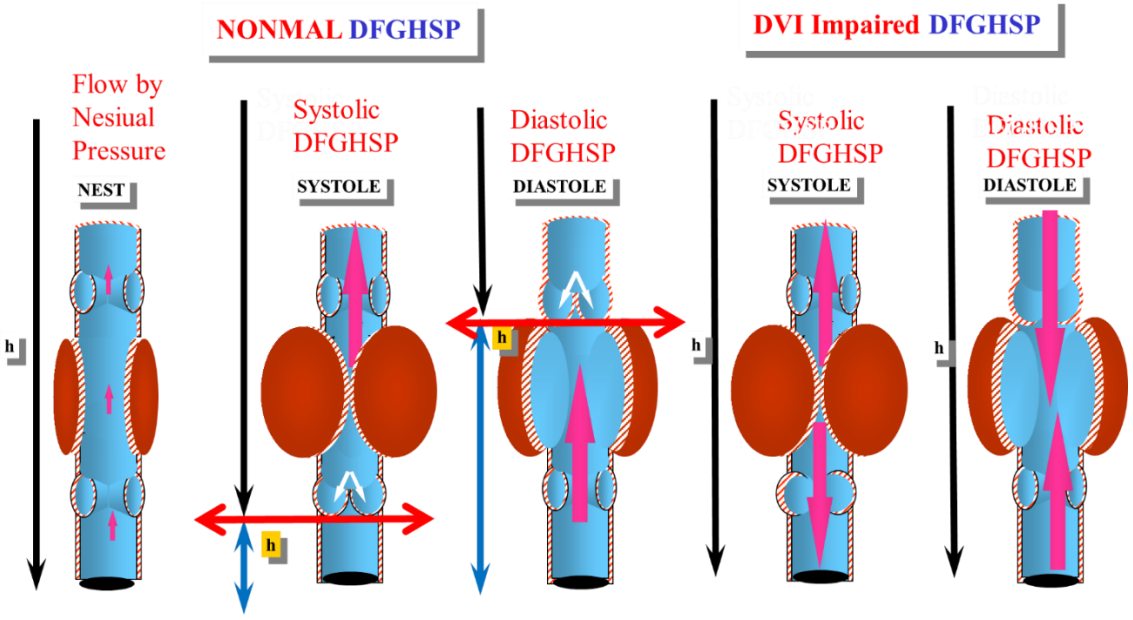
*Absence **or incompetence of the inlet valves** produces **systolic reflux** at its level.*

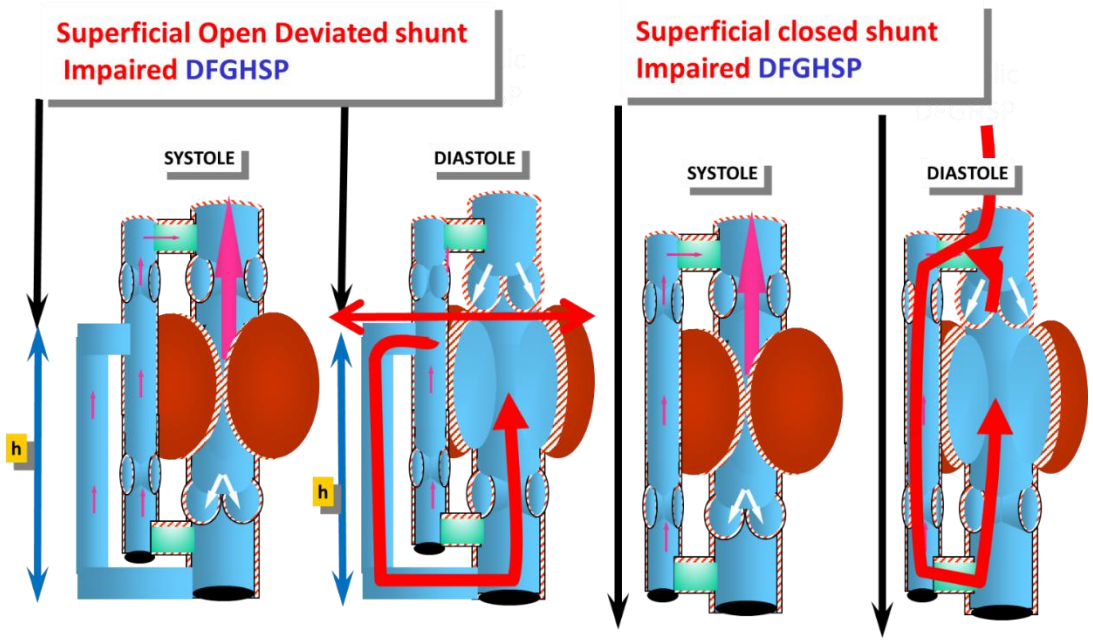
***The degree of Dynamic Fractioning Gravitational Hydrostatic pressure insufficiency** depends on the height of the incompetent column upstream and downstream of the pump and the degree of valve degradation.*

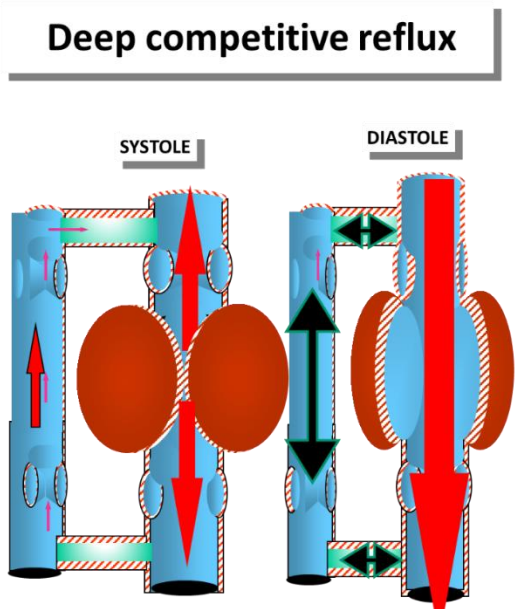


Valvular Incompetence impairs the Dynamic Fractionation of the Gravitational Hydrostatic Pressure DFGHSP . It is not pathogenic at rest, but only during walking !!!!

The obstacles are pathogenic at rest and especially during walking: venous claudication







Competitive deep reflux. In case of deep and superficial valvular incontinence, deep reflux dominates and prevents, when it is major, the reflux of the greater saphenous vein, even though it is varicose.
No reflux of the great saphenous vein on Doppler and the Perthes test does not result in its collapse

Measurement of the degree of incompetence of the valves downstream of the valvulo-muscular pump.

Total, segmental, and partial reflux.

***I call Total Reflux (TR)**, a triangular diastolic reflux symmetrical in speed and duration of the systolic flow. It reflects a total incompetence of the downstream valves. This is the case of a popliteal reflux upstream of a total ilio femoro-popliteal incompetence.*

***I call Segmental Reflux (SR)**, a triangular diastolic reflux of equal duration but of small quantity than the systolic flow. It reflects a limited height of the incompetent column, thus competent valves above the incompetent segment. This is the case of a popliteal and superficial femoral reflux upstream of a common competent femoral valve. **This segmental reflux can also occur without deep incompetence,** in a segment of deep vein located between 2 competent valves, because it refluxes into the closed shunts of a superficial vein, as is the case of popliteal incompetence "cured" by disconnection of the incompetent Small Saphenous vein.*

I call partial Reflux (RP),** a diastolic reflux of a quantity that can be equal to the systolic flow, but over a much longer time with a lower velocity profile and in plateau. This reflux is due to a leaky or incompletely closed overlying valve. This is the case of a reflux from a popliteal incompetence which does not present a clear diastolic peak but rather a plateau. The time is all the longer and the velocity plateau lower than the incompetence of the valve(s) less important**. So, the reflux time is not necessarily proportional to the venous incompetence.

***I call Closed Shunt Reflux (CSR)** a triangular diastolic reflux **of greater quantity, diastolic peak and duration than the systolic flow.** They reflect an overload of diastolic flow by the flow of the competent vein to which it is connected by an escape point. In deep veins, this is the case of an incompetent superficial femoral vein whose diastolic reflux is increased by that of the competent deep femoral vein via the escape point represented by their junction.*

I have proposed a Dynamic Reflux Index (DRI) = (VmR². tR) / (VmS². tS)

-VmR = Mean diastolic reflux velocity, tR = Duration of diastolic reflux, VmS = Mean systolic velocity, tS= Duration of systole ref 1997: Measurement and interpretation of venous flows during stimulation maneuvers. Manual compressions and Parana maneuver. Dynamic Reflux Index (DRI) and Psatakis Index.
https://www.researchgate.net/publication/363335718_Mesure_et_interpretation_des_flux_veineux_lors_des_manoeuvres_de_stimulation_compression_manuelle_et_manoeuvre_de_Parana_Indidice_dynamique_de_reflux_IDR_et_Indice_de_Pastatkis_Journal_des_maladies_va

DRI** proportional to the hemodynamic value of reflux, **gives a "true" value of the reflux** demonstrated by less value in case of partial reflux compared to total reflux, **despite its longer duration.

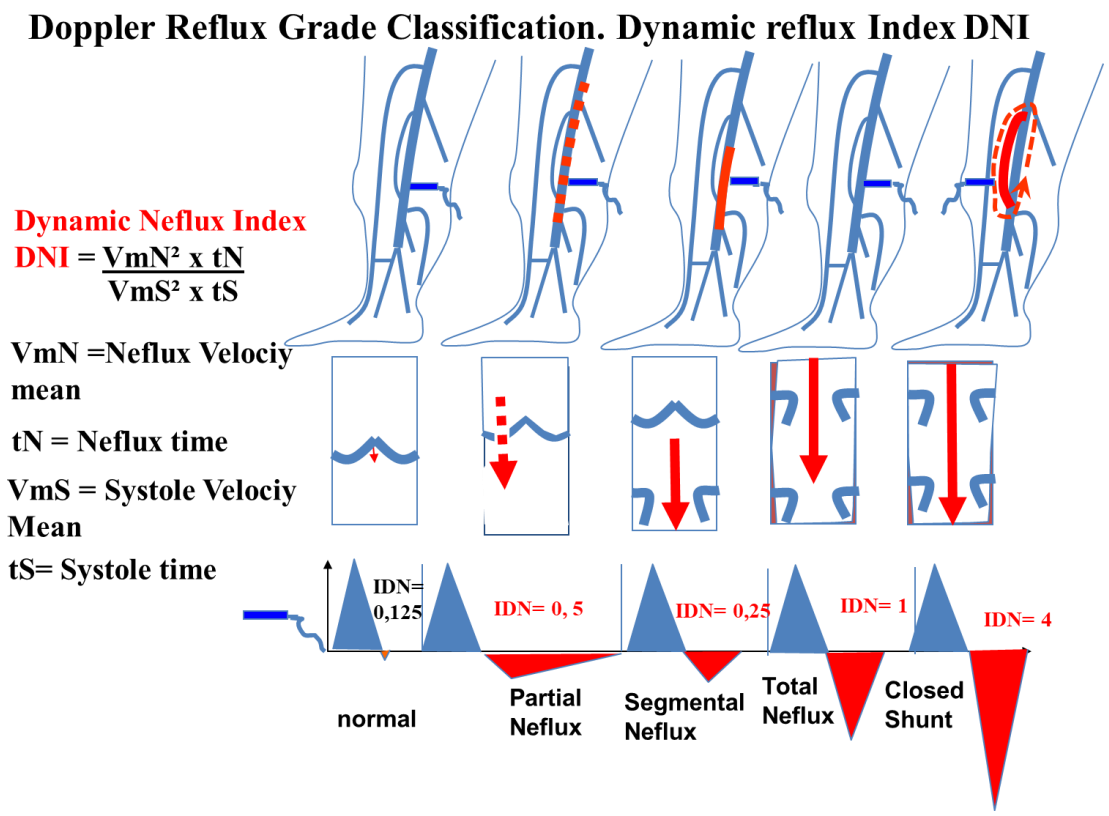
-Physiologic normal Reflux = 0.125,

-Total Reflux (TR) = 1,

-Segmental Reflux (SR) = 0.25,

-Partial Reflux (PR) = 0.5,

-Closed Shunt Reflux (CSR) = 4



5121- *Incompetence of the deep femoral-popliteal veins and leg veins*

This incompetence has variable hemodynamic values *depending on whether they are total, segmental, or partial.*

Their degree and topography *must be recognized because they guide the therapeutic strategy.*

The presence of deep closed *shunts in the veins of the thigh or leg, allows their simple disconnection as proposed by the* ***deep CHIVA CHIVP****.*

Most often, it consists of disconnection of *a closed shunt of the superficial femoral connected to a homologous collateral or a deep femoral competent and sometimes of an incompetent posterior Tibial Vein connected to a homologous collateral or a competent peroneal (fibular) vein.* ***Video:*** ***https://www.youtube.com/watch?v=EFkbRiRV2nM***

5122-*Incompetence of superficial veins or collaterals*

When the pump inlet and outlet valves are competent, *diastole causes* ***reflux into the incompetent collateral vein****, which* ***negates the effect of diastolic pump fractionation*** *DFGHSP.*

In addition, diastolic aspiration *causes all or part of the blood ejected downstream during the preceding systole to flow back upstream of the pump.* ***This creates a closed-circuit effect*** *that is reproduced with each muscular movement, especially in the calf during walking.*

***I have called this closed circuit, CS closed shunt**. It produces a double effect.*

1-On the one hand, it prevents the dynamic fractionation of the gravitational hydrostatic pressure DFGHSP** in systole as in diastole**.

***2-On the other hand, it overloads** in pressure and flow not only the pump, but also and **especially the incompetent collateral which receives**, during diastole, **all or part of the deep blood previously ejected by systole**.*

The kinetic energy of this overload**, which is proportional to the volume of the mass m and the velocities v, ½mv², **increases varicose dilatation and tissue damage

*This explains that **walking aggravates venous insufficiency** by valvular incompetence, especially with closed shunts.*

Varicosities are favoured and sometimes provoked by a refluxing vein. They fade when the feeder vein is disconnected. VIDEO https://www.youtube.com/watch?v=JScby8a0zZY&t=8s

They must be distinguished from those related to an obstacle to drainage, as we shall see later.

5123-Incompetence of superficial and deep veins

During diastole, the associated deep and superficial refluxes rush into the valvulo-muscular pump of the calf.

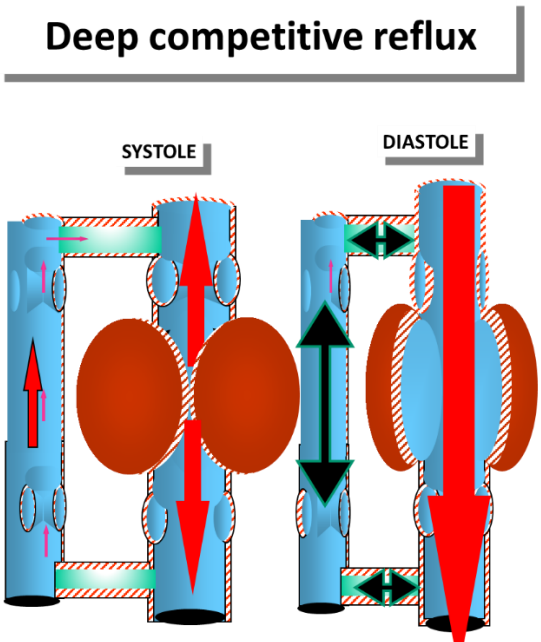
***It is the first to arrive, and therefore the most powerful, that wins and can fill all the space for itself**.*

*This is the case when an obviously incompetent great saphenous vein (very dilated and varicose) does not reflux during diastole stimulated by the Paraná test or simulated by the relaxed compression of the calf. This is what I called incompetent saphenous vein without reflux due to a **competitive deep reflux**. **No reflux of the great saphenous vein on Doppler and the Perthes test does not result in its collapse, when deep reflux wins the race! The return of reflux** of the great saphenous vein is a **test of the effectiveness of corrections of deep valve incompetence.***

***Indeed, the importance of the diastolic reflux of the superficial veins** is proportional not only to the superficial incompetence, but also to the good valvular continuity of the Valvulo-muscular ODS pump which aspires it.*

The greater the reflux from a closed or open deviated shunt, the more rapid and effective the expected result of a CHIVA disconnection. Which means, that:

Whatever the deep valve incompetence or obstruction, the Paranà diastolic reflux attests for a "good re-entry" in a deep pump which makes CHIVA disconnection efficient.



Competitive deep reflux. In case of deep and superficial valvular incontinence, deep reflux dominates and prevents, when it is major, the reflux of the greater saphenous vein, even though it is varicose.
No reflux of the great saphenous vein on Doppler and the Perthes test does not result in its collapse

5124-Pelvic veins Incompetence

Varicocele and especially hypogastric tributary varicose incompetence occur mainly in women, during pregnancy, and then regress in part, to evolve again with each pregnancy.

*Practically constant**, they are most often asymptomatic.***

*It can thus be said that **it is "normal" to find asymptomatic pelvic varicose veins** in mono or multiparous women.*

***They are sometimes responsible for the "clinical syndrome of pelvic congestion". T**hough it was too often ignored, it i**s nowadays too often confused with other causes of pelvic pain**.*

The conditions of their occurrence are hemodynamic and hormonal.

Hemodynamic for 3 reasons.

1-Hyper flow/pressure generated by the placenta overloads the pelvic veins for 9 months.

2--Pregnant uterus compresses to varying degrees the inferior vena cava and especially the left iliac vein already more or less pinched between the right iliac artery and the spine, sometimes already stenotic by synechiae.

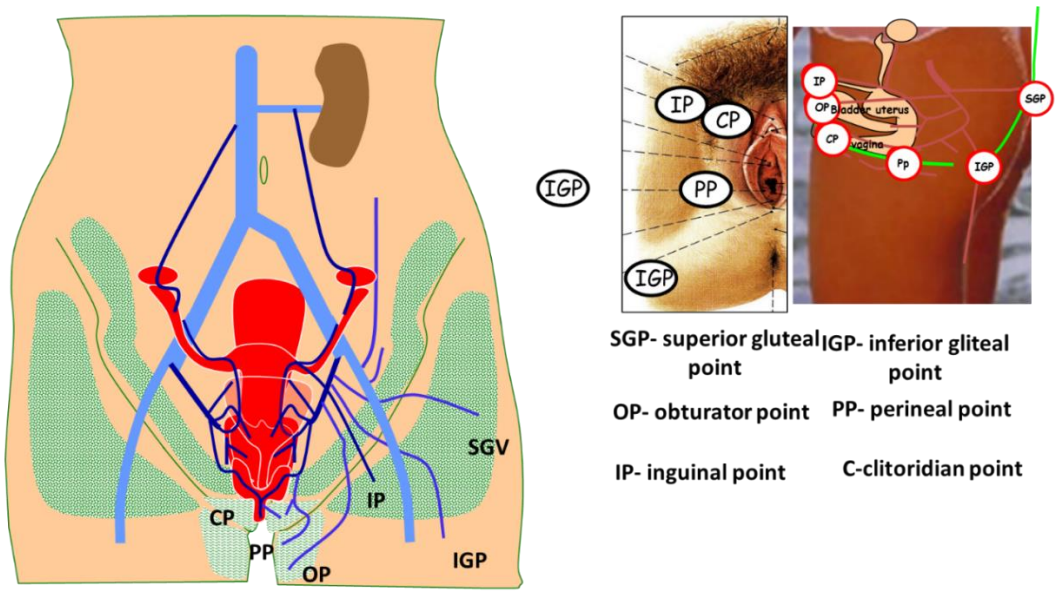
3--Hormonal oestroprogestative condition reduces the parietal tonus.

Pelvic venous pressure sometimes forces the valves of the visceral tributaries of the hypogastric vein, which transmit their reflux to the superficial pelvic veins via the pelvic

escape points that <u>I have described using echodoppler: perineal point (point P) and Clitoral point (point C) via the internal pudendal vein, Inguinal point (point I) via the vein of the uterine round ligament</u>. Ref: 1. Franceschi C, Bahnini A. Points de fuite pelviens viscéraux et varices des membres inférieurs. Phlébologie 2004;57:37-42.2. Franceschi C, Bahnini A. Treatment of lower extremity venous insufficiency due to pelvic escape points in women. Ann Vasc Surg 2005;19:284-8. 3. Franceschi C. Anatomie fonctionnelle et diagnostic des points de fuite bulboclitoridiens chez la femme (point C). J Mal Vasc 2008;33:42

In men, the equivalent of C point can be found*. The internal pudendal vein refluxes into the dorsal vein of the penis and then into the tributaries of the Great Saphenous Vein via its anastomoses.*

The inferior rectal vein *under pressure from the internal pudendal* ***vein dilates the hemorrhoidal veins, which distort and narrow the anal canal. In pregnant women, these "haemorrhoids" interfere with defecation, which mechanically and chemically attacks this canal and leaves faecal residues. These "haemorrhoids" can sometimes persist and evolve after pregnancy.***



Schematic venous anatomy of the pelvis of women, the pelvic and parietal veins receive their extra-pelvic afferents through connection points of the pelvic and abdominal walls

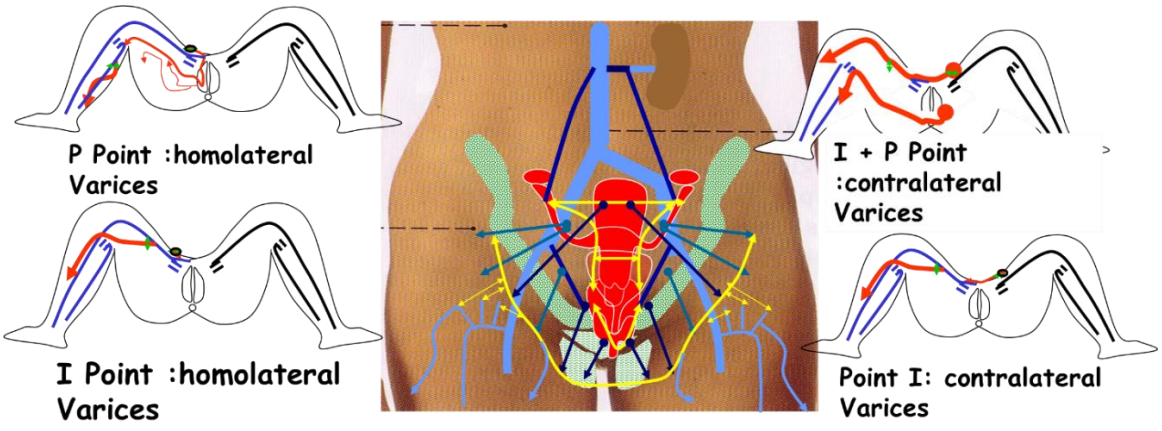
Leak points = penetration of superficial veins to the pelvis

Pelvic venous plexuses.

The pelvic, hemorrhoidal, uterine, vaginal, vesical and periurethral <u>plexuses do not have valves</u>.

They <u>anastomose the visceral tributaries</u>, which can <u>exchange their flow</u> and reflux vertically from top to bottom and horizontally from one side to the other.

<u>Functional systematization</u> simplifies the anatomical complexity by <u>considering for diagnosis and treatment only the escape and end points of reflux.</u>



These 12 tributary branches (6 on each side) can flow back into each other on the same side or on the other side via the plexus and/ or open the lower limbs and the perineum via the pelvic leakage points which can also communicate with each other on the same side and on the other side via the superficial anastomoses

Creating as many leakage points towards the perineum and the lower limbs and can communicate between them (rich and anarchic anastomoses) which can in their turn reflux into: The saphenous or extra-saphenous networks

51241—Varicocele: Varicose dilation of the gonadal veins

511411-Female varicocele may be mono or bilateral, but it Is usually located on the left.

It may also transmit its reflux to homo and/or contralateral visceral veins via anastomoses and plexuses.

Reflux of the left ovarian vein, overloaded by the flow of the left renal vein.

1-It is usually an open deviated shunt ODS.

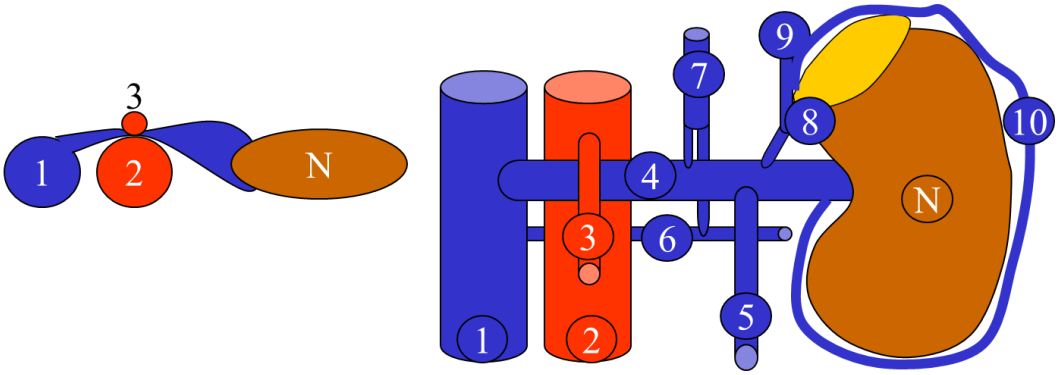
2- It is an open vicarious shunt OVS in the true Nutcracker Syndrome.

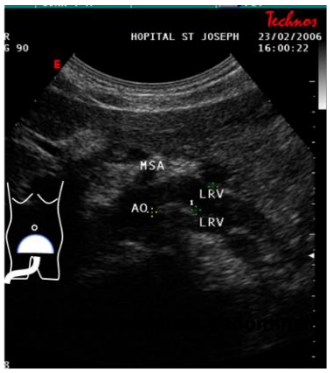
3-It is favoured by posture (sitting, standing) and thoracoabdominal pump systole, especially during defecation efforts and the Valsalva test.

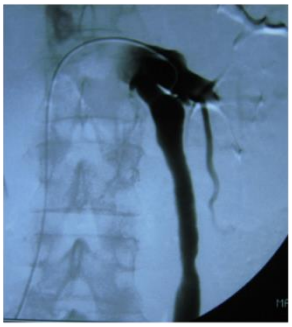
<u>When it is only due to an ODS</u>**, i.e., when it does not compensate for an obstacle to renal vein flow**, this reflux disappears in the Trendelenburg position** (head lower than the pelvis) as shown by the echodoppler**.** In this case, the renal flow evacuates without resistance, either directly into the inferior vena cava, or via compensatory renal-azygo-lumbar anastomoses which are effective in the case of nutcracker syndrome (NTS**). <u>Thus, reflux persists in this</u>

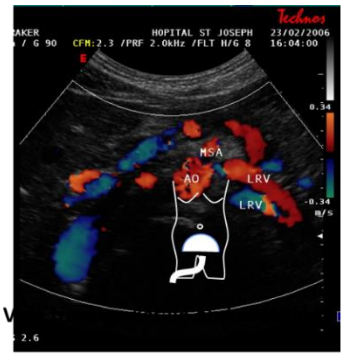
position when the shunt is not a simple ODS that can be safely disconnected, but a mixed shunt both open deviated shunt and open vicarious OVS compensating for a hemodynamically significant aorto-mesenteric stenosing clamp (Nutcracker syndrome NTS). Because of the risk of the severe risk of kidney drainage defect, this OVS can only be disconnected by embolization or ligation of the ovarian vein if the ODS by treatment of aorto-mesenteric stenosis is previously treated.

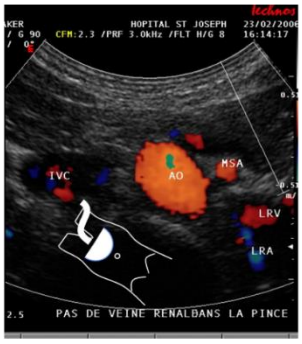
Compression of the left renal vein (4) against the Aorta (2) by the superior mesenteric artery (3) can generate a dangerous hemodynamic stenosis for the left kidney (N) due to a drainage deficit and a vicarious overload of the hemi-azygos (7), lumbar (6) adrenal (8) and phrenic (9) veins, the perirenal venous circle (10) (hemorrhagic risk) and the genital vein (5) whose reflux and pressure generate a left varicocele (spermatic in men and ovarian in women) sometimes responsible for a pelvic congestion syndrome.









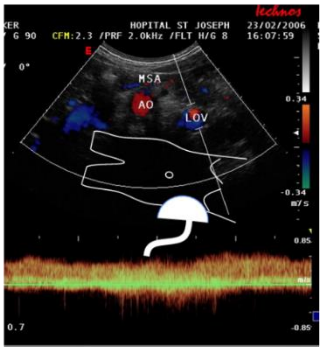


Trans-abdominal scan in Trendelenburg position.

Aorto-Mesenteric clip.
Venous flow = 0

No reno-azygo-lombar compensation

PENMANENT left ovaric vein reflux in Trendelenburg position (head lower than the feet).



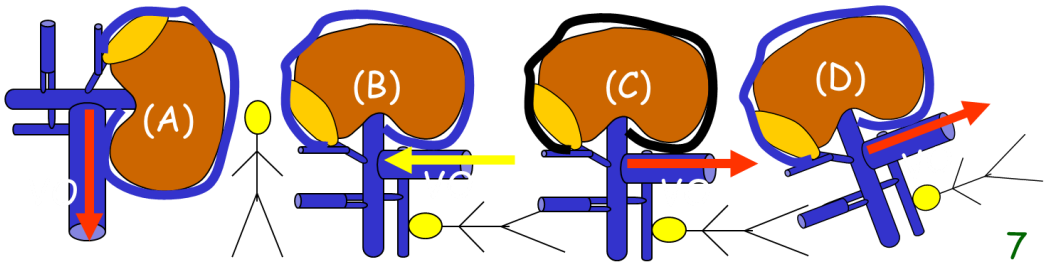
Trans-abdominal scan in Trendelenburg position.

Nutcracker Syndrome: Total Aorto-Mesenteric clip
Single bypass through the left ovarian vein

How to assess the risk? Measure the pressure in the renal vein, spontaneously and during the occlusion of the refluxing ovarian vein. By echodoppler: the reflux in the ovarian vein (VO)

-perceived in standing, in the sitting and half-sitting position and modulated by respiration, it can be due to the force of gravity alone without needing residual renal venous pressure (A). It is normalized in decubitus (B)

-If it is perceived as permanent and in decubitus, it can no longer be related to the force of gravity but to a saving vicarious effect (C). This test is made more sensitive by tilting the patient into Trendelenburg where the reflux is seen to persist (D). This pressure can be approximated by measuring the inclination necessary to stop this reflux.

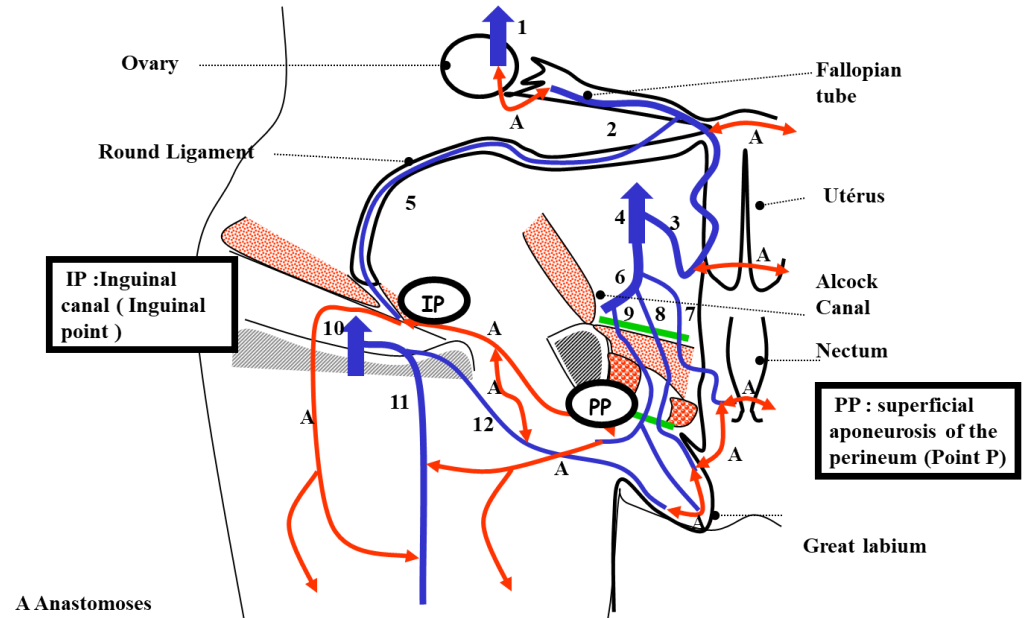


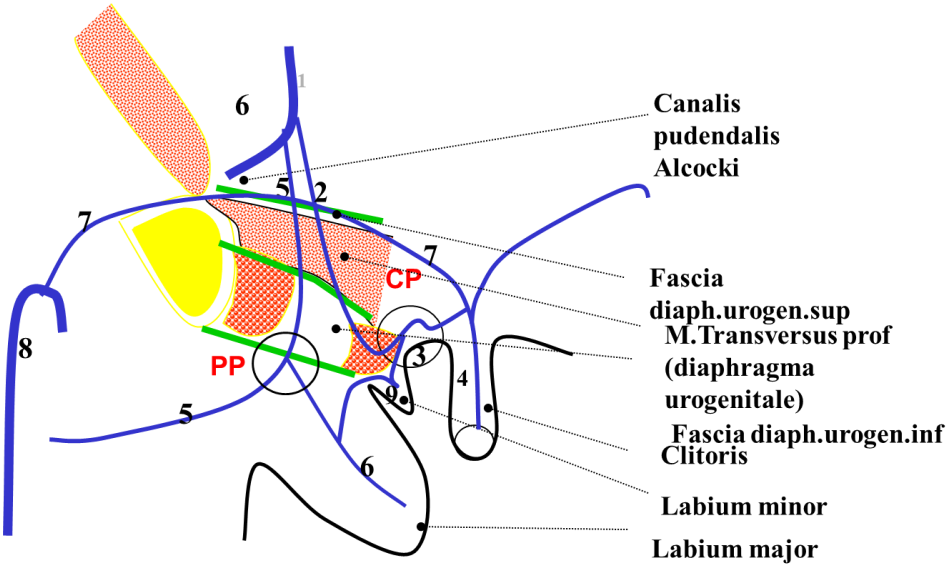
7

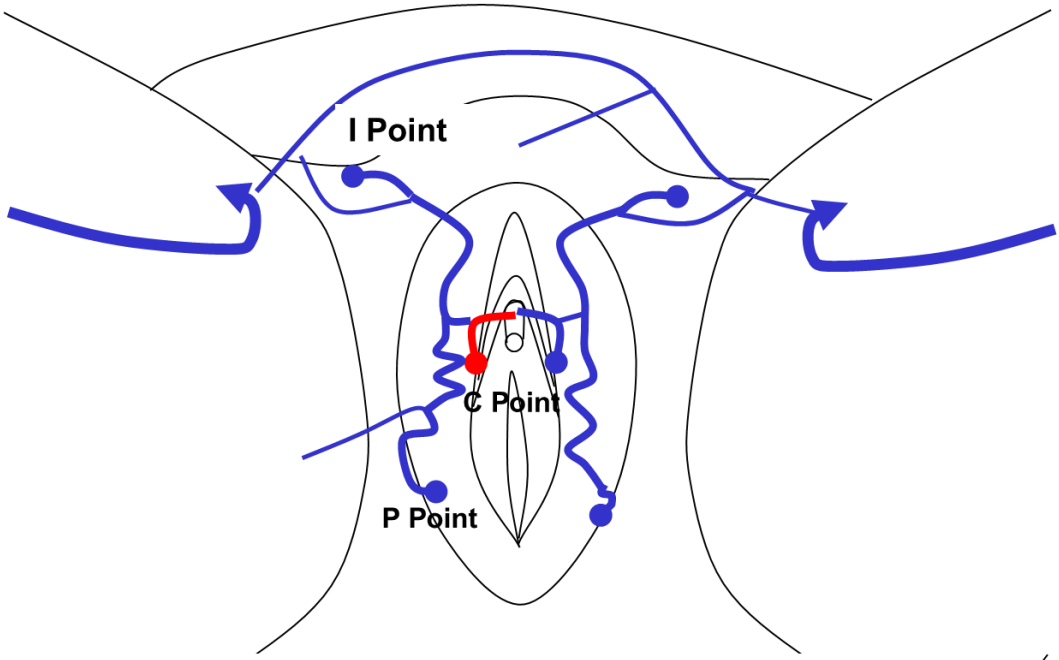
512412-Male varicocele is rarely associated with Nutcracker

Inconsistently responsible for male infertility, NTS syndrome manifests as intra-scrotal varicose veins but may sometimes communicate its reflux to the veins of the lower extremities via the tributaries of the great saphenous vein.

51242-Incompetence of the visceral tributaries of the hypogastric vein







Plexus ven. Commnicans (Clit. Et Bulbus vest.): During Valsalva maneuvre, reflux from Internal Pudendal vein into Anterior Saphena varicose vein through retrograde flow in bulbar vein, Plexus Ven. Communicans (Clit. Et Bulbus vest.), Dorsal Clitoris Vein and External Pudendal

*51242**1**- Internal Pudendal Vein:*

5124211-In women, the Internal Pudendal Vein refluxes

1-into the Perineal and Labial veins via the <u>Perineal point (P point)</u> at the union of the ¾ anterior and ¼ posterior of the vulvo-perineal fold

And/or into the dorsal vein of the clitoris via the bulbar vein <u>(Clitoral point C)</u> at the base of the clitoris

and then into the <u>veins of the perineum and/or the lower extremities homo and/or contralateral.</u>

512412-In men, the internal pudendal vein refluxes

into the <u>dorsal vein of the penis (point C?)</u> and then into the tributaries of the homolateral great saphenous vein via its anastomoses.

512422 -The vein of the round ligament of the uterus

1-communicates with the ovary veins via the tubal anastomoses of the vein

2- and refluxes through <u>Inguinal escape point (I point I)</u> into the veins of the Venus mount

3-and then into the <u>tributaries of the Great Saphenous vein</u>.

512423-Varices of the broad ligament show no reflux nor supply individualizable (Masters and Johnson)

512424-- Hemorrhoidal vein and "haemorrhoids"

The superior rectal veins drain into the inferior mesenteric vein. The middle and inferior rectal veins drain into the hypogastric vein via the internal pudendal vein. ***The inferior rectal veins drain the rectum via their internal hemorrhoidal tributaries and the anal canal via the internal and external hemorrhoidal veins.***

These veins communicate with each other via the submucosal hemorrhoidal plexus which constitutes a porto-caval anastomosis. The internal plexus is located in the upper part of the anal canal and the external plexus is located at the anus.

Dilated sometimes by portal hypertension, but most often independently of this pathology, they produce the disease called "haemorrhoids", internal in the anal canal and external in the anus.

Several physiopathological theories to explain sphincter** hypertonicity, procodence, rectal bleeding, strangulation, venous dilatation **have been proposed, of which 3 factors are most often cited.

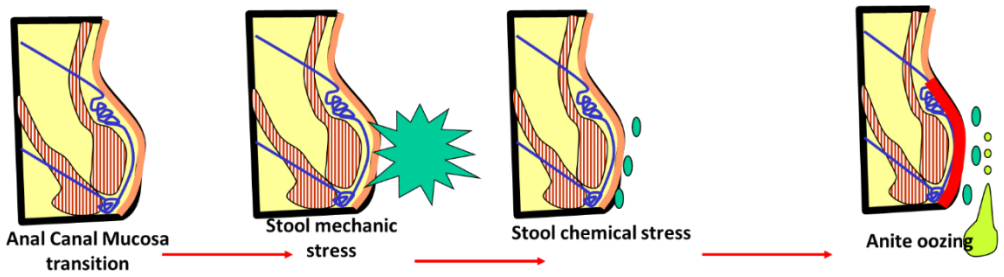
***1**-Vascular: decrease in venous return due to abdominal thrust during defecation*

***2**-Modification of pelvic and digestive vasomotricity: erythema, bleeding.*

***3**- Mechanical: submucosal laxity and supporting tissues*

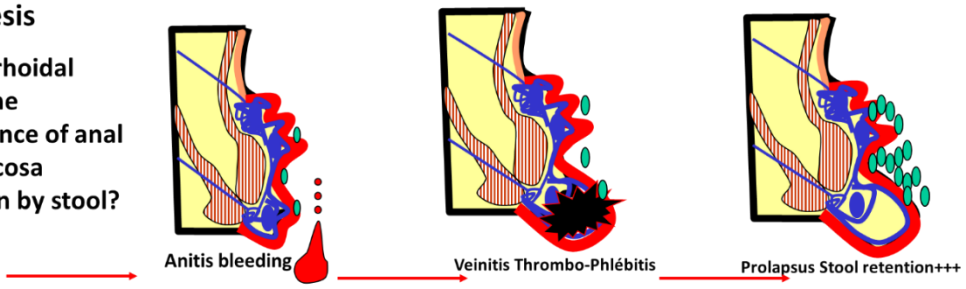
The fourth factor that I have proposed is consistent with the fact that it combines the first three as complications of the fourth. Ref: 1.C.Franceschi. Hémorroïdes : maladie des veines ou d'un quatrième facteur. Essai d'analyse physiopathologique. Conséquences thérapeutiques. Actualités Médicales Internationales. Angiologie (8), n° 145, décembre 1991***. 2.***B.Vergeau,R.Clément,M.Massoneau,C.Franceschi. Evaluation de l'efficacité et de la tolérance d'un nouveau procédé de traitement des hémorroïdes symptomatiques : Intrajet. Med.Chir.Dig. 1995 -24- 109-111. VIDEO https://youtu.be/1FoYynLlb98***.***

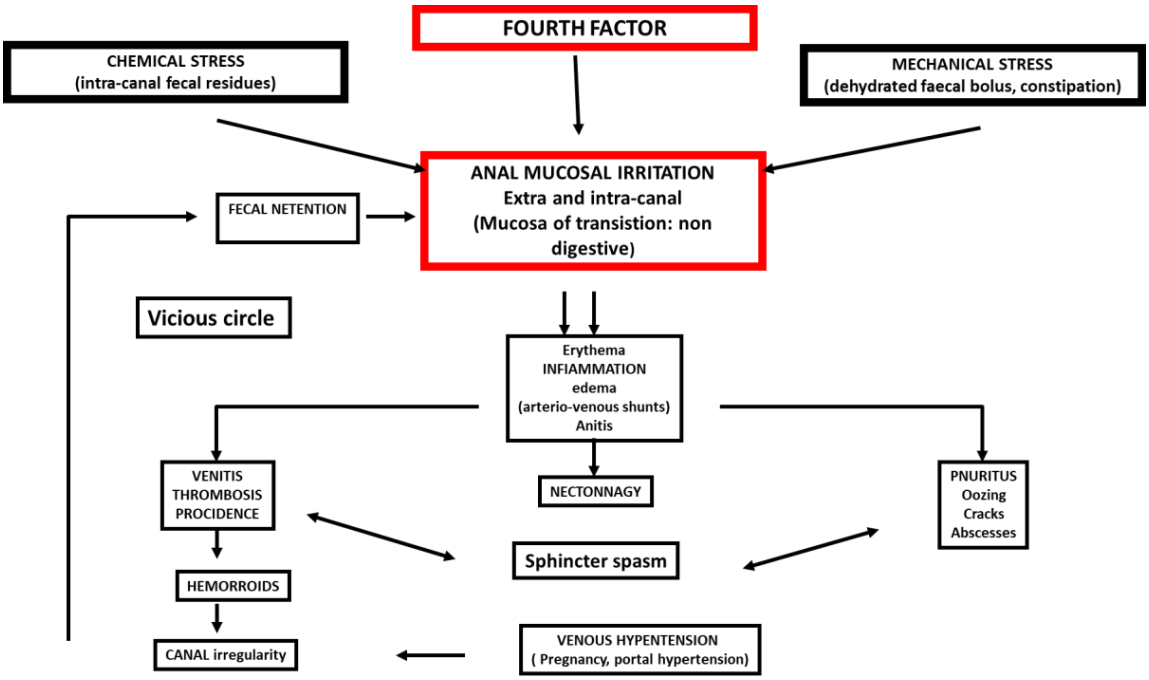
***It is the mucosa of the anal canal intolerance to stool and to traumatised by defecation. It is intolerant because** it is a transitional mucosa in the same way as the mucosa of the oropharynx. The complications are local irritation, inflammation, transmitted to the hemorrhoidal veins which thrombose and dilate, bleeding by mucosal inflammation etc. The **treatment consists of facilitating non-traumatic defecation and cleaning the residues after defecation.** A special water jet allows water to penetrate the lower rectum, without contact with the body because the device is 5 to 7 cm away. Before defecation, it allows without cannulation a micro-lavage which facilitates a non-traumatic evacuation and without excessive push. After defecation, it allows a rinsing of the macro and micro residues of faecal matter. An independent RCT study has demonstrated its effectiveness. Eliminates pain, pruritus, bleeding and stops the evolution of the disease.* VIDEO https://youtu.be/1FoYynLlb98

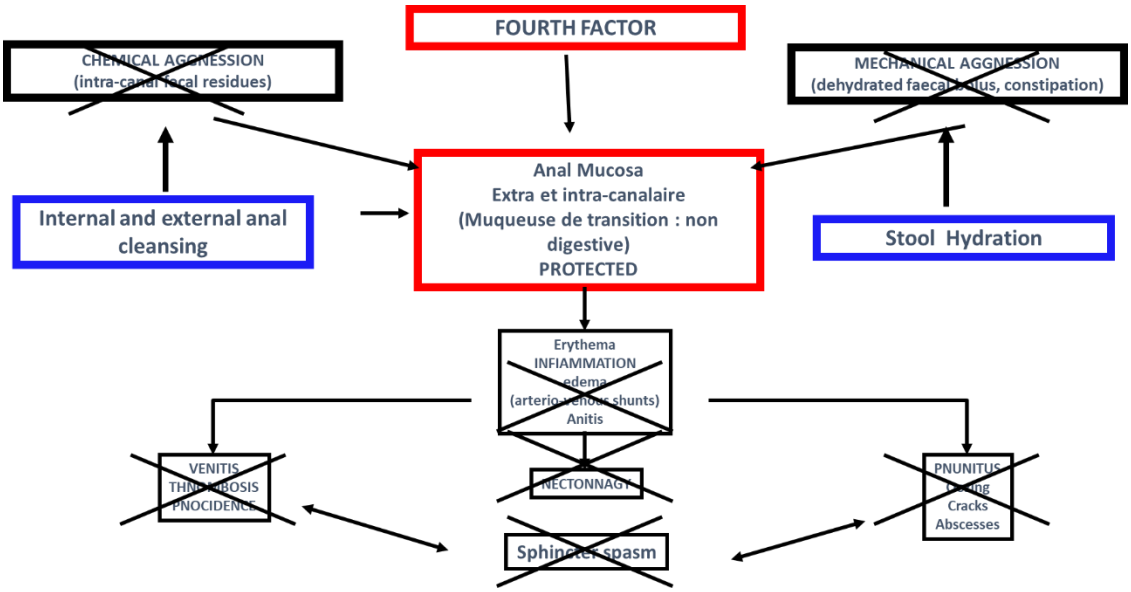


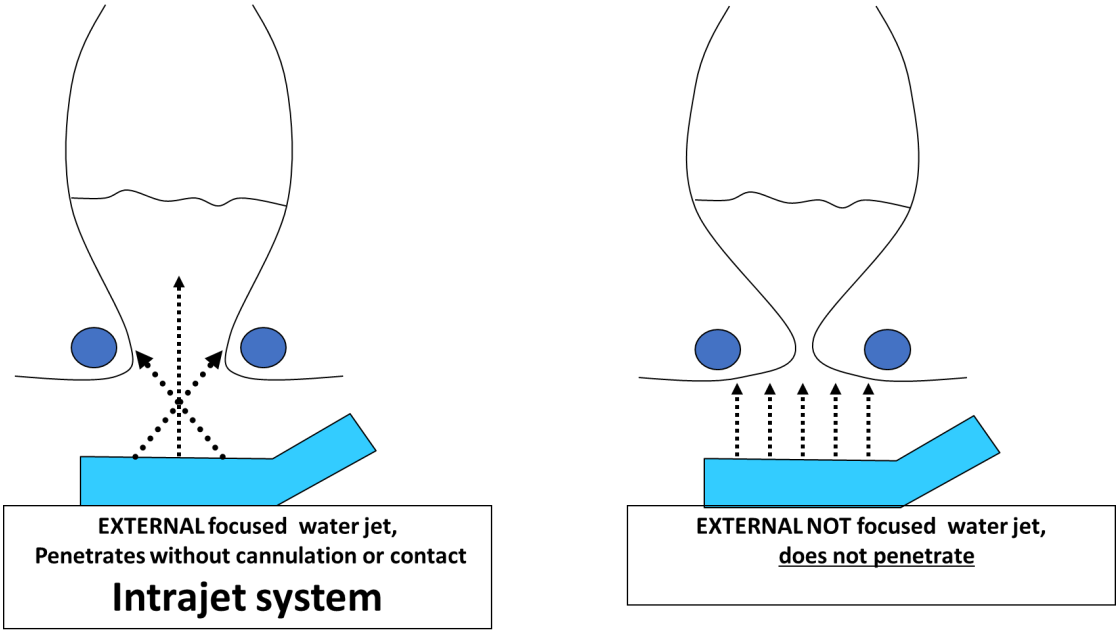
Hypothesis

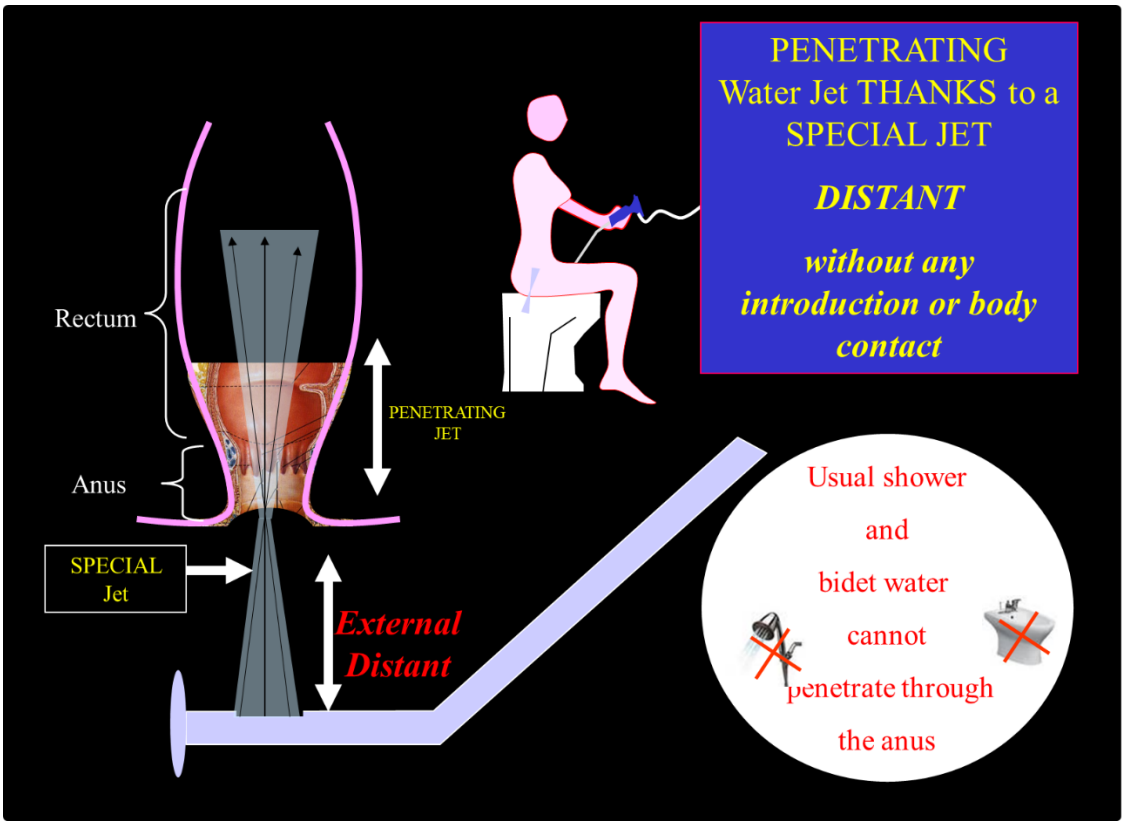
Is Hemorrhoidal disease the consequence of anal canal mucosa aggression by stool?






FOURTH FACTOR
CHEMICAL AGGNESSION
(intra-canal fecal residues)
MECHANICAL AGGNESSION
(dehydrated faecal residus, constipation)
Internal and external anal cleansing
Anal Mucosa
Extra et intra-canalaire
(Muqueuse de transition : non digestive)
PROTECTED
Stool Hydration
Erythema
INFIAMMATION
edema
(arterio-venous shunts)
Anitis
VENITIS
THROMBOSIS
PNOCIDENCE
NECTONNAGY
Sphincter spasm
PNUNITUS
Cracks
Abscesses





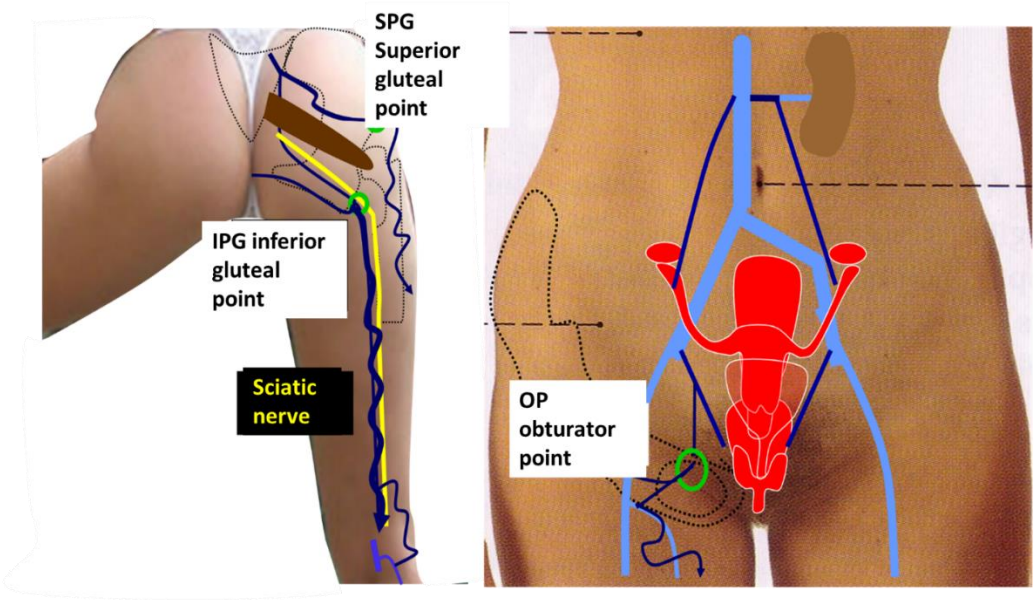
51243- Incompetence of the parietal tributaries of the hypogastric vein

Incompetence of the parietal tributaries of the hypogastric vein is rarer. It is <u>mostly found in venous malformations and in deep vein thrombosis</u>.

***512431--The Obturator vein, tributary of the hypogastric vein** communicates with the femoral and/or great saphenous vein, into which it may reflux via the <u>obturator point (O point)</u> located opposite the obturator foramen.*

***512432- The Superior Gluteal Vein** passes at the superior border of the Pyramidal muscle and refluxes via the <u>superior gluteal point (SPG)</u> into the superficial veins of the buttock.*

***512433-The inferior gluteal vein (also called the Ischiatic vein)** passes under the pyramidal muscle along with the sciatic nerve and refluxes via the <u>inferior gluteal point(IGP)</u> . The reflux may be <u>accompanied by the nerve and/or supply superficial reflux from the popliteal fossa.</u>*



Pelvic leak points of the hypogastric tributaries. Gluteal and obturaror veins.

52- *Venous Obstacles*

Superficial or deep, venous obstacles <u>reduce tissue drainage and increase residual pressure RP in proportion to their resistance and their compensation by collaterals</u> (open vicarious shunts OVS).

They can be <u>benign</u>, responsible for varicosities and recurrences secondary to destructive surgical or endovenous treatments.

They can be <u>severe</u> because of their functional impact when they involve the deep veins, or even very severe when they cause <u>ischemia</u> (phlegmatia cerulea).

521-Superficial venous obstructions

5211-Superficial dermo-hypodermic venous obstructions

Venous incompetence is the cause of varicosities and telangiectasias very frequent, especially in women, cause aesthetic discomfort, but without any functional severity.

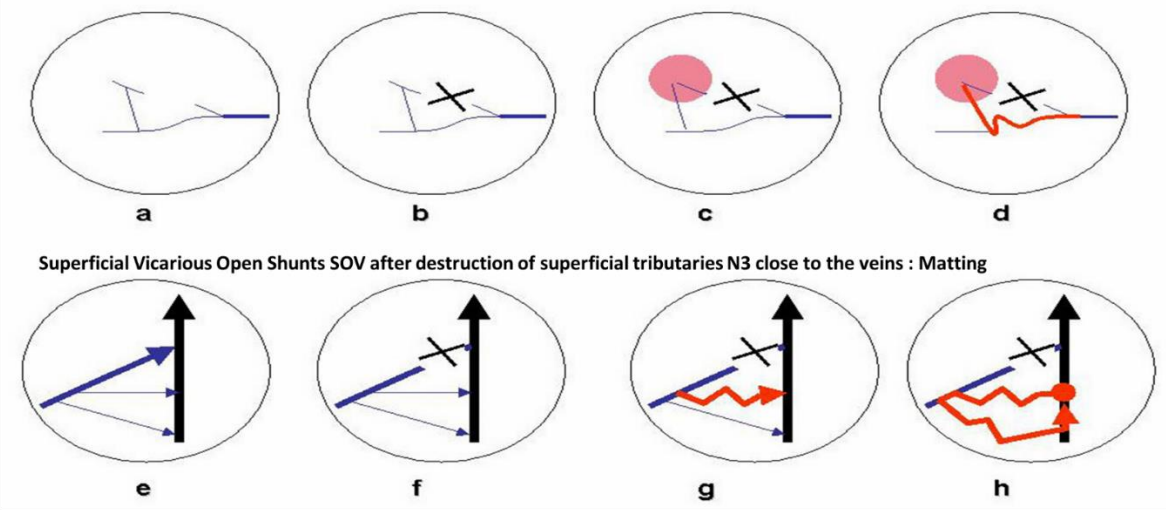
The obstacle seems to be due to dermal-hypodermal sclerosis that compresses the venules, especially in areas usually poor in drainage paths such as the lateral face of the thigh and the medial face of the knee, where there is a cellulite thickening or, on the contrary, a very thin skin and sub-skin.

The first case could be that of young women who are both affected of cellulitis andweakened by oestroprogestative impregnation.

The second case could be that of older women, whose skin degraded by aging is sometimes aggravated by excessive sun exposure. The microcirculation bypasses these obstacles and dilates collateral venules and capillaries.

These varicosities must be distinguished from those related to reflux, as the therapeutic strategy is different.

5212-Superficial venous obstacles due to venous obstruction is most often the consequence of destructive, non-hemodynamic treatment of varicose vein.



522- Deep venous obstructions

Systolic deep shunts are open vicarious shunts OVS.

Hemodynamic evaluation of the impact on TMP requires measurement of residual upstream venous pressures in supine position, which eliminates the proportion of gravitational hydrostatic pressure GHSP and any associated valvular incompetence.

Deep venous obstacles increase the upstream residual pressure RP *at rest and even more so during exercise (flow Q/arteriolo-capillary pressure + flow Q/Valvulo-muscular pump pressure). P=Q.Rt. The total resistance Rt is equal to the resistance of the obstacle to the flow N1 in the absence of compensating collaterals (open vicarious shunt OVS). It decreases with the opening of the collaterals of resistance N2. This is equivalent to Ohm's law.* ***The total resistance Rt decreases with the opening of the collaterals*** *which are resistances in parallel N2 because 1/Rt=1/N1+1/N2. In the absence of open vicarious shunts, the resistance N2 is infinite, 1/N2=0 so Rt=N1, P=Q.N1. The opening of open vicarious shunts reduces N2, so Rt. 1/Rt=1/N1+1/N2 so Rt=N1.N2/N1+N2 and P=Q.N1. N2/N1+N2.*

It seems logical to advise physical exercise (walking) with lower limb compression to develop compensatory collaterals (decrease N2).

Thus, a revascularization procedure can be avoided if this compensation leads to a sufficient reduction in pressure.

Staged resistances *are resistances in series which, contrary to parallel resistances, are added Rt = N1+N2. Thus, the posterior tibial vein pressure P = Q.(N1+N2).*

In this case, recognizing the part of N2, requires an additional pressure measurement between N1 and N2.

The venous flow can become pulsatile cardiopetal, *synchronous with the cardiac rhythm when the flow resistances are such that the decrease in velocities reduces the pressure drop (Poiseuille) of the microcirculatory resistances and reduces their damping effect****. This is particularly true since there may be a reflex opening of the micro-shunts.***

5221-Pelvic venous obstruction

52211-Nutcracker syndrome or aorto-mesenteric clamp.

The left renal vein may be compressed by the superior mesenteric artery against the aorta.

This stenosis is anatomically very frequent, *but it is not necessarily hemodynamically significant.*

When it is hemodynamically significant, it can be compensated by an open vicarious shunt OVS formed by the renal-azygo-lumbar system and/or by a reflux in the left gonadal vein (ovarian in women and spermatic in men) called varicocele. ***The horizontal supine posture can cause this stenosis. For this reason, its pathological character can only be affirmed if it persists in a half-sitting position.***

When it is poorly compensated, it can lead to renal venous hypertension with impairment of the function, which causes proteinuria and haematuria. It requires treatments that are

***not yet fully evaluated*, such as stenting or transposition of the left renal vein, or renal transposition or gonadal vein-inferior vena cava anastomosis.**

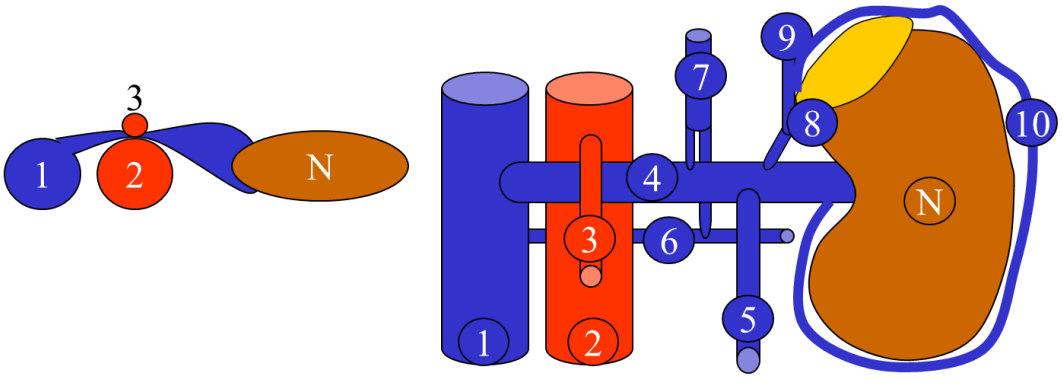
The association of left renal vein stenosis and left varicocele *does not necessarily mean that the varicocele is compensatory.*

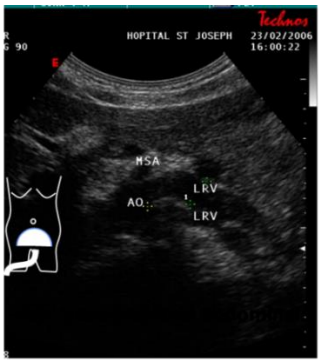
When well compensated by a varicocele**, without manifestation of pelvic congestion, the varicocele is considered an **open vicarious OVS shunt to be preserved.

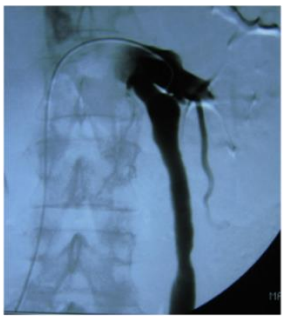
***When it is well compensated by a varicocele, but is complicated by a pelvic congestion syndrome,** embolization/ligation of the varicocele of this open vicarious OVS shunt can only be considered in association with the treatment of the left renal vein stenosis. **This varicocele is compensatory only if the reflux of the varicocele persists in anti-Trendelenburg decubitus** (feet higher than the head) during echodoppler or phlebography.*

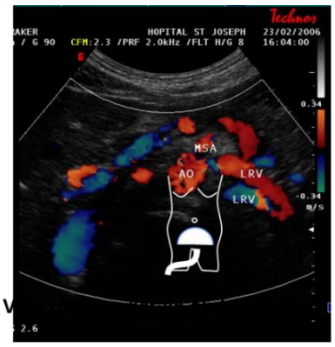
When it is well compensated by a varicocele, and is not complicated by a pelvic congestion syndrome, but is associated with varicose veins of the perineum and/or the lower extremities,** treatment should be limited to the treatment of the pelvic escape points... unless the latter reflux not only during dynamic tests, but also at rest, which would indicate a compensation pathway for the renal vein stenosis.* ***Ref: Delfrate R, Bricchi M, Franceschi C. Minimally invasive procedure for pelvic escape points in women. Veins and Lymphatics. 2019; 8:7789, 10-16.

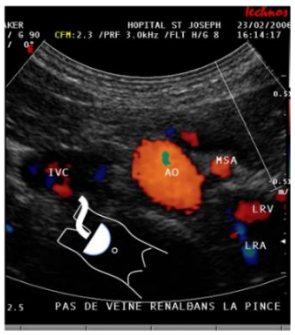
Compression of the left renal vein (4) against the Aorta (2) by the superior mesenteric artery (3) can generate a dangerous hemodynamic stenosis for the left kidney (N) due to a drainage deficit and a vicarious overload of the hemi-azygos (7), lumbar (6) adrenal (8) and phrenic (9) veins, the perirenal venous circle (10) (hemorrhagic risk) and the genital vein (5) whose reflux and pressure generate a left varicocele (spermatic in men and ovarian in women) sometimes responsible for a pelvic congestion syndrome.







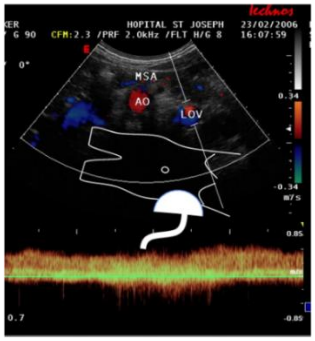




Trans-abdominal scan in Trendelenburg position.

Aorto-Mesenteric clip.
Venous flow = 0

No reno-azygo-lombar compensation
PENMANENT left ovaric vein reflux in Trendelenburg position (head lower than the feet).



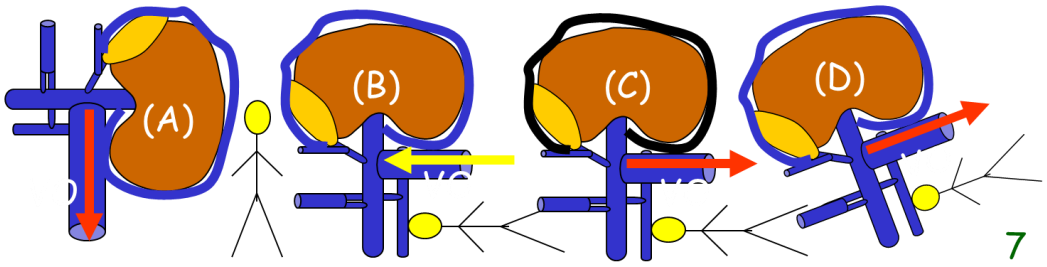
Trans-abdominal scan in Trendelenburg position.

Nutcracker Syndrome: Total Aorto-Mesenteric clamp Single bypass through the left ovarian vein

How to assess the risk? Measure the pressure in the renal vein, spontaneously and during the occlusion of the refluxing ovarian vein. By echodoppler: the reflux in the ovarian vein (VO)

-perceived in standing, in the sitting and half-sitting position and modulated by respiration, it can be due to the force of gravity alone without needing residual renal venous pressure (A). It is normalized in decubitus (B)

-If it is perceived as permanent and in decubitus, it can no longer be related to the force of gravity but to a saving vicarious effect (C). This test is made more sensitive by tilting the patient into Trendelenburg where the reflux is seen to persist (D). This pressure can be approximated by measuring the inclination necessary to stop this reflux.



52212- *May Thurner syndrome MTS*

May Thurner syndrome, otherwise known as Cockett syndrome, is the stenosis of the left iliac vein by the clamp formed by the right iliac artery and the lumbar spine, more or less associated with synechiae.

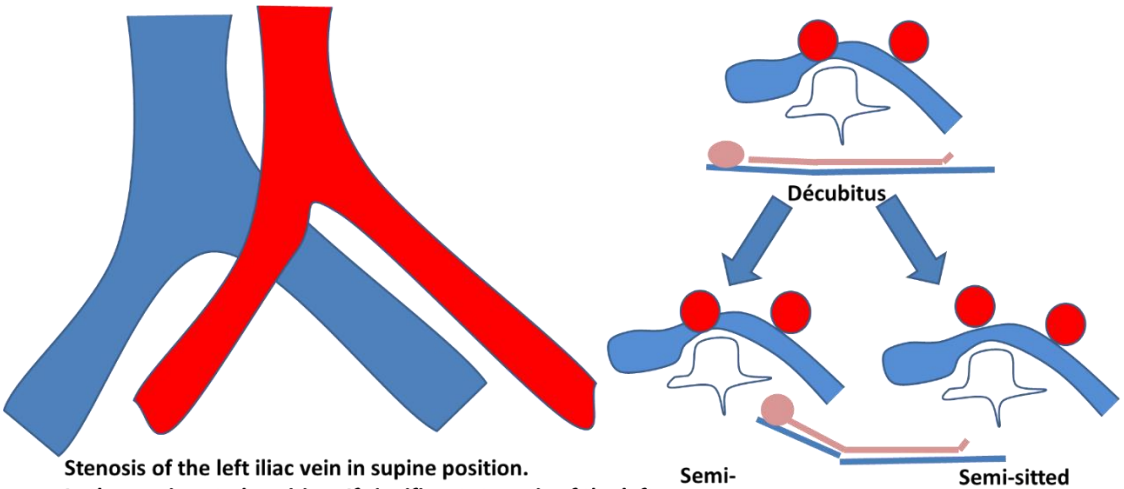
This stenosis is frequent but *most of the time it is not hemodynamically significant****.***

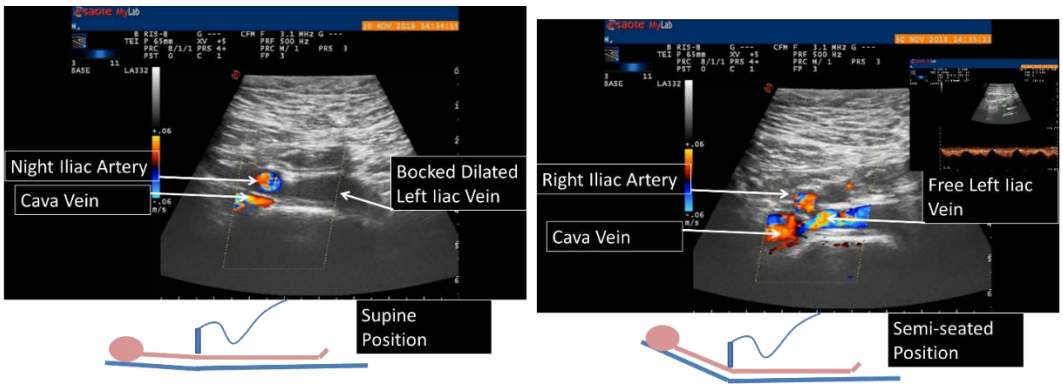
It has the particularity of varying with posture. I have shown with the echodoppler that it can be complete in horizontal supine position with cessation of flow and open up very clearly with a correct flow as soon as the patient is in a semi-seated position. Ref: Paolo Zamboni, Claude Franceschi, Roberto Del Frate.The overtreatment of illusory May Thurner syndrome Veins and Lymphatics 2019; volume 8:8020. VIDEO: Pseudo MTS : https://www.youtube.com/watch?v=h931XXo2hdk&t=23s

For this reason, phlebography cannot distinguish postural non-pathogenic illusory MTS from permanent true MTS, because it is performed only in horizontal supine position. This explains why a study has shown many phlebography stenosis in healthy subjects but also, the possible overtreatments. Ref: van Vuuren TM, Kurstjens RLM,Wittens CHA, et al. lllusory angiographic signs of significant lliac vein compression in healthy volunteers. Eur.J Vasc Endovasc Surg 2018;56:874- ***Moreover, the activation of the collaterals indicates its hemodynamic character but also its compensation by OVS effect. The effectiveness of this compensation must be evaluated by invasive or Doppler measurement of the upstream venous pressure in the decubitus and semi-sitting position. The normal pressure in the semi-seated position symmetrical with the right lower limb eliminates any pathogenic character of the MTS. The endoluminal ultrasound in the same positions cannot affirm the significant hemodynamic character of this clamp even if it shows synechiae, because we know that only the hemodynamic measurement can evaluate of a stenosis and its compensation.***

In short, any MTS that does not result in an excess of TMP should be considered benign.

However, the local conditions of these stenoses, although not hemodynamically significant, explain the risk of left iliac thrombophlebitis, especially during pregnancy.





May Thurner Syndrome at phlebography but
Pseudo May Thurner Syndrome on Echodoppler: only postural

5222- *Thrombosis or agenesis of the iliac and/or cava veins.*

Thrombosis and agenesis of the iliac and/or vena cava develop

superficial OVS via the epigastric and then superficial thoracic veins and

deep OVS via the iliolumbar, gonadal, and small and large azygos veins.

Accurate assessment of deep OVSs does not affect therapeutic strategies, *except when OVSs overload spinal canal veins with neurological consequences.*

Compensations through superficial abdominal veins should *avoid compressive abdominal garments and belts.*

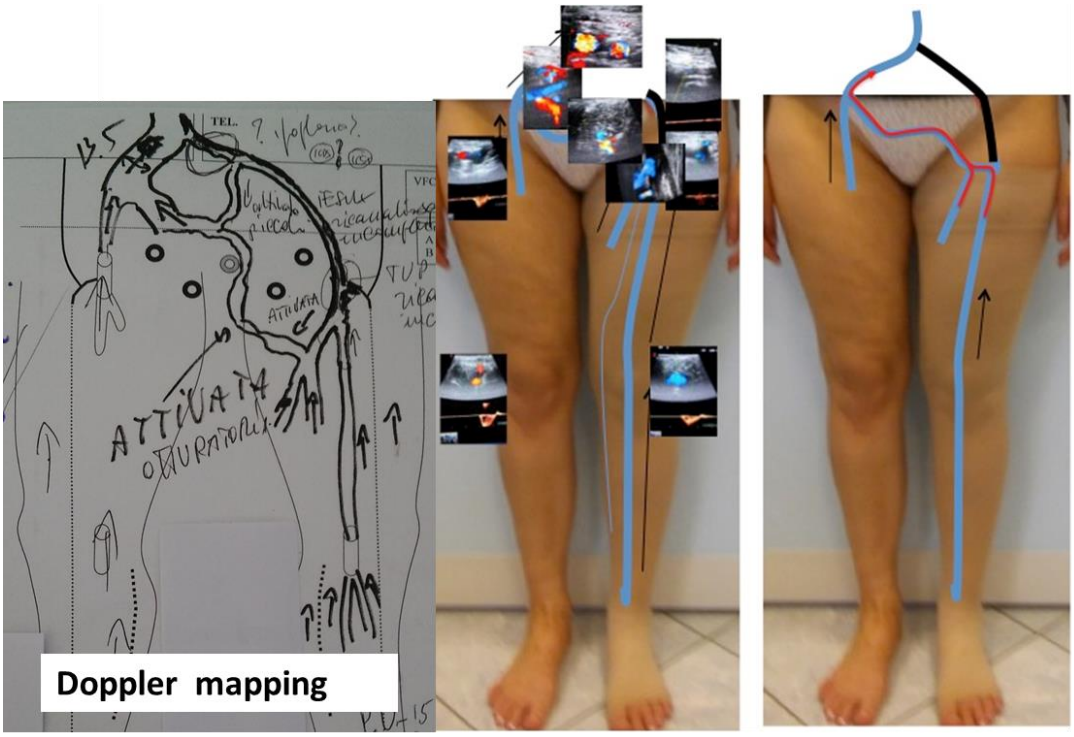
Revascularization procedures should be preceded by an assessment of the respective proportion of the obstruction and incompetence by measuring upstream pressures in decubitus and should be performed only if the pressures are significantly elevated.

5223-Deep venous obstructions of the lower extremities

Any deep vein of the lower extremities can be occluded and increase the upstream TMP to a greater or small extent, depending on its location and collaterals.

Thus, isolated thrombosis of *a duplicated superficial femoral vein, a tibial, fibular, soleus or gastrocnemius vein does not always lead to oedema of the leg and may be perfectly asymptomatic.* ***This explains the frequency of clinically silent phlebitis.***

Compensatory OVS, particularly via the great saphenous vein, may present a clinical aspect of essential varicose veins and lead to inappropriate treatments. This further demonstrates the mandatory necessity of hemodynamic evaluation of any varicose vein.



Compensated Iliac Vein Occlusion: clinical and hemodynamic:
Doppler Left Posterior Tibial Vein Pressure = 20 mmHg

5224- Deep and superficial associated shunts

In the same subject, open vicarious shunts OVS and closed shunts CS are distributed and associated in a variable way between the superficial N3 and N2 and deep N1 networks.

These shunts are found in malformations and post-thrombotic syndrome PTS where it is necessary to evaluate the respective parts of the incompetence and the obstacle in order to elaborate the best therapeutic strategy.

They most often constitute <u>mixed shunts MS</u> consisting of a compensatory pathway OVS of the superficial femoral vein or iliac vein obstruction and a <u>closed shunt CS</u> of the homo and/or contralateral great saphenous vein.

<u>Measurement of tibial venous pressure in the decubitus position allows selective evaluation of the hemodynamically significant part of the obstacle</u>, *because in this position venous incompetence does not alter the pressure (the height of the gravitational hydrostatic pressure column is negligible).*

523-Thoracoabdominal obstruction

The thoracoabdominal pump may <u>increase residual pressure due to diaphragm paresis and/or inferior vena cava compression</u>, particularly in obese subjects.

In these patients, we see ***clinical signs of excessive transmural pressure TMP*** *as oedema and varicosities of the ankles* ***whereas the venous system is normal****. It can also be assumed that this relative stasis facilitates the deterioration of the valves and the occurrence of varicose veins****.***

<u>In pregnant women</u>, compression of the inferior vena cava in horizontal decubitus position, in addition to thrombosis risk of the left iliac vein, may cause <u>acute deactivation of the cardiac pump</u> by the failure of the reservoir effect.

524-Cardiac resistance

Each diastole of the right ventricle sucks venous blood, reducing venous volume and residual pressure. ***Any increase in right atrial and right ventricular pressure increases residual pressure when the reservoir effect of the venous bed is exceeded.***

Any systolic <u>backflow from the tricuspid valve emits a retrograde pressure wave in the veins that becomes pulsatile</u>. *This wave is accompanied by pulsatile venous reflux synchronous with the pulse when the valves are incompetent****. This is particularly the case when the great saphenous veins are incompetent.***

This cardiofugal reflux should <u>not be confused with cardiopetal anterograde flow</u> due to downstream venous obstacles.

525- Reflux and inflammation

<u>Contrary to what has been suggested, it is not the retrograde direction of flow (reflux) that causes inflammation</u>, but the hemodynamic overload, regardless of the direction. Indeed, inflammatory phenotypes decrease markedly after reduction of the reflux load, without having changed its direction. This was demonstrated in the aftermath of the CHIVA treatment. It is therefore the excess flow/ venous pressure that produces cytokines. Ref.

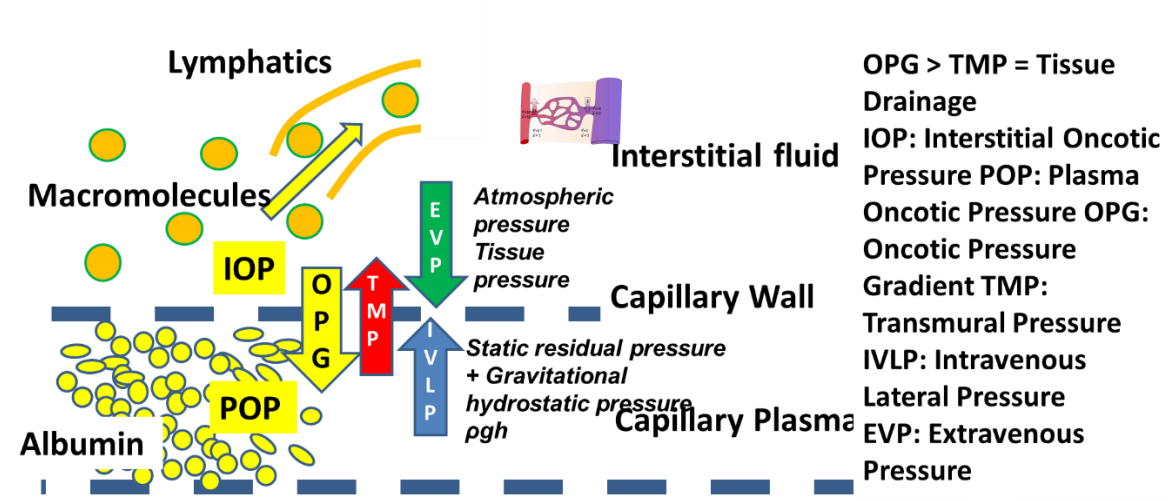
ZAMBONI P. et Al.:Oscillatory flow suppression improves inflammation in chronic venous disease. journal of surgical research _ september 2016 (205) 238-245

526- Veno-lymphatic insufficiency

The venous and lymphatic systems are 2 complementary drainage systems.

1-The venous system drains interstitial tissue *fluids and their contents, except for macromolecules.*

2-The lymphatic system drains these *macromolecules not accepted by the venous system, among its multiple functions, notably digestive, anti-infectious and immune.*



5261- Lymphatic drainage impaired by venous insufficiency

-<u>Insufficient venous drainage</u> leads to <u>tissue suffering which produces inflammatory reactions</u> to catabolites, necrotic waste products which are drained by the lymphatics and can overload them.

-Secondary <u>lymphatic insufficiency</u>, either due to <u>a poor lymphatic network or to overloading</u>, leaves undrained interstitial liquid, chronic inflammation, and its sclerotic evolution.

5262- Venous drainage impaired by lymphatic insufficiency

Lymphatic insufficiency impairs venous drainage by increasing interstitial oncotic pressure IOP which lowers the oncotic pressure gradient OPG. <u>The consequence is less force opposite to the transmural pressure, thus less draining flow in the capillaries</u>.

Indeed, according to Starling, *the drainage flow FD of the interstitial fluid drains into the venous portion of the venous capillary. Normally, the oncotic pressure gradient GO (plasma oncotic pressure POP- interstitial oncotic pressure IOP) is higher than the Transmural capillary pressure TMP (TMP= Lateral venous pressure IVLP-Venous extra-capillary pressure PEV).*

Drainage flow FD = POP-TMP = GO - TMP) = (POP - IOP) - TMP.

If IOP increases, GO decreases and FD decreases.

By design, I have introduced TMP rather than IVLP alone because compression treatments, restore drainage by reducing TMP although IVLP remains unchanged.

527-Varicogenesis

Excessive transmural pressure from the PTM dilates the veins. These dilated veins are called varicose veins, especially when they are irregular in shape.

The mismatch between the flow velocity ½ mv² and the calibre exceeds the Reynolds number. The laminar flow becomes turbulent. ***The turbulence carries shocks against the walls***. ***When the gauge reaches a value that reduces the velocity,*** *the flow becomes laminar again and is less aggressive to the wall. Therefore, the* ***varicose vein size stops growing and remains stable for years***. *These overloads in flow/pressure necessary for varicogenesis are* ***produced by veno-venous shunts, therefore only in subjects who walk***. ***The non-dynamic fractionation of the gravitational hydrostatic pressure (DFGHSP) at rest is sufficient to hamper the drainage but insufficient to cause varicose veins***. *In fact, varicose subjects who have become* ***paraplegic*** *and who remain seated all the time, thus with a high hydrostatic gravitational pressure, experience oedemas but* ***their varicose veins "disappear".***

Paradoxically, sport aggravates varicose veins *by overloading the shunts with flow and pressure.*

Varicose open vicarious shunts OVS *increased by walking* ***are welcome*** *because they reduce venous insufficiency by improving upstream drainage.. By contrast, varicose veins in closed CS and open ODS shunts worsen venous insufficiency*

528-Remodeling

Remodelling results from the adequacy of calibre to the reduction of Transmural pressure TMP.

A dilated vein gradually returns to normal calibre when it is no longer overloaded. This is true for the veins of the N1, N2 and N3 networks

The time required for remodelling depends on the time of restructuration of the musculo-conjunctive wall elements. For this reason, non-destructive hemodynamic treatments require a "healing" time. This is particularly the case after CHIVA treatment, where the regression of the calibre of varicose veins to normal requires time for remodelling. ***Ref:*** Mendoza E., Diameter reduction of the great saphenous vein and the common femoral vein after CHIVA Long–term results, Phlebologie, 2013, 42: pp. 65–69.

53-Venous ulcer

An ulcer is a cutaneous wound that <u>does not heal in the usual time frame</u>.

There are <u>many causes</u>, of which <u>excess venous transmural pressure</u> TMP is the most common. *It can be aggravated by arterial ischemia or lymphatic insufficiency.*

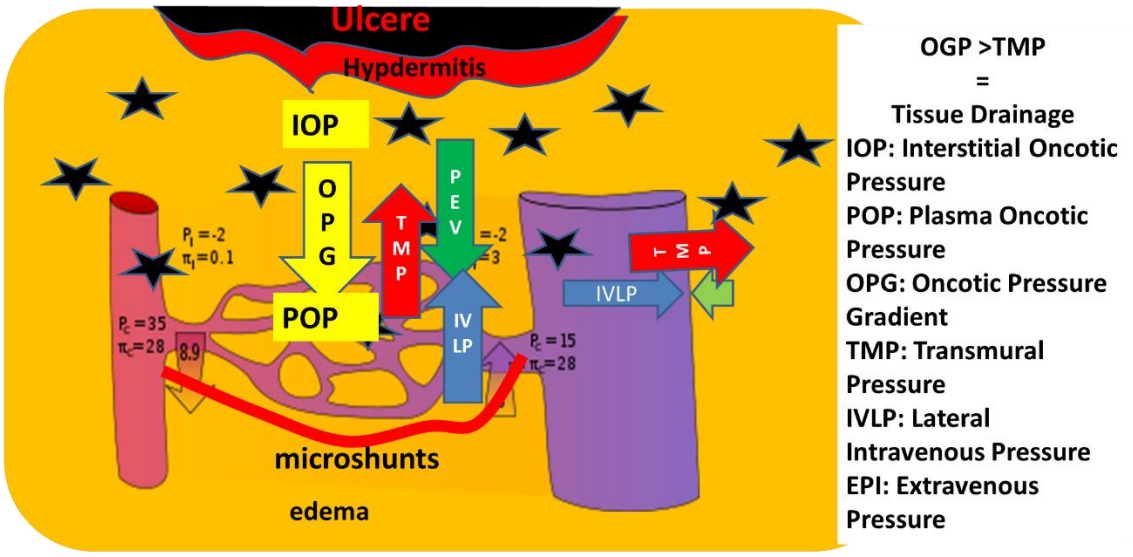
But most often, <u>it is the superinfection which aggravates it:</u> *inflammation, extension, and delay.*

The reduction of drainage *by <u>excessive capillary transmural pressure</u> is accompanied by a <u>slowing down or even abolition</u> of* ***the capillary flow "stolen" by the micro-shunts.***

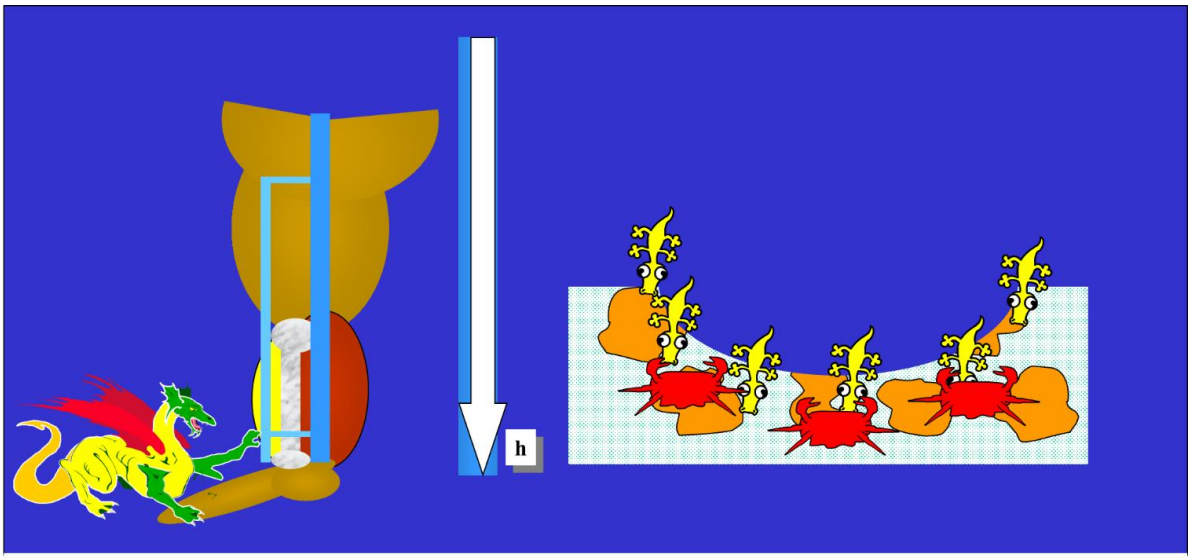
It accumulates toxic catabolites and other residues in the tissues which become necrotic and trigger an inflammatory reaction which overloads the lymphatics.

T*he decrease in microcirculatory resistance (vasodilatation and "steal" by the micro-shunts), accounts for* ***the <u>paradoxical association of necrosis and properly oxygenated blood.</u>***

The fragility of poorly drained tissues complicates any trauma, however slight, in ulcers. It favours <u>superinfection</u> which in turn increases inflammation and tissue destruction. This <u>vicious circle of pathological events is caused by excess TMP</u>.



Drainage failure due to Transmural Pressure TMP Excess
Edema, accumulation of toxic catabolites, Hypodermitis
Opening of micro-shunts



Venous ulcer begins at the ankle promoted by: Hydrostatic pressure + Bony subsoil + Poor drainage pathways
Aggravated and extended by superinfection

TMP reduction *by lowering the lateral intravenous pressure (IVLP) and/or increasing the extravenous pressure (EVP****) is therefore a prerequisite for lasting healing. It restores the defences against infection and relieves the lymphatic system.***

Treating the superinfection *with local antiseptic means can reduce its effects but* ***does not remove the cause.***

Any local treatment without persistent TMP lowering is doomed to failure and recurrence, despite the *illusory multiplication of local medications****. A simple, non-aggressive local antiseptic treatment, respecting the regenerative cells, can activate healing in association with the reduction of the TMP***

The mixture of 60% powdered sugar and 40% Vaseline once a week without debridement has been shown to be effective against resistant bacteria and to accelerate healing. Ref: Claude Franceschi,Massimo Bricchi, Roberto Delfrate. Anti-infective effects of sugar-vaseline mixture on leg ulcers. Veins and Lymphatics 2017; volume 6:6652.

54-Venous malformations

Congenital and hereditary venous malformations are the result of abnormalities in mesodermal embryogenesis, occurring during angiogenesis in the early stages of hematopoietic and angiopoïétique development.

They involve capillaries and veins.

Capillary malformations are presented in the form of simple, nodular, or cavernous venous megacapillaries.

The <u>venules and venous trunks</u> present as cavernomas, varicosities, varicose veins, absence of valves, hypoplasia, and truncal aplasia.

They are often <u>associated with other anomalies</u> which may be lymphatic, cutaneous, and bony-muscular malformations or secondary to the hemodynamic disturbances they produce such as ulcers, compensatory varicose veins, limb lengthening by venous hypertension*.*

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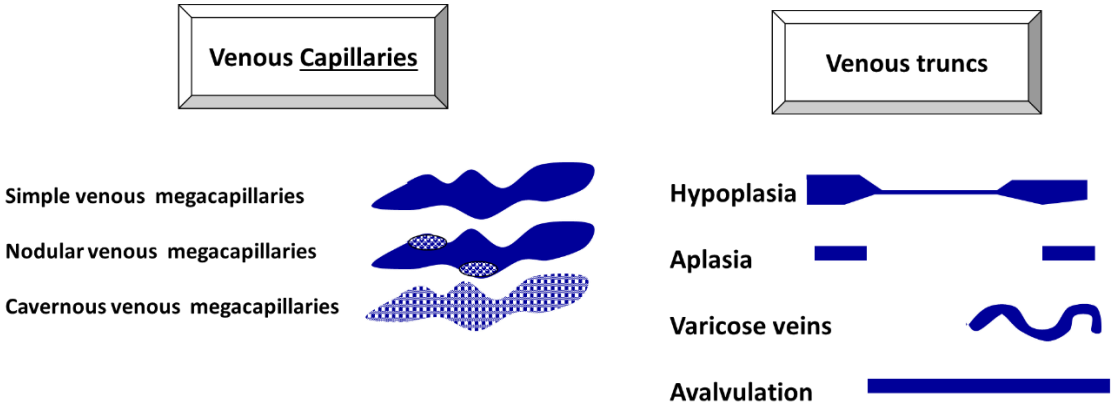


Venous Malformations

These malformations are associated in an anarchic way although sometimes metameric which do not allow a topographic and malformities classification.

Some associations have been proposed in the past.

The Klippel-Trenaunay-Weber KTW syndrome *associates superficial varicose veins, deep hypoplasia, plane angioma and elongation of a lower lim****b.***

The Proteus syndrome which *associates venous, lymphatic and osteo-muscular malformations****.***

Kasabach-Merritt syndrome is *a haematological complication (DIC) of large venous cavernomas****.***

In fact, each patient represents a variable association of.

Closed CS, Open deviated shunt and Open vicarious shunts.

Each of them must be searched for*, to be diagnosed and treated as rationally as possible, to avoid unfortunate disconnection of* ***<u>OVS which must imperatively be respected.</u>***

A simpler classification has been adopted: truncal malformations versus extra-truncal malformations.

Varicose veins of truncal venous malformations should not be confused *with those secondary to congenital arteriovenous fistulas such as Parkes-Weber syndrome because their*

diagnosis, prognosis, and treatment are very different, the latter being more frequently life-threatening.

55-Hierarchy of networks and shunts

This aspect of venous insufficiency is central to diagnosis and treatment. I will repeat here the elements already described to ensure a better understanding.

551- Hierarchy of networks

I have proposed an anatomical and hemodynamic classification of the veins and drainage compartments, based on valuable echodoppler data. Ref : C.Franceschi. *La cure Conservatrice et Hémodynamique de l'Insuffisance Veineuse en Ambulatoire CHIVA* Editions de L'Armançon. 1988 Précy sous Thil France.

This classification was then adopted by anatomists.

The venous networks of the lower extremities are classified into R1, R2, R 3, R 4 networks (translated as N1, N2, N3 networks by anatomists) and the Vn (venules network)

Located in 4 specific compartments delimited by fascias and which drain into one another according to a precise physiological hierarchy.

The inversion of this hierarchy by shunts is a major cause of venous insufficiency.

Vn, N3 and N2 drain superficial tissues, i.e. the skin for the most part, into the deep network N1.

Vn network *is made up of intradermal venules that drain through the N3 network.*

-N3 network, in the supra-fascial compartment,

-is made up of subcutaneous supra-fascial veins that drain into the N2 network or into the N1 network via perforators.

-drains the Vn venules from the microcirculatory units and the "phlebosomes", cutaneous drained territories specific to each vein of the N3 network. Thus, occlusion of a vein will disturb the drainage of its phlebosome

- The N2 network is in a compartment formed by a fascial sheath.

It consists of the trunk of the Greater Saphenous Vein (GSV), which can be recognized in the "Egyptian eye" delimited by the fascial separation of the thigh, and the Small Saphenous Vein (SSV)

It does not drain directly the Vn microcirculatory units, from which it receives blood via the veins of the N3 network.

-It drains into the deep N1 network via perforators and the saphenofemoral and saphenopopliteal junctions.

-Giacomini's vein is also part of the N2 network.

-It joins the arch of the small saphenous vein to the trunk of the great saphenous vein, which drains into N1.

-It can also drain directly into N1 via a perforator. The calibre of these veins varies greatly from one subject to another.

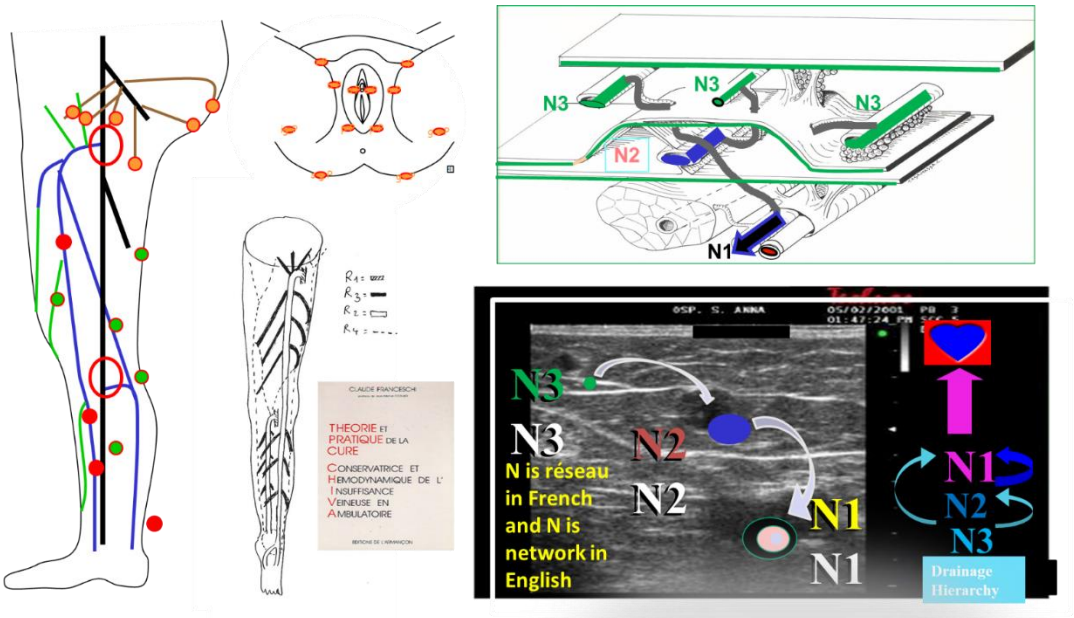
-They are sometimes partially aplastic but without any hemodynamic pathological impact. It is therefore not the calibre alone that can be considered pathological.

*-**N4 networks connect segments of N2, N2>N4>N2***

-<u>Longitudinal N4L</u> connects 2 levels of the trunk of the great saphenous vein

-<u>Transversal N4T</u> connects the trunk of the great saphenous vein with that of the small saphenous vein.

***The N1 network consists of** all deep veins located in the subfascial compartment. It drains superficial tissues indirectly via N2 and N3. Its flow drains the skin and all other tissues and muscles of the lower extremities towards the heart (cardiopetal*)

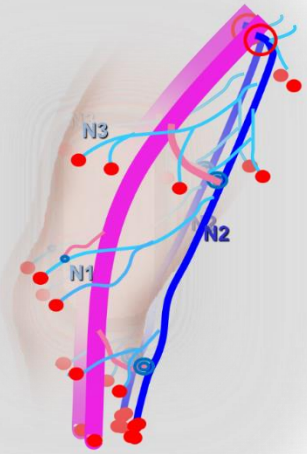


Potential escape points

- **Saphenofemoral**
- **Saphenopopliteal junction**
- **Pelvic leakage points**
- **Numerous perforators**

Networks in 4 hierarchical levels of drainage

- **N1, N2, N3, N4 according to their topography and hemodynamic function**



- ***This hierarchy of drainage, N3>N2>N1 and N3>RI is functionally physiological****. It <u>does not matter which direction the flow</u> is in a vein if it drains hierarchically.*

<u>True reflux is not the retrograde direction, but the direction between two networks contrary to the drainage</u>

-A N3 vein flow remains "physiological" regardless of its direction (retrograde, classically called "reflux" or orthograde) if it drains only its phlebosome into N2 or N1.

-Similarly, a N2 vein remains "physiological" regardless of its direction (retrograde or orthograde) if it drains N3 into N1*. For this reason, <u>a reverse (retrograde) flow in a vein, classically called reflux, has no pathogenic significance if it respects the hierarchy</u>**.

-A "reflux" (retrograde flow) in a segment of the trunk of a great saphenous vein GSV N2 draining into N1 is not pathogenic but "physiological" *if it is not overloaded by N1 flow: N2>N1. It is pathological if it receives an N1 flow, thus in the opposite direction to the hierarchy: N1>N2>N1.*

- On the other hand, an N2 or N3 flow can be of normal orthograde direction *but pathological.*

-This is the case, for example, of ***the flow normally descending from an N3 epigastric vein towards the arch of the great saphenous vein but which becomes pathological without changing direction when it is overloaded by N1 pelvic blood via a N1>N3>N2>N1 pelvic escape point.***

- This is also the case for ***the trunk of the great saphenous vein N2 whose ascending flow, therefore not refluxing, is pathological because it is overloaded by a systolic escape point N1>N2 to by-pass a deep obstacle N1: N1>N2>N1.***

-I call true pathological reflux, not only any retrograde drainage but also orthograde when it makes 2 networks communicate in the opposite direction to the hierarchy. It occurs when the incompetence of the communications between the networks causes the abnormal overloading of one network by another less superficial one. N2>N3, N1>N2, N1>N3.

-In practice, true reflux is any flow that passes from a deeper network into a more superficial one.

These inversions of hierarchy take place at the junctions between networks. These reflux junctions are called "escape points" EP.

These hierarchically reversed flows N1>N2, N2>N3, N1>N3 then drain into N1 by crossing what I have called the RP re-entry points (saphenofemoral and saphenopopliteal junctions, perforators).

The pathophysiological relevance of hierarchical flow inversions is not univocal. *This value varies according to the conditions of their occurrence. At rest, during systole or diastole of the different cardiac, thoracoabdominal and especially valvulomuscular pumps.*

552- Veno-venous shunts

A veno-venous shunt is a vein whose physiological drainage flow is overloaded by a flow that it"steals" from another vein through an escape point EP and that it restores through a re-entry point RP.

These shunts have the common characteristic of reversing the physiological hierarchy of drainage between the various networks and compartments.

They differ according to the conditions of their activation (rest, dynamic diagnostic manoeuvres (Paranà, Valsalva) and their effects on the Transmural pressure TMP.

Reminder. *Veno-venous shunts are responsible for most of the excess transmural pressure, and therefore for the signs and symptoms of venous insufficiency (pain, heaviness, oedemas, trophic disorders, varicose veins, hypodermatitis, ulcers). Their diagnosis is essential for hemodynamic treatment. They are characterized by the diversity of their anatomical and hemodynamic configuration that only the echodoppler can recognize and map.*

Remember that a ***shunt is in physics a conduit that diverts a fluid. It is closed when it returns back the fluid to its upstream source and open when it directs it elsewhere downstream****.*

I call a veno-venous shunt a vein or several veins that diverts all or part of the blood from other veins. It contains physiological drainage blood from the capillaries, which is overloaded by all or part of the blood it diverts. This blood is diverted via an escape point EP and returned via a re-entry point RP.

It is an ***open vicarious shunt OVS*** *when it restores the diverted flow downstream of the occluded vein at rest pushed by the residual pressure, and/or at effort under the thrust of the systole of the valvulo-muscular pump.*

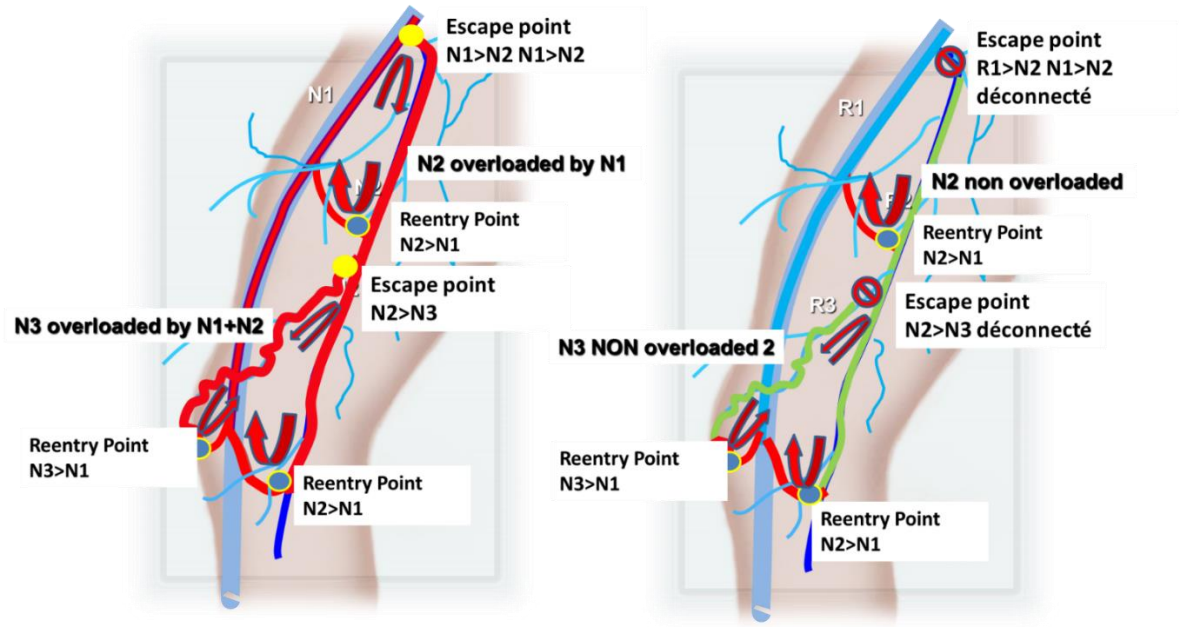
It is an ***open deviated shunt ODS*** *N2>N3>N1when activated by the diastole of the valvulo-muscular pump which sucks in N1 the N2 blood diverted by N3.*

It is a ***closed CS shunt*** *when all or part of the blood pushed by the systole downstream of the valvulo-muscular pump, returns back upstream during the diastole via superficial (or deep) veins that it overloads. This is the concept of the private circuit imagined by Trendelenburg that I confirmed and improved thanks to echodoppler.*

It is a ***mixed shunt*** *consisting of an open vicarious shunt OVS combined with a closed shunt CS . They share the* ***same escape point EP*** *but which drain via* ***different re-entry points RP.***

The notion of hemodynamic overload helps to understand why shunts dilate the veins and disturb tissue drainage*. It explains why* ***treatment must eliminate this overload without eliminating the physiological draining flow****. Destroying the veins of the shunts removes the overload,* ***BUT*** *also the physiological drainage with its consequences in terms of tissue suffering and recurrences. Only a precise diagnosis of each hemodynamic configuration can provide the best "taylord" treatment. Only echodoppler mapping allows these diagnoses. To*

do so, it must be performed according to rigorous static and dynamic protocols, which can be acquired by ***those who make the effort to learn and understand the hemodynamic physiopathology of venous insufficiency.***



Example of SHUNT I N1>N2>N1 (closed shunt) + SHUNT II N2>N3>N1 (open shunt by deviation
N2 and N3 overloaded with reflux through leak points N1>N2 and N2>N3 by inversion of the drainage hierarchy
Disconnection of the shunts: N2 and N3 remain in reflux, but respect the drainage hierarchy: collapsed varicose veins and physiological drainage respected

5521- *-Superficial shunts*

55211- Closed superficial shunts CS.

Flow activated necessarily by both

-Diastole of the valvulo-muscular pump: Paranà positive , compression relax psitive

-Systole of the thoraco-abdomnial pump: Valsalva positive V+.

Escape points:

The escape points are fed by the deep network N1.

They first overload N2 (N1>N2) or N3 (N1>N3)

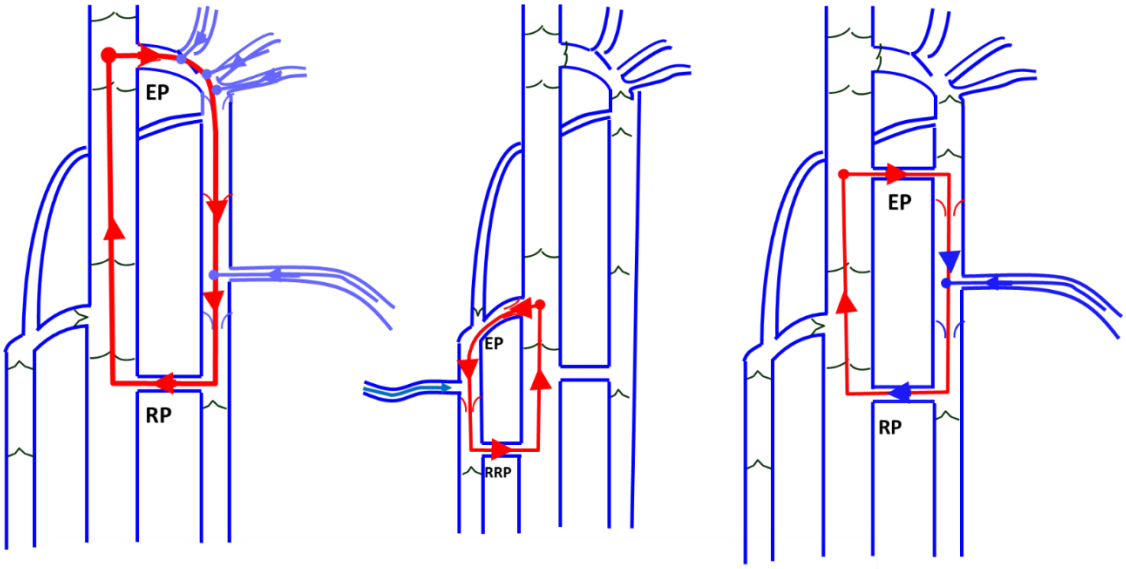
to re-enter by Re-entry Points N2>>N1 or N3>N1 after a pathway feature depending on the veins used (Shunts I, II, IV, V, VI and MS).

SHUNT TYPE I.

EP escape point: Saphenofemoral junction or thigh or leg trunk perforator.

Pathway: N2.

Re-entry: N1



SHUNT 1:Closed Shunt N1>N2>N1: 3 examples of closed SHUNT CS . Recirculation. N2 Overload by N1
3 examples
EP:Escape Point, SFJ, SPJ and Thigh Perforator. RP: Reentry Points

SHUNT TYPE III:

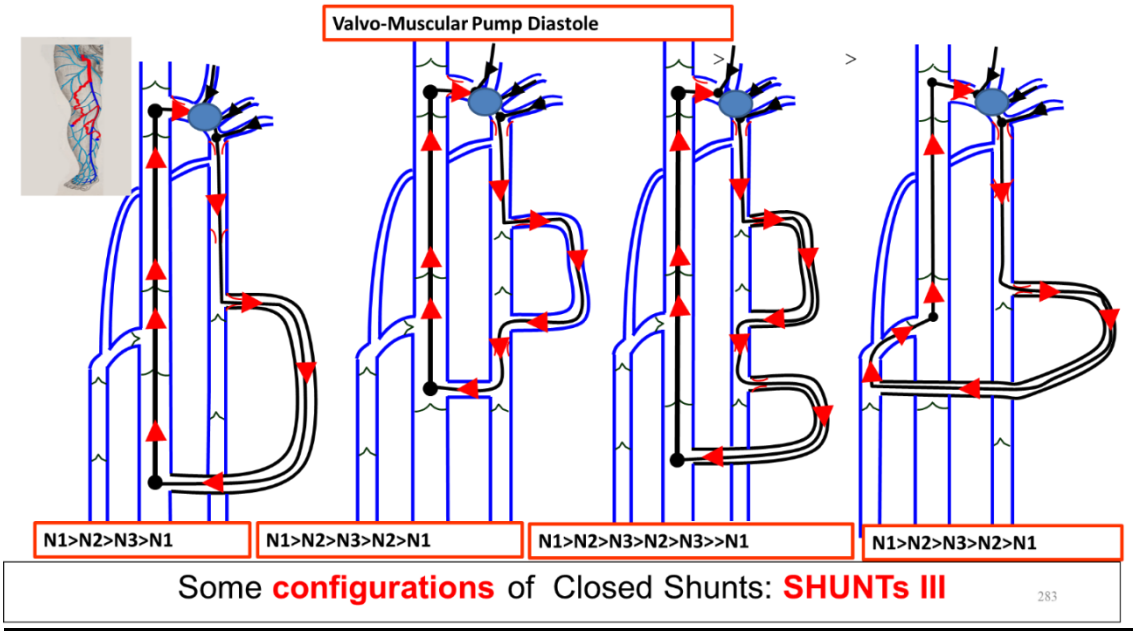
EP escape point: Saphenofemoral junction or thigh or leg trunk perforator.

Pathway: N2>N3.

<u>Note that there is no N2>N1 re-entry on the N2 saphenous trunk path</u>.

Re-entry: N1

Shunt III subgroups according to the pathway: N1>N2>N3>N1, N1>N2>N42>N2>N1, N1>N2>N4L>N3>N1, N1>N2>N4T>N2>N1. <u>Note again that there is no re-entry on N2.</u>

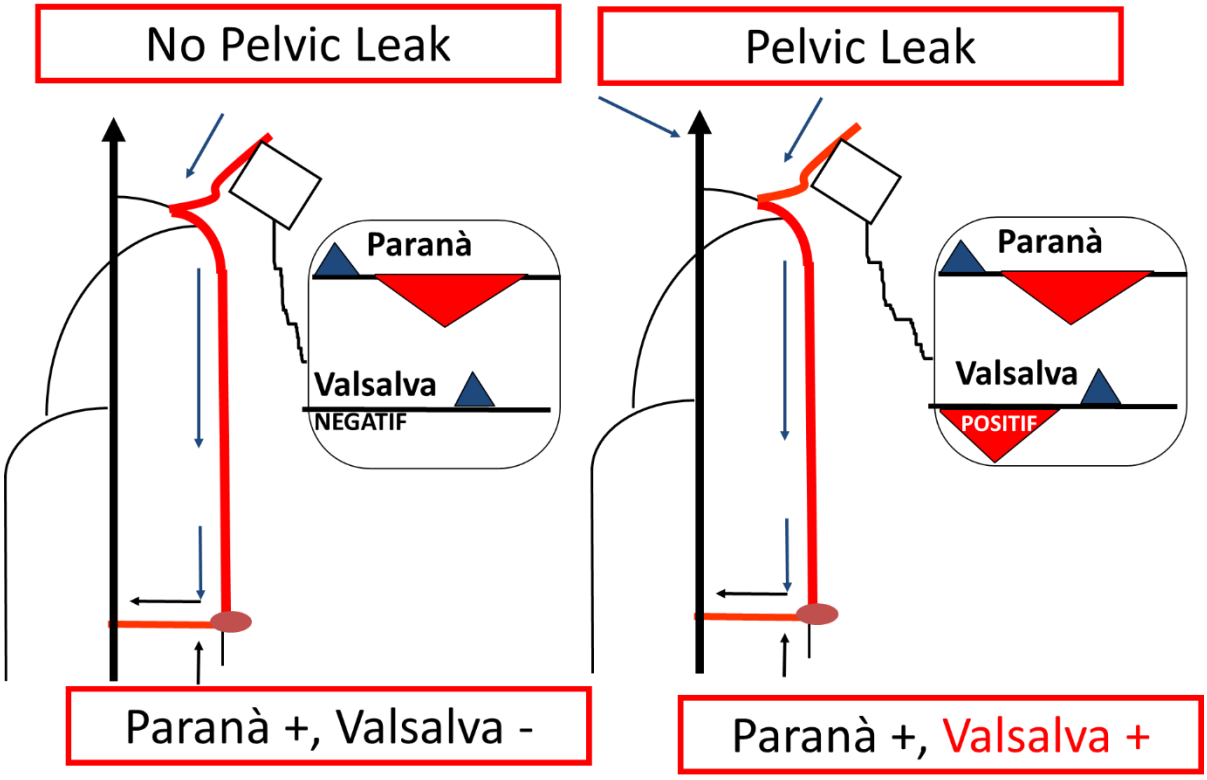


SHUNT TYPE IV: N1>N3>N2>N1

EP escape point: <u>pelvic escape points</u> N1>N3.

Pathway: N3>N2

Re-entry: N2>N1,

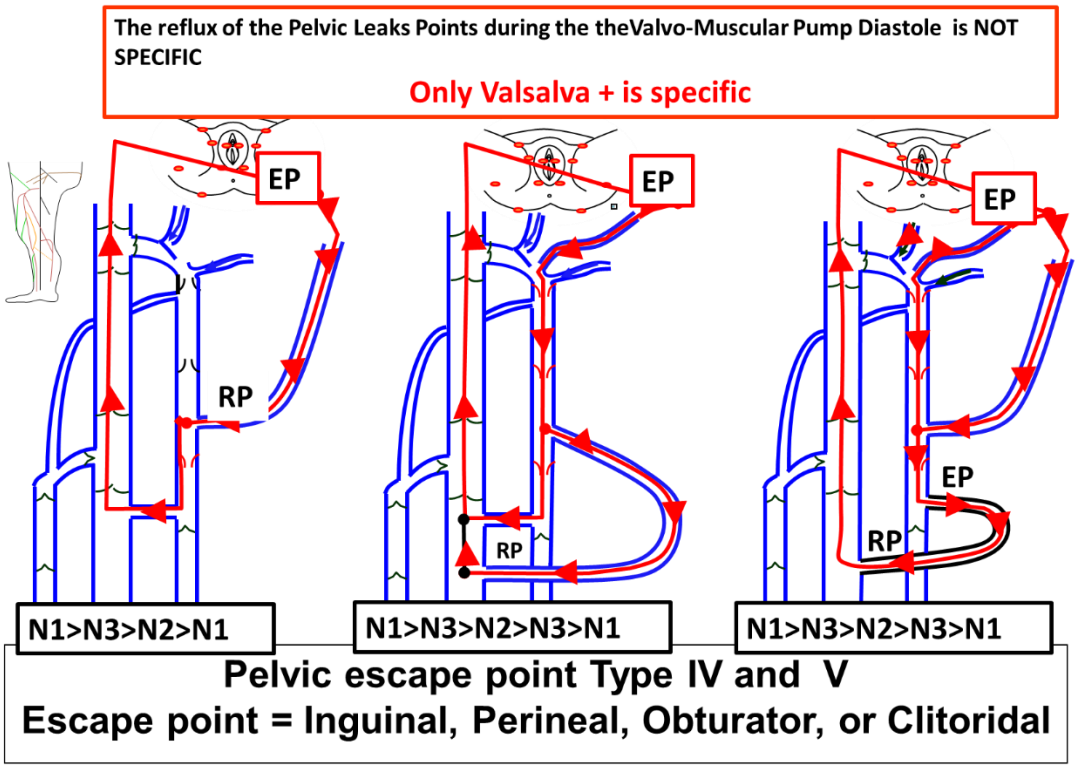


SHUNT TYPE V: N1>N3>N2>N3>N1

EP escape point: <u>pelvic escape points</u>.

Pathway: N3>N2>N3.

Re-entry: N1

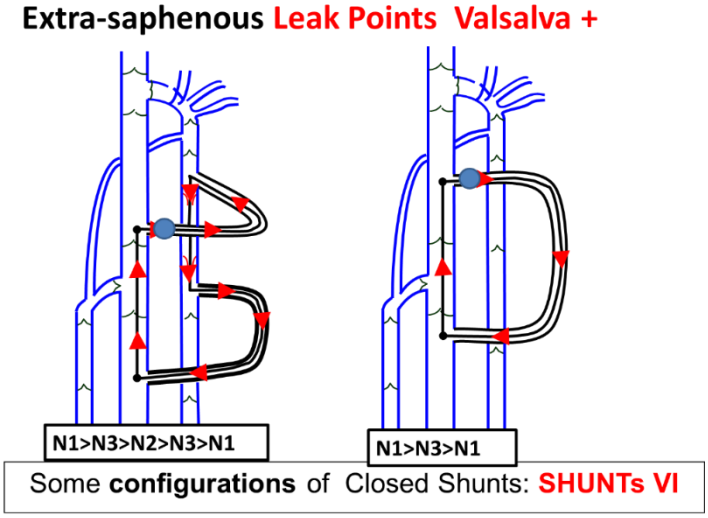


SHUNT TYPE VI:

Escape point EP: <u>Extra-saphenous perforator</u>.

Pathway: N3.

Re-entry: N1



55212- Open deviated shunt ODS : Shunts Type II

Superficial escape point N2>N3

SHUNTS TYPE II, not fed by N1, are open deviated shunt ODS and more rarely closed CS:

Activated by diastole of the valvulo-muscular pump(Paranà +, Compression relax +) but negative Valsalva V- (no systolic Valsalva reflux).

ODS:

Escape point EP: N2-N3 junction

Pathway: N3 or N4 (look at sub groups below).

Re-entry: N1.

Subgroups: (not necessarily to remind…but to map)

1-Junction N2-N3.

pathway: N4T.

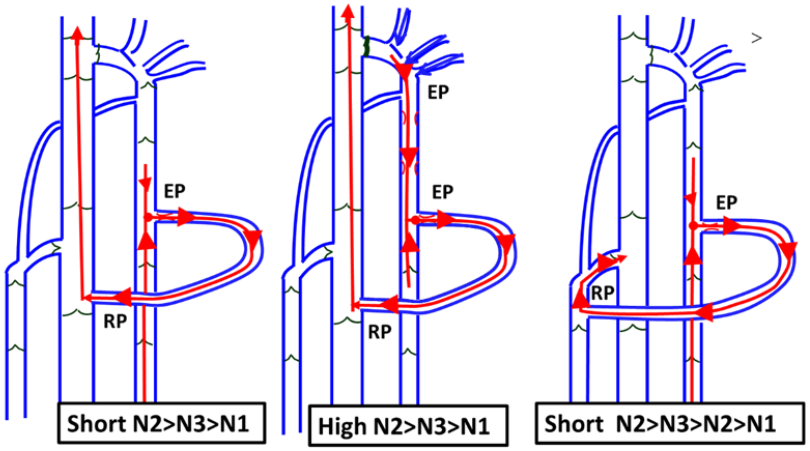
Re-entry N2

2-SHUNT Type II a N2>N3>N1 without N2 incompetence.

3-SHUNT Type II B N2>N3>N1 with N2 incompetence, but without EP N1>N2 nor RP N2>N1.

4-SHUNT Type II C: N2>N3>N1 N2>N1 (shunt 0): common N2 but different N2>N1 and N3>N1 re-entries.

5-SHUNT N4L closed but without deep escape point: EP escape point: N2-N3 junction. path: N4L. Re-entry N2: N2>N4L



SHUNT open by deviation ODS. No Recirculation.
Type II N2>N3>N1 and N2>N3>N2>N1 : N3 Overload by N2 3 examples
EP = Escape point N2>N3 PN = Re-entry points N3>N1 and N3>N2

55213- *Shunts O*

<u>By definition, a shunt with no escape point cannot be called that because it is not overloaded</u>. However, we have called Shunt 0 the refluxes without an escape point.

Their function is <u>not pathogenic</u> because they drain only their "phlebosme" only and according to the <u>physiologic hierarchy</u>

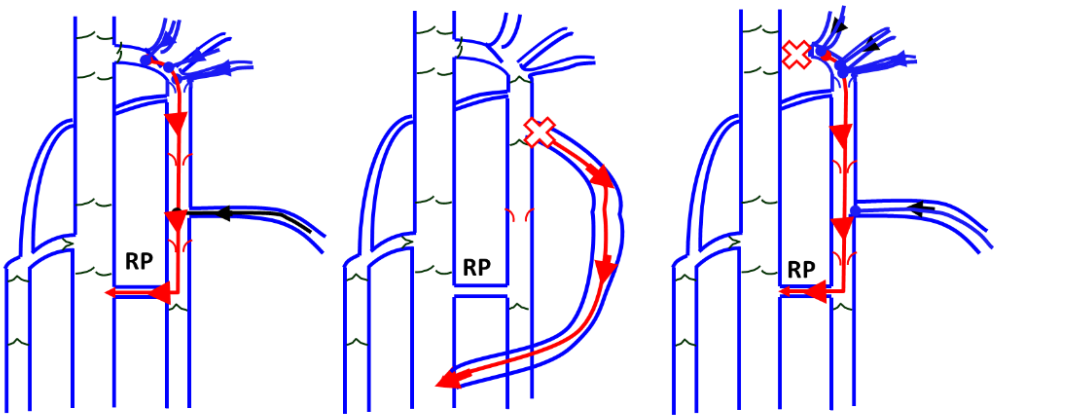
SHUNT TYPE 0:

No escape point EP:

Pathway: N2 or N3

Re-entry N1

They are <u>activated by the diastole of the valvulo-muscular pump</u> (Paranà +) <u>but negative Valsalva</u> (no systolic reflux during the Valsalva thrust).



SHUNT O : N2>N1 segmental reflux not overloaded

SHUNT O : N3>N1 post disconnection N2>N3 of an open deviated shunt type II (CHIVA)

SHUNT O : N2>N1 post disconnection N1>N2 of a closed shunt Type I (CHIVA)

Shunt Type 0 NON pathological
No EP= Escape points N1>N2 nor N2>N3 nor N1>N3 nor N2>N3
PR = Reentry points N2>N1 and N3>N1

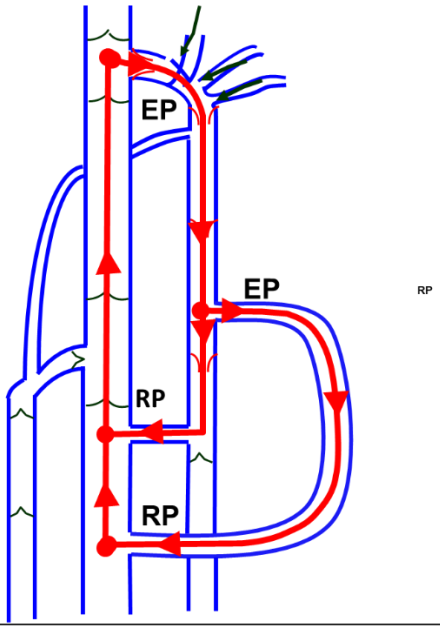
55214 -*Combined superficial diastolic shunts:*

Combined superficial diastolic shunts.

SHUNT I +II: N1>N2>N1a + N2>N3>N1b, (SHUNT II connected to SHUNT I).

SHUNT I + IV: N1>N2>N1 + N3>N2 (SHUNT IV connected to SHUNT I)

SHUNT III+ V: N1>N2>N3>N1 + N3>N2>N3>N1 (SHUNT V connected to SHUNT III) , common N2 and N3



Combined Shunt Closed Type I + Open by Deviation Type II
EP= Escape points N1>N2 and N2>N3 PN = Re-entry points N2>N1 and N3>N1

.

55215- *Superficial systolic Open Vicarious shunts OVS*

By-pass of

-superficial obstruction N2 (constitutional or acquired or iatrogenic): N2>N3>N2 or

-N1 Deep obstruction shunt: N1>N2>N1, N1>N2>N3>N1, N1>N3>N1 etc. depending on the succession of superficial vicarious veins.

55216 - Mixed Superficial Shunt: MS

A venous shunt is mixed MS when it <u>combines</u>

an <u>open vicarious shunt OVS and a closed shunt CS</u>.

OVS and CS have <u>in common</u>

an escape point N1>N2 and

a first segment N2

-that <u>reflux (antihierarchical direction :anterograde or retrograde) successively in systole and diastole</u>.

<u>Then N2 divides into 2 pathways (N2 and or N3)</u> to <u>two different re-entries</u> (N2>N1and/or R>3) of which the

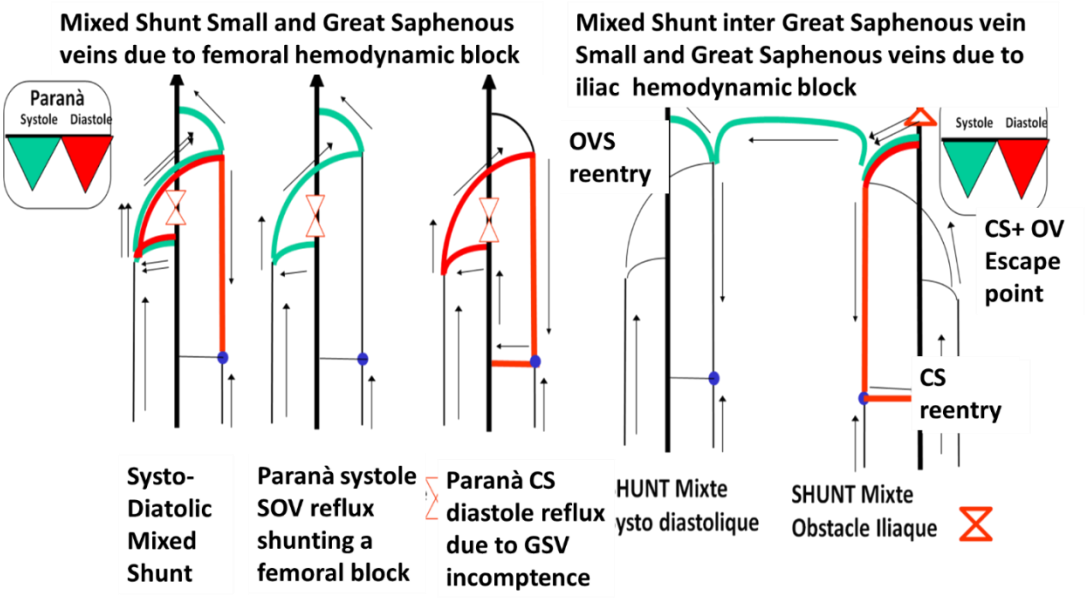
OVS one is activated only in systole and

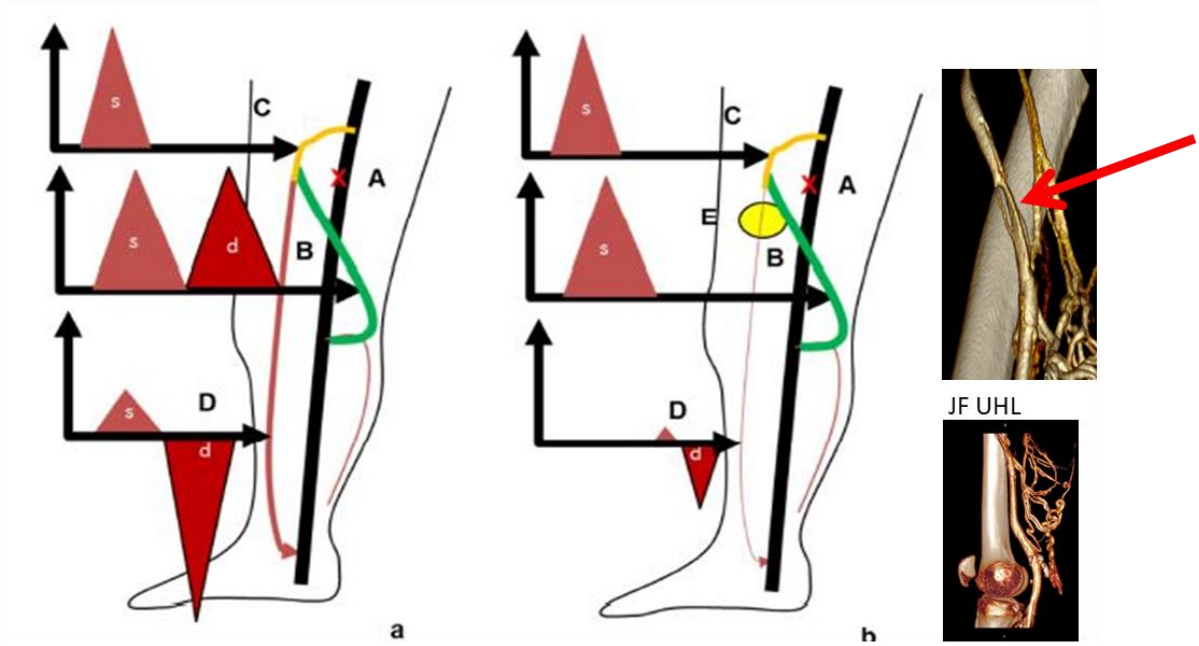
-the CS one only in diastole.

They are <u>mainly seen in 2 cases</u> of hemodynamic obstruction.

<u>Obstacle of the superficial femoral vein</u> acquired or congenital (stenosis at the level of Hunter's canal that I had evoked with the Doppler because of the OVS and a CS of the saphenous grade associated in a MS fed by a saphenopopliteal escape, confirmed later in the cadaver).

<u>Iliac obstruction</u> compensated by a <u>spontaneous Palma</u> via the cross-over between the great saphenous veins.





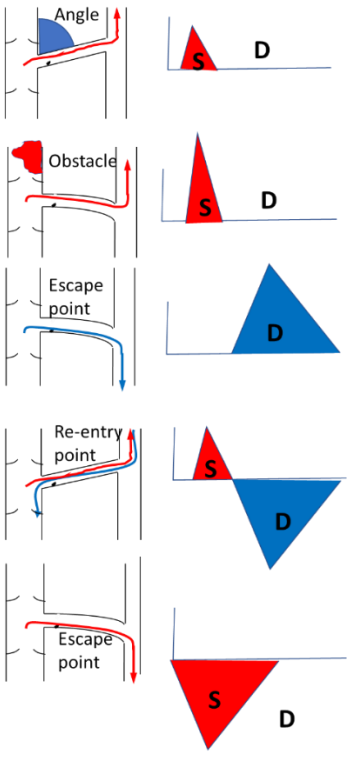
Obstacle of the Superficial Femoral Vein diagnosed hemodynamically by echodoppler that I called vicarious open shunts and mixed shunts and treated by CHIVA before anatomical confirmation by Dr JF UHL

55217: Perforators

Perforator refluxes and calibre meanings according to echodoppler

C;Franceschi, R.Delfrate, M.Cappelli

- **During the valvomuscular pump activation (Paranà, Squeezing-relaxation)**
  - Systolic reflux of perforator forming an acute upward angle with the deep vein without deep downstream obstacle is due to incompetence. It is not pathogenic.
  - Systolic reflux due to deep obstacle is pathologic but not pathogenic because compensatory , so to be respected . It is the escape point of an open vicarious shunt.
  - Diastolic reflux is pathogenic: escape point of a closed shunt.
  - Biphasic perforators (systolic reflux followed, by an inward re-entry diastolic flow) are not pathogenic if the diastolic flow prevails over the systolic reflux. It is usually the re-entry perforator of a shunt (Closed shunt CS, Open Deviated Shunt ODS, Shunt 0)
- **During Valsalva maneuvre**
  - Systolic Vasalva reflux is always pathogenic
- **Most of the time, large calibre doesn't mean reflux but overloaded to be respected re-entry**



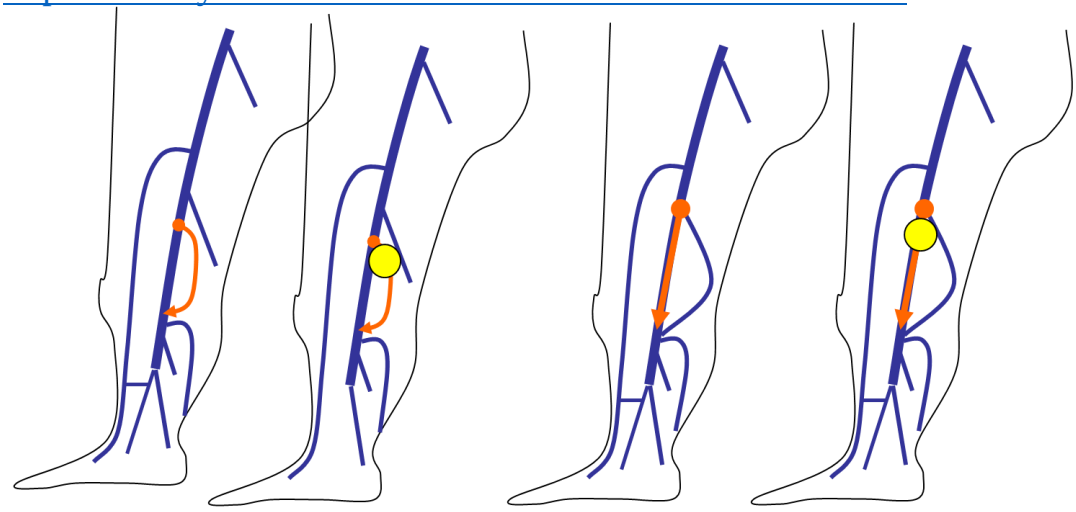
55218 - *Classification of diastolic deep shunts*

Deep veins present closed shunts when an incompetent deep venous segment A is connected by its two ends to a competent deep vein B. A suck in B during diastole.

This is often the case for closed shunts of the superficial femoral vein supplied either by its competent collateral or by the deep femoral vein.

This is also the case for closed shunts of the posterior tibial vein supplied either by its competent collateral or by the competent perineal vein.

Video: Deep closed shunts in Deep CHIVA:
https://www.youtube.com/watch?v=t6vGDEwx9XI&t=230s



Double superficial femoral vein with an incontinent collateral is a closed shunt corrected by CHIVA

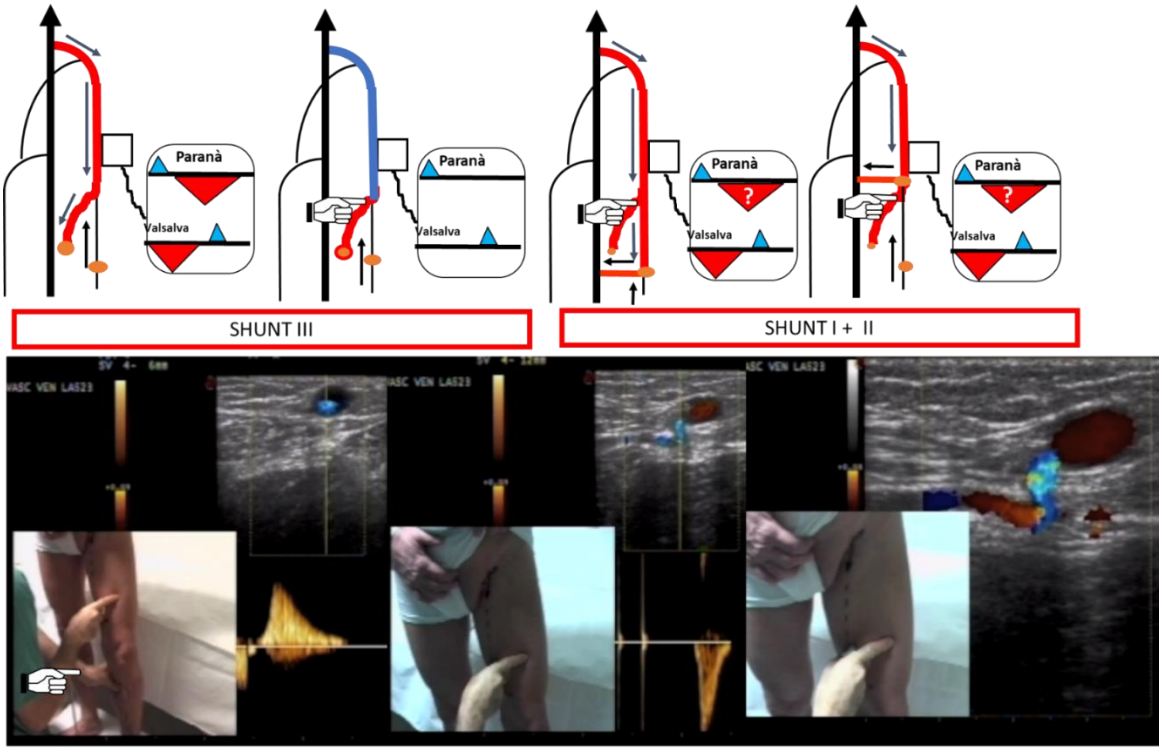
Incontinent superficial femoral vein and continuous deep femoral vein connected to the popliteal vein is a closed shunt corrected by CHIVA

The variability of the anatomy of the femoral veins is crucial for the treatment of venous insufficiency

55219. *Practical and theoretical shunts*

This detailed classification is useful to define each type of shunt to adapt a specific strategy. However, it is important to memorize the basic principles and to look for the types of escape point to differentiate between closed shunts CS, open deviated shunt ODS, open vicarious shunts OVS and mixed shunts MS whose differentiation are the key points of the CHIVA therapeutic strategy.

In current practice, it is the differential diagnosis between type III shunts and SHUNT I + II, for which the CHIVA therapeutic strategy must be established most often. This differentiation is provided by a particular test under echodoppler.



Differential diagnosis SHUNT III vs SHUNT II+I

Effective manual compression of N3: if diastolic reflux -Paranà or compression-release of the N2 trunk of the Great Saphenous Vein persists, a re-entrant perforator must exist on N2. When the perforator drains into the femoral, the Paranà or compression-release maneuver may not show it. The Valsalva maneuver is essential to show it.

Chapter 6

Each chapter includes some of the elements of the previous chapters and anticipates those of the following chapters.

6- Clinical venous insufficiency

61-Definition

62--Diagnostic clinical conditions and patient information

63-Limitations of the clinical examination and CEAP

64-History

65-Signs and symptoms

651-Chronic venous insufficiency

6511-Heat intolerance

6512-Essential varicose veins and varicose veins

6513-Deep vein thrombosis DVT disease

6514-Pelvic varicose veins

65141-Pelvic congestion syndrome.

65142-surfacevaricose veins of pelvic origin.

65143-Hemorrhoids.

6515-Venous malformations

6516- "Physiological" venous insufficiency.

65161- "Varicose veins" in athletes

65162- "Physiological venous insufficiency" and lifestyle.

6517-Ulcer

652-Acute venous insufficiency

6521-Sudden swelling of the extremities

6522-Painful swelling of the foot related to a non-displaced fracture.

6523-Acute venous insufficiency in pregnant women

66-Differential diagnosis

661-Sudden oedema

662-Chronic oedema

6621-Bilateral white oedema

6622- Unilateral oedema related to an unilateral cause

663-Dermohyperodermatitis

664-non-venous ulcer.

6641-Arterial ulcers

6642-Necrotic angiodermatitis (Martorell's ulcer)

6643-Basal cell carcinomas or squamous cell carcinomas

6644-Ulcers due to infectious, degenerative, hematologic diseases

665-non-venous pain

67- Clinical manoeuvres

671-Persistence of visible varicose veins in the supine position

672-Painful Homans manoeuvre

673- Perthes test

6- *Clinical venous insufficiency*

61-Definition.

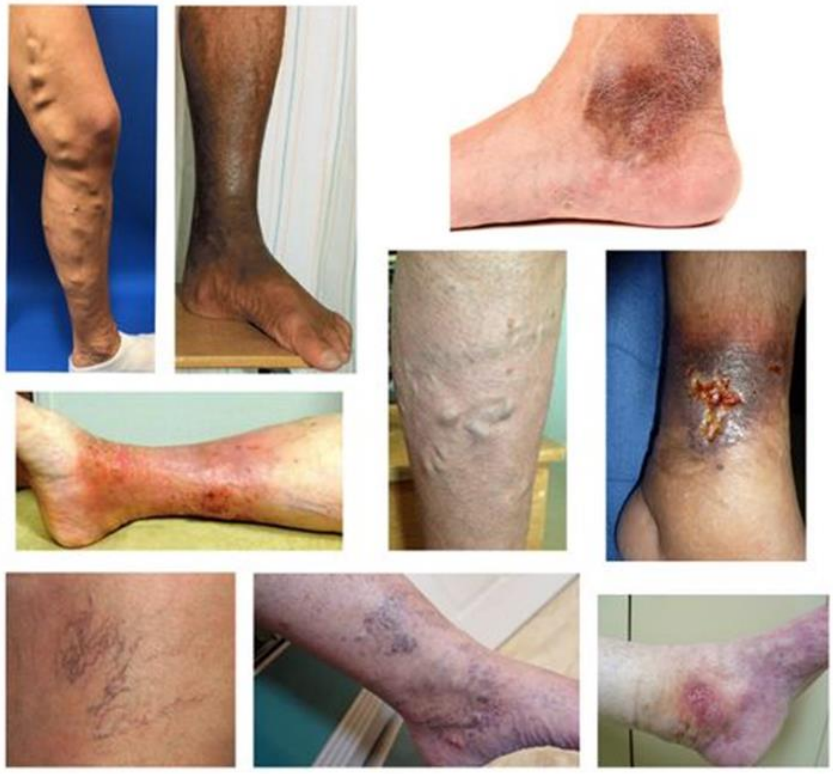
There is no venous insufficiency without an excess of transmural pressure, whatever the hemodynamic cause.

Clinical signs and symptoms of venous insufficiency occurs when hemodynamic venous insufficiency is sufficiently important to alter one or more venous functions. Most hemodynamic impairments may be asymptomatic.

Signs *are the objective visible and/or palpable abnormalities, and* ***symptoms*** *are the subjective abnormalities perceived by the patient.*

Most signs and symptoms are not specific to venous insufficiency. Therefore it is necessary to eliminate any other cause, lymphatic, neurological, visceral or osteo-muscular, including in a patient with varicose veins, varicosities or oedema.

Whatever the etiology, signs and/or symptoms, venous insufficiency is always due to an excess of Transmural Pressure.



62-- *Clinical conditions of the diagnosis and patient information.*

Patients most often consult for 3 types of reasons

Usually for unsightly manifestations (varicose veins, spider veins),

More rarely for severe signs and symptoms of trophic disorders (hypodermitis, ulcers).

Exceptionally For serious complications of simple varicose veins (haemorrhages, phlebitis, pulmonary embolism), or

<u>The answer must be clear</u>.

This is where "informed consent" takes on its full importance, both ethically and medico-legally.

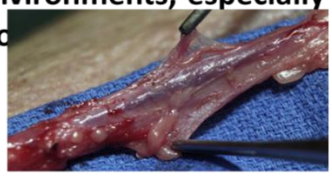
In the first case, ***reassure the patient by confirming that his pathology is benign and its complications rare and easy to avoid by simply wearing support socks.***

*In the second and third cases**, explain to the patient the possibilities and real limits of the various treatments according to the results of the Doppler hemodynamic mapping.***

*In all cases, **inform the patient that the great saphenous vein**, whether competent or not, is a precious material in case of necessity of **a vital arterial by-pass.** **Add that it can be preserved by medical (compression stockings, lifestyle) and/or surgical (CHIVA) treatment methods, conservative and hemodynamic of venous insufficiency.***

Bioprotec (Lyon France)
collects, freezes and sells stripped great saphenous veins as allografts

The great saphenous vein, continuous or incompetent
-Aorto-coronary bypasses
- Peripheral bypasses
Patch, vascular access
Bypass surgeries in septic environments, especially pro



- **No touch" harvesting**
- **Less spasm**
- **No dilation (less endothelial trauma)**
- **Conservation of vasa vasorum,**
- **less parietal ischemia**
- **Conservation of NO synthesis (less intimal hyperplasia**

Problème éthique de la destruction du capital veineux (G.DE WAILLY)

Principe de non malfaisance

- Probabilité de la nécessité d'un pontage artériel après chirurgie veineuse : 3% (1)
  - chirurgies veineuses : 200 000 / an + 6000 000 sclérothérapies

⇒ **6000 pontages / an**

- Pontages veineux aorto-coronariens
  - 70% des malades ayant des varices avaient des segments veineux compatible avec réalisation du PAC (2)

(1) Lofgren EP. In Bergan JJ, Yao JST (eds). Surgery of the veins 1985 285-299
(2) Cohn et al, Ann Thor Surg 2006 81(4) 1269-4

The informed consent I provide to patients is this:

Varicose vein treatment:

The saphenous vein is vital for future peripheral and coronary venous bypasses and should not be destroyed without the informed consent of the patients, especially since this benign disease can be treated simply by compression socks or minimally invasive surgical methods (CHIVA cure), which are less expensive, ALWAYS conservative and validated (CHIVA), and

better than destructive surgical (stripping) or endovenous techniques (sclerosis, foam, laser, Radiofrequency C).

The great saphenous vein (GSV) in healthy subjects, but also presenting varicose veins (the saphenous veins removed from a patient to treat his varicose veins are frozen by a Lyon France -based company (BioProtec) and then sold to perform bypasses on another patient. It can be harvested in the patient who needs a coronary or limb by-pass surgery.

Scientific studies:

A- Coronary venous by-pass surgery equivalent to mammary artery by-pass surgery

1-No touch technique of saphenous vein harvesting: Is great graft patency rate provided?

Papakonstantinou NA J Thorac Cardiovasc Surg. 2015 Oct;150(4):880-8. doi: 10.1016/j.jtcvs.2015.07.027. Epub 2015 Jul 15.

2-The no-touch saphenous vein for coronary artery by-pass grafting maintains a patency, after 16 years, comparable to the left internal thoracic artery: A randomized trial. Samano R1, ClinicalTrials.gov NCT01686100.Copyright © 2015 The American Association for Thoracic Surgery. Published by Elsevier Inc. All rights reserved.

B- great Saphenous bypass of the lower extremity arteries is still the most effective. -

1-Meta-analysis of infrapopliteal angioplasty for critical limb ischemia

Marcello Romiti, (J Vasc Surg 2008;47:975-81.)

2-Meta-analysis of popliteal-to-distal vein by-pass grafts for critical ischemia

Maximiano Albersand (J Vasc Surg 2006; 43:498-503.)

C- CHIVA cure

1-CHIVA method for the treatment of chronic venous insufficiency. Bellmunt-Montoya S1, Cochrane Database Syst Rev. 2015 Jun 29;(6):CD009648. doi: 10.1002/14651858.CD009648.pub3

2-Hemodynamic classification and CHIVA treatment of varicose veins in lower extremities (VVLE)Hua Wang1, et al, China. Int J Clin Exp Med 2016;9(2):2465-2471 www.ijcem.com /ISSN:1940-5901/IJCEM0016552 "".

3- Carandina S, Mari C, De PAlma M, Marcellino MG, Cisno C, Legnaro A, et al. Varicose Vein Stripping v hemodynamic Correction (CHIVA): a long-term randomized trial. European Journal of Vascular and Endovascular Surgery 2008;35(2):230-7

4- Pares JO, Juan J, Tellez R, Mata A, Moreno C, Quer FX, et l. Varicose vein surgery: stripping versus the CHIVA Method: a randomized controlled trial. Annals of Surgery 2010;251(4):624-31

5- Iborra-Ortega E, Barjau-Urrea E, Vila-Coll R, Ballon-Carazas H, Cairols-Castellote MA. ComPArative study of two surgical techniques in the treatment of varicose veins of the

lower extremities: results after five years of follow-up. Estudio comparativo de dos técnicas quirúrgicas en el tratamiento de las varices de las extremidades inferiores: resultados tras cinco años de seguimiento]. Angiología 2006; 58(6):459-68.

6-] P. Zamboni and all: Minimally Invasive Surgical management of primary venous Ulcer vs. compression Eur J vasc Endovasc Surg 00,1 6 (2003)

7- Chan, C.-Y. a, Chen, T.-C. b, Hsieh, Y.-K. a, Huang, J.-H.c Retrospective comparison of clinical outcomes between endovenous laser and saphenous vein-sparing surgery for treatment of varicose veins (2011) World Journal of Surgery, 35 (7), pp. 1679-1686

8- The CHIVA method gives less recurrence than destructive methods (Stripping, Laser, radiofrequency, sclerosis, foam). Guo et al. Long-term efficacy of different procedures for treatment of varicose veins A network meta-analysis. Medicine (2019) 98:7

63-*Limitations of the clinical examination and CEAP.*

The CEAP classification is only clinical and describes only the progressive stages of the disease, whatever the cause. It does not allow for the identification or hemodynamic classification of venous insufficiency.

Symptoms and signs, such as sensations of heaviness during the day, oedema, lower limb swelling, varicose veins, varicosities, ulcers do not provide sufficient elements to develop an appropriate therapeutic strategy.

Therefore, topographic and hemodynamic mapping is essential. It is only of value if it is performed according to a rigorous method, capable of recognizing and evaluating the different types of veno-venous reflux and shunts.

Only hemodynamic echodoppler can diagnose and evaluate the cause and pathophysiological configuration (topography and types of shunts) responsible for the TMP excess at the origin of the signs and symptoms.

64-*The history should investigate the conditions of onset and evolution of signs and symptoms as well as the notion of episodes of venous thrombosis and hemodynamic or destructive treatments.*

65-*Signs and symptoms.*

Pelvic pain, heaviness, oedema, dyschromia, ulcer of the lower extremities are non-specific signs and symptoms of venous insufficiency. *Pelvic and lower limb varicose veins are signs of venous insufficiency BUT not specific to a particular hemodynamic or etiologic form. However, some signs and symptoms can be linked to their causes in particular clinical conditions.*

651-*Chronic venous insufficiency*

6511*-Heat intolerance** ***may be related to an inadequacy of venous flow to the needs of thermoregulation by excess or by lack of sufficient drainage (varicose stasis)

6512-The essential varicose veins and varicosities

The so-called essential varicose veins are the most frequent cause of chronic venous insufficiency. They are called "essential" because they are often familial, with no defined aetiology.

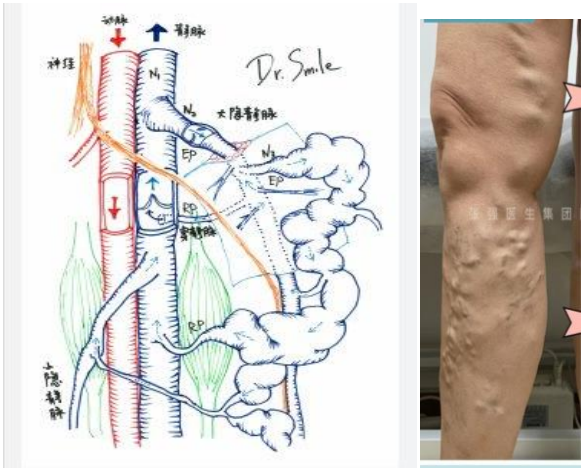
They are most often benign and asymptomatic. Although in a small percentage, they are nevertheless related to trophic complications (hypodermitis, ulcers) and superficial thrombosis.

They are not the cause, but the effect of a common hemodynamic disorder.

This means that the rational treatment is not the destruction of the varicose vein, but the correction of its hemodynamic cause (reduction of TMP by compression, antigravitational postures and CHIVA treatment).

In accordance with the ethical and legal rules of informed consent, the patient must therefore be informed of the benignity and the possibilities of effective non-destructive functional and aesthetic treatment of his varicose veins.

But above all, he must be informed that his great saphenous veins, whether competent or incompetent, will be of vital interest in case of need for venous by-pass of an occluded artery. Indeed, the great saphenous vein is still the most reliable by-pass material for distal bypasses (limb salvage) and comparable to the mammary artery (thoracic) for coronary by-pass when it is harvested by the "no touch" method. Ref: 1. No touch technique of saphenous vein harvesting: Is great graft patency rate provided? Papakonstantinou NA J Thorac Cardiovasc Surg. 2015 Oct;150(4):880-8. doi: 10.1016/j.jtcvs.2015.07.027. Epub 2015 Jul 15. 2-The no-touch saphenous vein for coronary artery by-pass grafting maintains a patency, after 16 years, comparable to the left internal thoracic artery: A randomized trial. Samano R1, ClinicalTrials.gov NCT01686100. 2015 The American Association for Thoracic Surgery.3-Meta-analysis of infrapopliteal angioplasty for critical limb ischemia Marcello Romiti, (J Vasc Surg 2008;47:975-81.)4-Meta-analysis of popliteal-to-distal vein by-pass grafts for critical ischemia Maximiano Albersand (J Vasc Surg 2006; 43:498-503.)



65122- Varicosities

Varicosities are made of small blue or red dilatation.

<u>They are not always associated with a reflux N3 of the saphenous or extra-saphenous tributaries.</u>

Their treatment is most motivated by aesthetic demands. ***VIDEO***
https://www.youtube.com/watch?v=JScby8a0zZY&t=8s

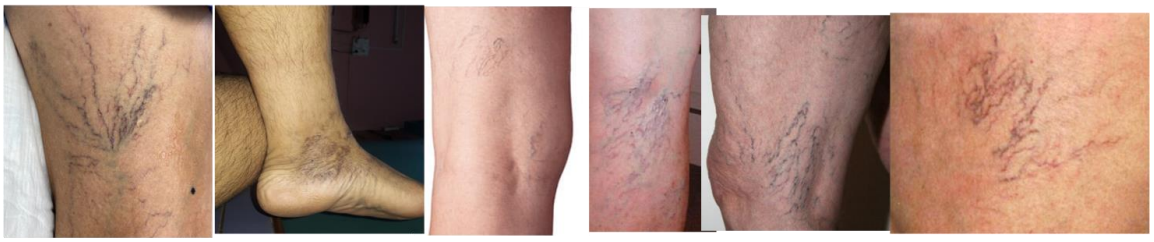
Most often located on the medial face of the knees and the lateral face of the thighs.

They look often like an upright or inverted tree, the trunk of which may be the overloading or draining vein.

They also can indicate an obstacle to venous drainage due to poverty or an obstacle to subcutaneous drainage (cellulitis).

They may also be due to reflux or upstream N2 and/or N2 obstacles.

This is how they frequently occur *<u>after procedures to destroy varicose veins, which increase the residual pressure in the capillaries and venules, sometimes very high, opening arterio-venous micro-shunts, as in red varicose veins called "matting</u>".*



Varicosités

6513-*Post deep venous thrombosis T disease PTD*

Post thrombotic PTD disease is clinically recognized in <u>the context of a history of deep thrombophlebitis.</u>

It is associated with varying degrees of heaviness, venous claudication, trophic disorders, ulcers, swelling of the limb depending on the importance of the occlusion and sequential valvular incompetence. <u>Obstruction of iliac and/or ilio-caval location is suspected when the swelling involves the thigh and/or suprapubic varicose veins are seen (spontaneous Palma)</u> .Ref: Fanceschi C.: Hémodynamique de la maladie postphlébitique : conséquences diagnostiques et thérapeutiques . Journal des Maladies Vasculaires 2008 Volume 33, numéro S1

6514-Pelvic varicose veins

<u>Pelvic varicose veins are common and asymptomatic in most single or multiparous women</u>, due to the "arteriovenous fistula" effect of the placenta for 9 months.

However, they can be complicated by the

-clinical syndrome of pelvic congestion and/or

-lower limb varicose veins by the opening of pelvic escape points that I have located precisely with the echodoppler. Ref: 1. Franceschi C, Bahnini A. Points de fuite pelviens viscéraux et varices des membres inférieurs. Phlébologie 2004;57:37-42.2. Franceschi C, Bahnini A. Treatment of lower extremity venous insufficiency due to pelvic escape points in women. Ann Vasc Surg 2005;19:284-8. 3. Franceschi C. Anatomie fonctionnelle et diagnostic des points de fuite bulboclitoridiens chez la femme (point C). J Mal Vasc 2008;33:42.

Dilatation and thrombosis of the hemorrhoidal veins are more often a complication of an attack on the anal canal than the effect of an incompetence or venous reflux. It appears from my studies and their beneficial therapeutic consequences that the primary cause is an inflammation/infection of the anal canal. Ref: C.Franceschi. Hémorroïdes : maladie des veines ou d'un quatrième facteur. Essai d'analyse physiopathologique. Conséquences thérapeutiques. Actualités Médicales Internationales. Angiologie (8), n° 145, décembre 1991 VIDEO https://youtu.be/1FoYynLlb98***.***

65141-Pelvic congestion syndrome.

Its definition is clinical, <u>based on symptoms,</u> <u>not signs</u>

It is the association of varying degrees of uninterrupted symptoms for a few months.

Pelvic pain that can be very disabling, aggravated by orthostatism and predominant at the end of the day, urgent urination, dyspareunia and even sciatic neuralgia.

<u>But this syndrome is not specific</u>, including when it is associated with pelvic, perineal, and vulvar varicose veins.

<u>The diagnosis can only be made after eliminating any other gynaecologic cause.</u>

65142-Superficial varicose veins of pelvic origin

These varicose veins occur <u>mainly in single or multiparous women</u>.

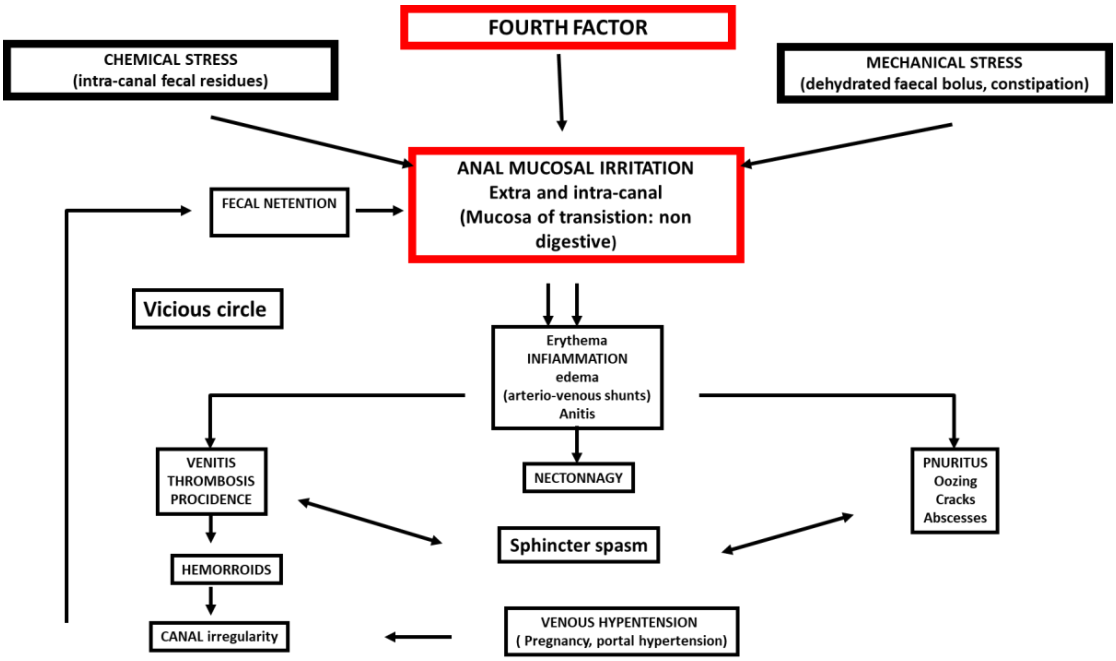
They are sometimes, but not always, visible, and palpable at the level of the perineum, the labia majora of the superficial ring of the inguinal canal.

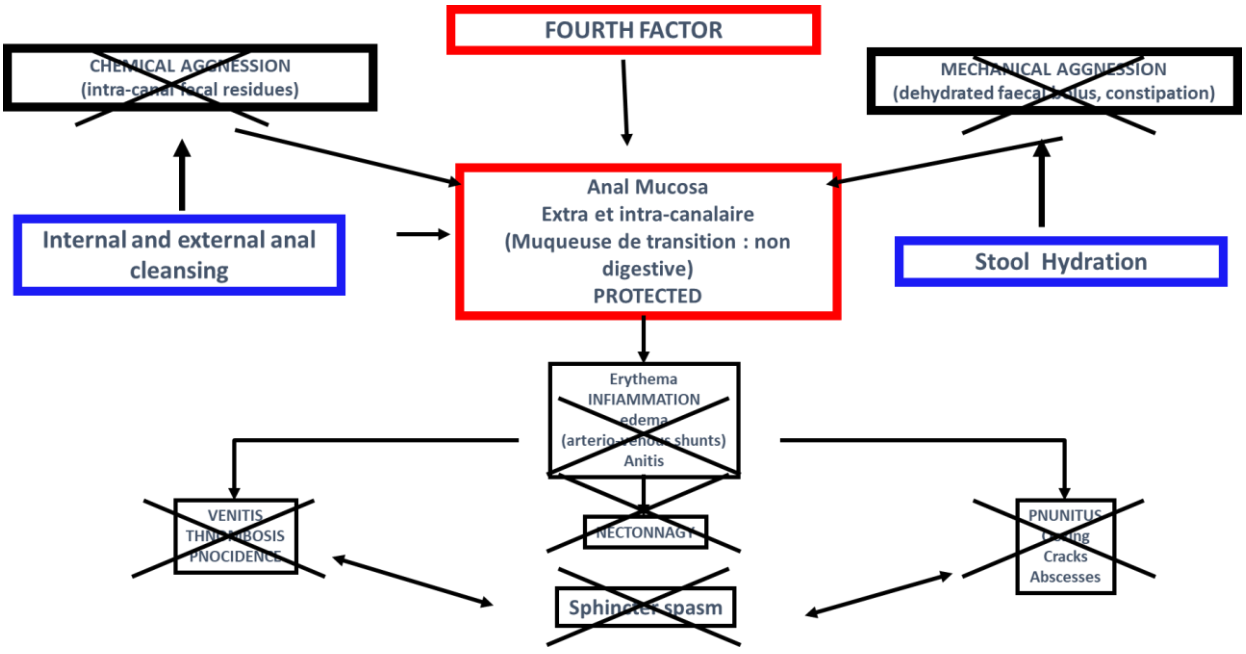
Varicose veins of the buttock are seen more rarely because they appear mainly in venous malformations.

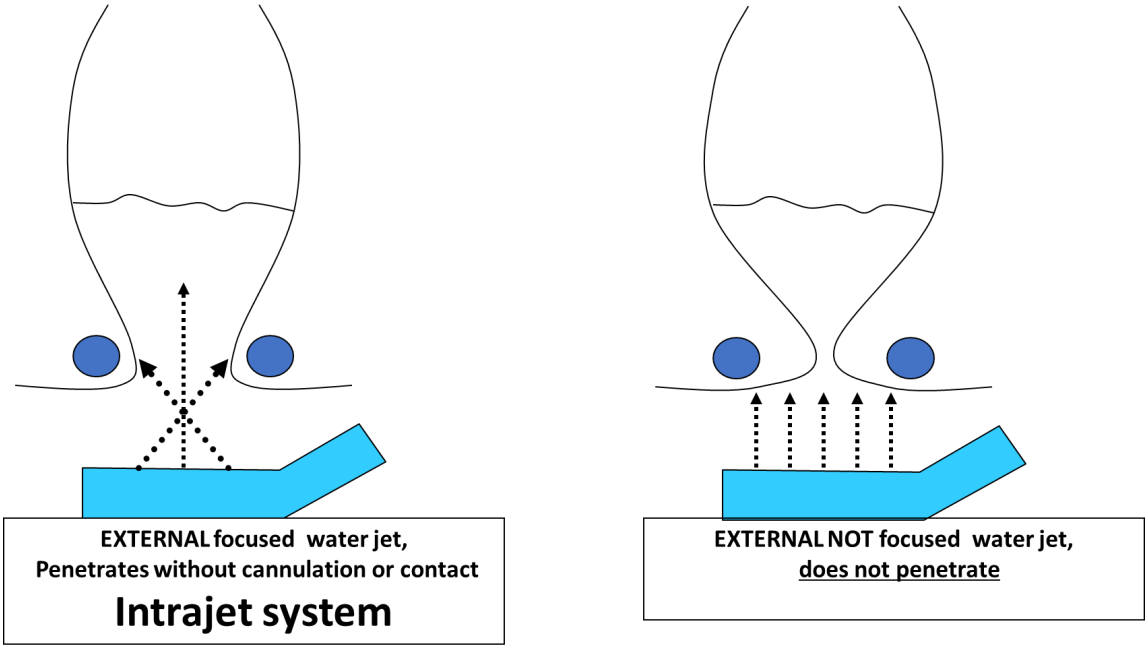
65143-Hemorrhoids.

Anorectal varicose veins, internal, external, retractable, or not, depending on their stage, thrombosed or not, are rarely due to venous hypertension. They are most often secondary to the <u>4th factor as I described in11991</u>. Ref: C.Franceschi. Hémorroïdes : maladie des veines ou d'un quatrième facteur. Essai d'analyse physiopathologique. Conséquences thérapeutiques. Actualités Médicales Internationales. Angiologie (8), n° 145, décembre 1991 VIDEO https://youtu.be/1FoYynLlb98**.**

This factor is the intolerance of the mucous membrane of the anal canal (transitional mucosa, not digestive, like the oropharynx) which becomes inflamed, traumatized, and infected by the mechanical and chemical contact of faecal matter. The hemorrhoidal veins, just under this mucosa, would suffer the consequences. The hemorrhoidal crisis of the pregnant woman is facilitated by the dilation of the hemorrhoidal veins which deform the mucous membrane of the canal. This deformation favours the pathogenic contact with faecal matter. An RCT study demonstrated the effect of pre and post defecation washing of the anal canal by a simple jet of water penetrating without contact with the cannula which remains at a distance from the anus. I have released the patent. Intrajet can therefore be freely copied


FOURTH FACTOR
CHEMICAL STRESS
(intra-canal fecal residues)
MECHANICAL STRESS
(dehydrated faecal bolus, constipation)
ANAL MUCOSAL IRRITATION
Extra and intra-canal
(Mucosa of transistion: non digestive)
FECAL NETENTION
Vicious circle
Erythema
INFIAMMATION
edema
(arterio-venous shunts)
Anitis
VENITIS
THROMBOSIS
PROCIDENCE
NECTONNAGY
PNURITUS
Oozing
Cracks
Abscesses
Sphincter spasm
HEMORROIDS
CANAL irregularity
VENOUS HYPENTENSION
(Pregnancy, portal hypertension)

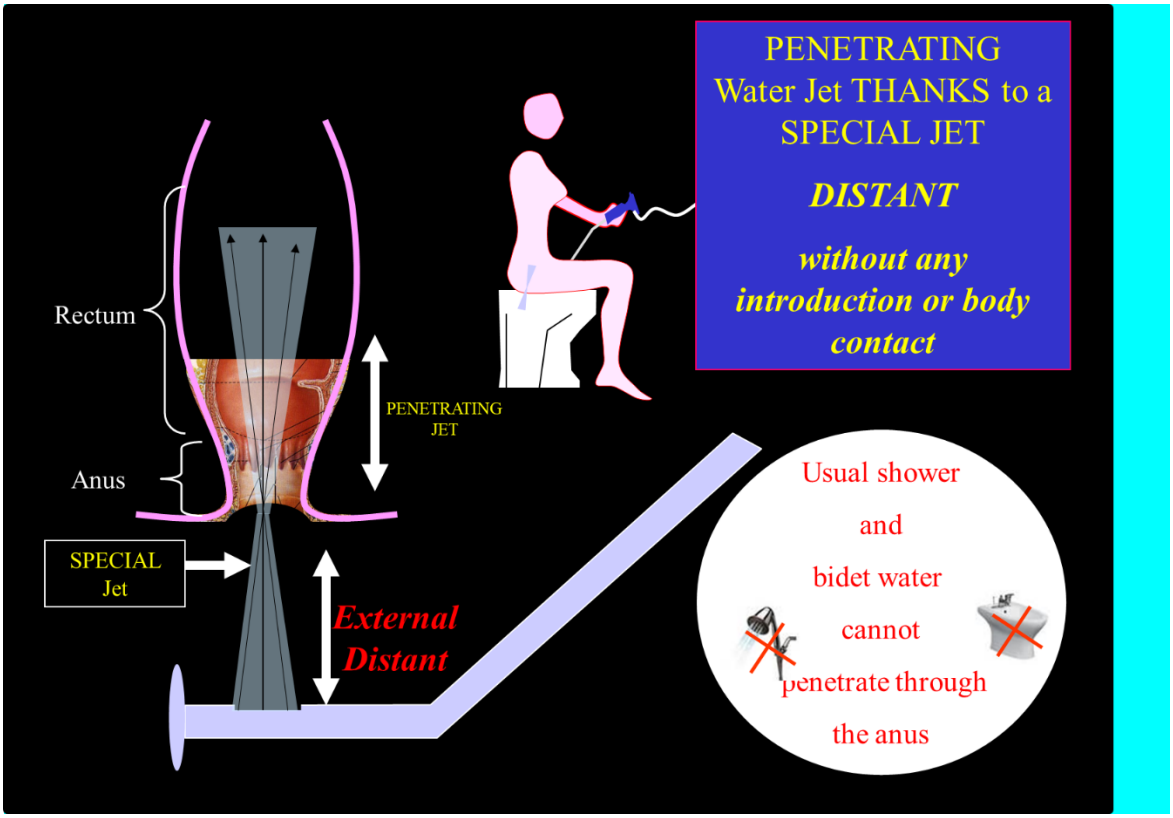

FOURTH FACTOR
CHEMICAL AGGNESSION
(intra-canal fecal residues)
MECHANICAL AGGNESSION
(dehydrated faecal bolus, constipation)
Internal and external anal cleansing
Anal Mucosa
Extra et intra-canalaire
(Muqueuse de transition : non digestive)
PROTECTED
Stool Hydration
Erythema
INFIAMMATION
edema
(arterio-venous shunts)
Anitis
VENITIS
THROMBOSIS
PNOCIDENCE
NECTONNAGY
PNUNITUS
Oozing
Cracks
Abscesses
Sphincter spasm



EXTERNAL focused water jet,
Penetrates without cannulation or contact

Intrajet system

EXTERNAL NOT focused water jet,
does not penetrate



MEDECINE ET CHIRURGIE DIGESTIVES
Tome 24 - N°2
Mars - Avril 1995

Med. Chir. Dig. 1995 – 24 - 109-111

ACTUALITES THERAPEUTIQUES

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Evaluation de l'efficacité et de la tolérance d'un nouveau procédé de traitement des hémorroïdes symptomatiques : Intrajet®*

*B. VERGEAU**, R. CLEMENT**, M. MASSONNEAU***, C. FRANCESCHI*****

(Vincennes, Paris)

Introduction

Les hémorroïdes feraient souffrir un sujet sur trois et constituent une véritable maladie sociale. L'étude IJ 301 avait pour but d'évaluer l'efficacité et la tolérance d'Intrajet® dans le cadre des hémorroïdes symptomatiques. Intrajet® est un dispositif qui permet l'introduction d'eau dans le canal anal au moyen d'un jet dont la particularité principale est d'être pénétrant sous pression modérée sans canulation ni contact de l'appareil avec le périné. Cette action est rendue possible grâce aux caractéristiques de focalisation et d'orientation du jet.

Cette étude était fondée sur une approche physiopathologique privilégiant l'intolérance de la muqueuse du canal anal aux résidus même minimes de matières fécales (C. Franceschi).

Matériel et méthode

Principe d'Intrajet®

Nous avons élaboré un système permettant d'une part de contrecarrer l'agression mécanique en ramolissant le bol fécal distal avant la défécation et d'autre part de supprimer les résidus fécaux consécutifs à la défécation sans agression chimique ou mécanique ni risque de contamination. Il fallait enfin que le système soit d'un emploi simple, quotidien, non contraignant et peu onéreux. Le principe d'Intrajet® consiste à faire pénétrer dans le canal anal, un jet d'eau, émis à distance de l'anus par un appareil externe, donc sans contact et non contaminant, de pression assez faible pour ne pas traumatiser ni remonter au-delà du bas du rectum, de forme et direction particulières afin de pouvoir être pénétrant. Ce jet est émis avant et/ou après la défécation pendant 4 à 6 secondes. Le dispositif Intrajet® est constitué d'une canne vectrice reliée à l'alimentation d'eau par un tuyau souple et munie d'un robinet poussoir en son manche, recourbée de 40° à son extrémité, de sorte que tenu entre les cuisses par le patient assis sur la cuvette des W.C, l'orifice de sortie du liquide aménagé dans cette extrémité se trouve en face et dans la direction du canal anal. Cet orifice est constitué d'une fente particulière en ce qu'elle génère un jet plat et triangulaire, dont la pointe se forme à 25mm de l'orifice pour se repartir en un léger éventail.

Protocole IJ 301

L'étude IJ 301 a obtenu l'accord du CCPPRB de la Pitié Salpétrière en 1992, a duré 16 mois et s'est interrompue en novembre 1993. Cette étude a été placée sous la responsabilité scientifique du Dr Bertrand Vergeau, chef de service d'endoscopie digestive de l'Hôpital d'Instruction des Armées Bégin. Il a été nécessaire d'utiliser une méthodologie originale car reposant sur un matériel d'hydrothérapie et non sur un médicament, il n'était pas possible d'utiliser un placebo. Il a donc été décidé de tester deux jets d'eau différents :

- Un jet sous pression modérée, orienté et non focalisé, assimilé dans le protocole à un placebo, qui est une douchette anale améliorée. Les douchettes n'ont jamais fait la preuve d'une action thérapeutique dans un protocole de ce type.

- Un Intrajet®, qui lui est un jet sous pression modérée, orienté et focalisé et permet un lavage externe équivalent au précédent et y associant un lavage interne du canal anal.

L'expérimentation IJ 301 a été réalisée en double aveugle contre placebo, ni le médecin ni le patient ne pouvant savoir quel était des deux jets celui qui était à sa disposition. Pour cela le protocole imposait au médecin lors de la première consultation une présentation de l'étude ne spécifiant pas la notion de pénétration. Après accord signé du sujet, un Intrajet® ne disposant pas de la canne terminale était installé dans les 24 heures. Le praticien revoyait le patient le troisième jour, lui remettait une enveloppe scellée et randomisée contenant soit une canne de jet externe soit une canne Intrajet® à effet

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**** Société IODP, 11 rue Ferdinand Duval. 75004 Paris.*
***** 12 avenue de Wagram, 75008 Paris.*

externe et interne. L'examen de départ était très complet et le patient acceptait de subir un examen proctologique et anuscopique complet à J-3, J+1, J+15 et J+90 jours.

Population

La population étudiée comprenait 31 patients présentant des hémorroïdes symptomatiques qui avaient donné leur accord pour participer à cette étude. L'un des patients est revenu sur sa décision dans le délai de réflexion de 3 jours. Le choix d'une consultation hospitalière avait pour but de tester l'efficacité d'Intrajet® auprès d'une population sévèrement atteinte et ayant subi de nombreux traitements antérieurs. Il apparaît que 20 patients présentaient une gêne quotidienne importante ou très importante à l'inclusion, et que 26 patients souffraient d'hémorroïdes depuis plus de 10 ans.

Le groupe bénéficiant du jet externe seul comprenait 16 patients.

Le groupe bénéficiant d'Intrajet® comprenait 15 patients.

Les deux groupes étaient équivalents en ce qui concerne tous les critères d'âge, de sexe, de poids et de taille et ne présentaient pas de différences statistiquement significatives.

Résultats

Sur le critère principal qui était l'amélioration globale ressentie par le patient, 53 % des patients sous Intrajet® (8/15) ont estimé dès le 15ème jour que l'amélioration globale était importante ou très importante contre seulement 25 % (4/16) dans le groupe sous jet externe, $p < 0.001$, (Fig. 1). L'un des patients bénéficiant de ce seul jet externe décrit déjà son action comme plus efficace qu'une simple douchette anale qu'il utilisait auparavant. A 90 jours, la satisfaction globale à l'égard d'Intrajet® se maintient.

En ce qui concerne les critères secondaires d'étude, aucun n'est statistiquement significatif car il s'agit de sous-groupes avec un nombre de patients trop petit. Cependant la diminution de la douleur est remarquable dans les deux groupes puisque sur l'ensemble des patients, l'intensité de la douleur est globalement divisée par quatre, la réduction du prurit est également importante dans les deux groupes puisque le prurit est globalement divisé par trois. L'amélioration du suintement est très en faveur d'Intrajet®, pouvant être considérée comme statistiquement significative, puisque le suintement est globalement divisé par cinq dans le groupe Intrajet® alors qu'il n'est divisé que par deux dans le jet externe seul (Fig. 2).

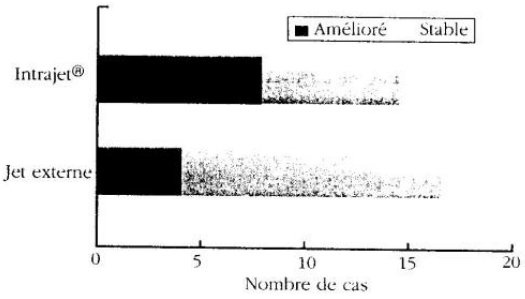


Fig. 1 - Critère principal d'étude : amélioration globale ressentie par le patient
Amélioration avec Intrajet® 53% (8cas) versus Jet externe 25% (4 cas) (p<0.001).

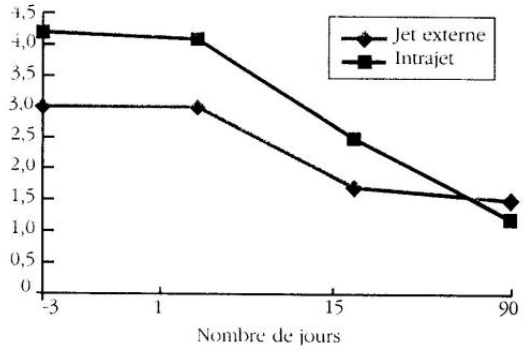


Fig. 2 - Evolution moyenne du suintement au cours du temps Echelle analogique visuelle horizontale (p non significatif)

Les patients présentant des diarrhées et bénéficiant d'Intrajet® n'en ont plus décrit à trois mois, l'un des patients sous jet externe seul présentait toujours des diarrhées à trois mois. Les patients présentant une constipation importante et bénéficiant d'Intrajet® n'en ont plus ressenti dès le quinzième jour, ce qui est confirmé à trois mois, l'un des patients sous jet externe seul présentait toujours une constipation à trois mois. La disparition totale des saignements lors de l'essuyage était observée dans le groupe Intrajet® dès J15 chez les six patients concernés ; chez les huit patients concernés sous lavage externe, quatre en souffraient encore à J15 et un à J90.

En ce qui concerne l'évolution des hémorroïdes, Intrajet® comme le lavage externe, entraîne une diminution modérée en taille des hémorroïdes et même une action étonnante sur les marisques qui ne peut s'expliquer que par une action anti-inflammatoire péri-hémorroïdaire.

Intrajet®, réduit plus rapidement le prolapsus hémorroïdaire avec disparition des cas de prolapsus II B dès le quinzième jour contre trois mois pour le lavage externe.

Les hémorroïdes compliquées semblent bénéficier d'Intrajet® puisque trois cas sur sept ne sont plus compliqués à trois mois contre aucune amélioration des six cas sous lavage externe seul. Enfin on note une disparition des cas de thrombose dès J15 dans les deux groupes et surtout

6515- *Venous malformations*

Venous malformations have very variable clinical manifestations, from a simple varicose vein to strong asymmetries of the extremities with important varicose veins, oedemas and cutaneous angiomas, sometimes better systematized as in the Klippel Trenaunay Weber syndrome and the Proteus syndrome. They all have the characteristic of having started in early childhood. Their complexity is specific to each patient and requires instrumental MRI and Echodoppler investigations.

6516-*Physiological" venous insufficiency*

Physiological" venous insufficiency is caused by an excess of TMP despite a normal venous system.

65161- *Varicose veins" in athletes*

In the athlete, superficial veins appear to be of large calibre because they are not masked by subcutaneous fat. Most often, they are continuous and simply dilated due to the hyper flow associated with intense physical activity. The treatment of their possible incompetence must be particularly conservative because of the high physiological superficial flow/pressure during sports activities.

65162- *"Physiological venous insufficiency" and lifestyle.*

Symptoms and signs of venous insufficiency (heaviness, pain, oedema, hypodermitis), exist in subjects whose lifestyle does not allow the dynamic fractionation of gravitational hydrostatic pressure DFGHSP.

Standing or sitting still for too long.

Warm places trigger a thermoregulatory reflex (decrease of the microcirculation resistance) which increases the motive pressure flow/surfaceand the TMP.

Immobility in hot places could explain the two combined reasons for venous insufficiency in some Professions, especially cooks.

The low atmospheric pressure decreases the extra venous pressure EVP and consequently increases the TMP. This is the case for those who live at high altitude.

In airplanes, the low atmospheric pressure combined with the immobile sitting position increases the TMP by reducing the extra-venous pressure and increasing the intra-venous pressure.

These "physiological" insufficiencies create the conditions for valvular incompetence probably by inflammatory destruction of the valves but also thrombosis due to excess venous stasis (Virchow's triad).

6517-*Ulcer*

Wound that does not heal in the usual period of time, the ulcer can know several causes opposed to the healing (Arterial, venous, capillary, infectious, paraneoplastic, neoplastic).

The start point of the venous ulcer is usually located at the ankle, where the drainage conditions are the most precarious, opposite the re-entry perforators. *It then extends mainly due to superinfection. Note that these re-entry perforators are NOT the cause of the ulcer, but only the point of excessive pressure/flow of the closed shunts. They destruction ablate not only the cause but also the drainage pathway.* ***Therefore, the disconnection of the responsible shunt ablates the cause and preserves the drainage****, which ensures a good and lasting healing of the ulcer****. An ulcer that begins higher or lower than the ankle is usually NOT venous.***

It is usually limited by irregular borders, with bleeding, fibrino-cruciate background, and important dermo-hypodermitis environment.

Discussions and controversies about the physiology of venous ulcers are meaningless when it is understood that all venous ulcers occur only if the transmural pressure TMP is excessive and heal if the TMP is normalized by hemodynamic treatments and this more rapidly as the infectious complication is effectively treated. The inflammation is not due to the direction of the flow (reflux) but its pressure volume overload. . Ref. ZAMBONI P. et Al.:Oscillatory flow suppression improves inflammation in chronic venous disease. journal of surgical research _ september 2016 (205) 238-245

652-*Acute venous insufficiency*

Acute venous insufficiency is mainly due to rapid venous obstruction.

6521-Sudden swelling of the extremities

When it is due to acute venous insufficiency, it is related to a major drainage disorder.

The most severe is represented by phlegmatia cerulea*, in which a massive thrombosis, in the absence of collateral pathways, stops venous flow and therefore also arterial flow****, which induces acute ischemia.***

This sudden swelling should ***not be confused with a Baker's cyst rupture, a hematoma, or an inflammation such as erysipelas.***

6522-Painful swelling of the foot related to a non-displaced fracture

is often due to an undiagnosed thrombosis of the plantar veins, which I described in 1997 thanks to the echodoppler, because classic phlebography could not show

them. ***Ref:*** FRANCESCHI, C., Thrombo-phlebitis of plantar veins. Actualités Vasculaires Internationale, N. 47 -January 1997, p. 29

6523-Acute venous insufficiency in pregnant women.

The supine position can trigger a deactivation of the cardiac pump due to a lack of reservoir effect caused by compression of the inferior vena cava by the pregnant uterus.

66-Differential diagnosis.

Elimination and/or recognition of non-venous causes helps to avoid errors in differential diagnosis.

Pain and heaviness not relieved by decubitus or compressive stockings are not a necessarily related to venous insufficiency, even if the patient has varicose veins or oedema. Cramps and tingling that occur while lying down are not due to venous insufficiency.

661-Sudden oedema.

Non-venous causes of sudden painful swelling of the leg are:

-Erysipelas, which is accompanied by heat and redness

-Haemorrhagic detachment of the medial gastrocnemius muscle from the soleus muscle, which has been inaugurated by a sensation of the classic "whiplash" formerly attributed to a venous rupture that was never seen in echodoppler!

-Rupture of a Baker's cyst (internal popliteal synovial cyst in relation to the knee joint).

-Angioedema is not usually localized to the lower extremities

662- Chronic oedema.

6621-Bilateral white oedema

Soft, depressible under the finger, bilateral white oedema may be due to venous insufficiency (right heart failure, valvular incompetence or bilateral venous caval or truncal obstruction).

They may also be due to decreased oncotic pressure (hypoproteinaemia): cirrhosis, malnutrition (kwashiorkor), nephrotic syndrome, acute glomerulonephritis (which associates hypoproteinaemia and hyper reabsorption of water), iatrogenic (corticosteroids, nonsteroidal anti-inflammatory drugs due to water retention, calcium channel blockers due to decreased arteriolar-capillary resistance, which increases the residual venous pressure)

6622-Unilateral oedema is related to a unilateral cause,

This unilateral cause may be venous, lymphatic or rheumatological inflammatory, acute or chronic.

They are often ***wrongly attributed to varicose veins*** *and venous insufficiency* ***in elderly subjects.***

The discovery of a Baker's cyst on echodoppler suggests inflammation of the knee.

663- *Dermo-hypodermitis*

Dermo-hypodermitis is caused by excessive Transmural pressure (TMP) which reduces the drainage of the skin.

It is a chronic inflammation of the skin and the subcutaneous layer, which is located at the ankle level. It is red or dark, hard, painful to the touch. *It can develop into an ulcer spontaneously or after a small trauma. Malleolar erythematous-squamous patches are called "varicose eczema" when they are pruritic. The* ***ochre dermatitis, red at the beginning, becomes brown due to hemosiderin deposits****. The white atrophy of ivory colour is a sclerotic evolution with obstruction of the dermal capillaries****.***

Acute, subacute, red, and painful hypodermitis of the leg progressively enlarges the leg and can evolve towards hypo dermatosclerosis, more or less pigmented, to form a real retractile gaiter.

Venous dermo-hypodermitis must be differentiated *from the many other forms and aetiologies of dermo-hypodermal damage that may more or less simulate venous insufficiency, but which are the responsibility of dermatology and internal medicine (Infection, Erythema nodosum, subacute nodular hypodermitis migrans, periarteritis nodosa, allergic vasculitis, granulomatous vasculitis, lupus, and many other diseases whose diagnosis requires a biological and anatomopathological workup)*

664-Nonvenous ulcer.

*A wound that does not heal within the usual time frame****, the ulcer may have several causes that prevent it from healing (Arterial, venous, capillary, infectious, paraneoplastic, neoplastic)***

6641-Arterial ulcers

Arterial ulcers are due to ischemic necrosis called "gangrene" which most often affect the foot (heel and/or toes).

The so-called "arteriovenous" ulcers located on the ankle are in fact venous ulcers in a subject who is also suffers arteriopathy of the lower extremities.

The only advantage of recognizing arteriopathy is to take it into account when treating with venous compression and to avoid any treatment that destroys the venous capital which, even if it is incompetent, can save the limb in case of worsening ischemia. This is

especially true since conservative treatments exist (compression and CHIVA). ***Ref****: 1-No touch technique of saphenous vein harvesting: Is great graft patency rate provided? Papakonstantinou NA J Thorac Cardiovasc Surg. 2015 Oct;150(4):880-8. doi: 10.1016/j.jtcvs.2015.07.027. Epub 2015 Jul 15. 2-The no-touch saphenous vein for coronary artery by-pass grafting maintains a patency, after 16 years, comparable to the left internal thoracic artery: A randomized trial.Samano N1, :ClinicalTrials.gov NCT01686100.Copyright © 2015 The American Association for Thoracic Surgery. Published by Elsevier Inc. All rights reserved. 3-Meta-analysis of infrapopliteal angioplasty for chronic critical limb ischemia Marcello Romiti, (J Vasc Surg 2008;47:975-81.) 4-Meta-analysis of popliteal-to-distal vein by-pass grafts for critical ischemia Maximiano Albersand (J Vasc Surg 2006;43:498-503.)D- The CHIVA method gives less recurrence than destructive methods (Stripping, Laser, radiofrequency, sclerosis, foam). 5-CHIVA method for the treatment of chronic venous insufficiency. Bellmunt-Montoya S1, Cochrane Database Syst Rev. 2015 Jun 29;(6):CD009648. doi: 10.1002/14651858.CD009648.pub3*

6642-Necrotic angiodermatitis (Martorell's ulcer)

Necrotic angiodermatitis is more often seen in diabetic and/or hypertensive subjects*. It has a sudden onset, is very painful, and is due to arteriocapillary obstruction located in the leg but often higher than venous ulcers.*

6643-Carcinomas *can ulcerate and venous ulcers degenerate.*

6644-Ulcers from infectious, degenerative, haematological diseases

These are haematological, infectious, metabolic or neurological diseases.

Pyoderma gangrenosum with a budding base and hypertrophic edges can reveal a carcinoma, -Infection (mycobacteriosis, Buruli ulcer (mycobacteriosis), tuberculosis, deep mycosis, parasitosis),

-Haematological disease (myeloproliferative syndromes, cryoglobulinemia, hypergammaglobulinemia, congenital haemolytic anaemia, sickle cell disease, Minkowski-Chauffard syndrome, thalassemia)

- Crohn's disease or ulcerative colitis

-Kaposi's disease.

665- Non-venous pain

The presence of varicose veins and/or varicosities ***too often leads to attributing the cause of the pain to the varicose veins and to proposing phlebological treatments*** *that are not followed by analgesic effects.*

It is often rheumatological and/or neurological pain.

A "therapeutic" test should always be performed*. It consists in having the patient wear an effective support against venous insufficiency. If it does not significantly reduce pain and heaviness, any treatment of venous insufficiency will be disappointing because it is ineffective on these symptoms.*

67- Clinical manoeuvres

671- The persistence of varicose veins visible in the supine position with the legs raised suggests venous obstruction or arteriovenous fistula. The latter is recognized by the presence of an arterial murmur with the stethoscope.

Palpation is used to identify painful points and their possible relationship to a vein, particularly in cases of superficial venous thrombosis.

672-Painful Homans' manoeuvre (Passive dorsiflexion of the foot)

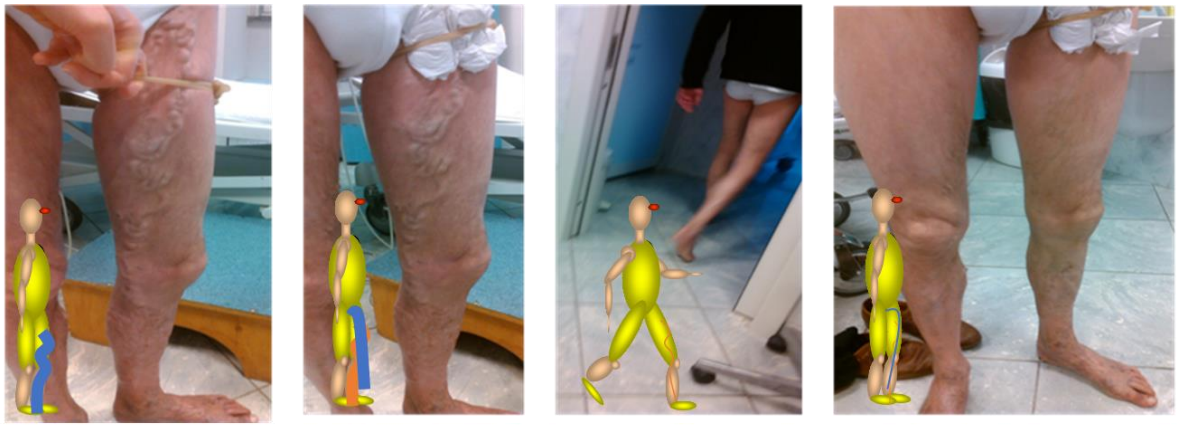
indicates recent thrombosis of the calf veins. It is not always positive in cases of phlebitis and can be misleading in cases of non-venous muscle damage.

673- The Perthes test (tight tourniquet of the thigh) allows differentiation between:

-varicose veins due to superficial venous incompetence which collapse when walking, and

-varicose veins which do not collapse when associated or caused by a deep venous obstacle or incompetence.

- Predicts the results of CHIVA tretment



The Perthes test is clinical and hemodynamic. The degree of collapsing of the varicose vein is proportional to the quality of the re-entry and allows visualization of the result of a disconnection of the escape point of the shunt responsible by both the physician and the patient,

Chapter 7

Each chapter includes some of the elements of the previous chapters and anticipates those of the following chapters.

. 7 -Instrumental diagnosis of venous insufficiency

71- Invasive methods

711-Phlebography

712-Catheterization pressure measurement

713-Endovenous ultrasound

72-Noninvasive methods

721-MRI angiography

722-Air plethysmography (APG)

723-Strain gauge Plethysmography (SPG)

724- Infrared plethysmography (IRP)

725- Hemodynamic and topographic Doppler.

7251- Device configuration

72511-Probes and frequencies

72512-Dynamics and contrast

72513-Doppler

725131-CW continuous-wave Doppler CW

725132-Pulsed Doppler

725133- Color Doppler

725134-Power Doppler

725135-B Flow

725136 In practice

73-Dynamic manoeuvres are the key to diagnosis and therapeutics

731-Compression-relaxation

732-The Paranà manoeuvre

733-The Valsalva manoeuvre

7331-Method of the Valsalva manoeuvre

7332- Interpretation of the effects of Valsalva +.

7333- Interpretation of the effects of Valsalva +.

7334- Interpretation of the diastolic effects of valvular pumps and the Valsalva manoeuvre.

7335- Interpretation of flows of descending tributaries of the great saphenous vein arch and pelvic leaks.

7336- Interpretation of systolic flow of the valvulo-muscular pump.

7337- Shunt I+II vs SHUNT III differentiation test.

7338- Valsalva and Shunt I+II vs SHUNT III differentiation test.

7339-Perforators

734- The venous tourniquet. Perthes test.

735- Doppler measurement of TMP venous pressure.

736- Positions for echo-Doppler examination.

7361-Diagnosis of pelvic occlusions and incompetence.

73611- Reclining and semi-recumbent position.

736111-Diagnosis of May Thurner (or Cockett) MTS and pseudo-MTS and Nutcracker Syndrome NTS

7361112- Indirect diagnosis of iliac and cava obstruction and incompetence:

73612--Position lying on right side, horizontally.

73613--Gynaecologic position

73614--Position standing, with one leg elevated

7362 Diagnosis of iliofemoral and leg venous occlusions and incompetence

73621-Standing position

73622-Sitting position

73623- Recumbent position

737-Echo-Doppler ultrasound examination: hemodynamic signs

7371-Supine and semi-supine examination

73711--Venous compression tests

73712--Femoral venous flow modulated by respiration

73713-- Reflux in the common femoral vein during Valsalva manoeuvre,

7372--Sitting on the edge of the examination bed

73721--Probe compression testing of the veins of the sole of the foot of the calf.

73722--Flow and reflux of the tibial, fibular, soleus and gastrocnemius veins.

7373-The foot examination:

73731-Venaopliteal:

737311-Venaopliteal and gastrocnemius.

737312-Large and small saphenous veins.

737313-Check for the presence of a popliteal cyst which can be a cause of pain and oedema.

73732- Groin

737321-Systolic and diastolic flow and reflux.

737322-Pelvic visceral escape point reflux

737323-Great saphenous vein GSV

7373231-Normal hemodynamics of the Great saphenous vein GSV

3732311-The Paranà manoeuvre activates the calf and sole of foot pumps (Léjars pump)

73732312-Manual calf compression

73732313-N3 veins tributary to the great saphenous vein N2.

7373232-Hemodynamic of the great saphenous trunk

7373233- RP re-entrant perforators of the great saphenous vein

7373234--Systolic reflux of Paranà N1>N2 at the Femoral saphenofemoral junction.

7373235--Systolic Paranà and reflux N1>N2 at the saphenopopliteal junction SPJ.

73733236--Tibio-safenous junction

737323237-Pulsed saphenous outflow

73732371-Pulsatile retrograde flow due to tricuspid heart valve reflux.

73732372-Anterograde pulsatile flow due to decreased arteriolar-capillary resistance: inflammation of leg tissues.

73732373-Anterograde pulsatile flow due to resistance to flow:

737324-Small saphenous vein (formerly known as short saphenous vein).

7373241-Small saphenous vein anatomy.

7373242-The hemodynamic function of the lesser saphenous vein is particular.

737325-Giacomini's vein.

7373251-Anatomy of Giacomini's vein.

7373252-Hemodynamic function of the vein of Giacomini's vein

7374-Deep veins of the lower extremities

73741-Examination in the patient lying semi-sitting:

73742-The examination in the seated patient, with the legs hanging off the examination bed.

73743-Examination in the standing patient

7375-Venous malformations.

7376-Post-treatment controls

7377-Topographic and hemodynamic mapping.

7378- Marking the approach points

7379-Ecodoppler by pathology

73791-Deep vein occlusions

737911-Nutcraker's syndrome NTS or aorto-mesenteric clamp:

737912-Iliac and/or cava vein occlusion.

737913-May Thurner Syndrome MTS

737914--Portal vein occlusion

737915--Common femoral vein occlusion

737916Superficial Femoral occlusion

737917--Popliteal vein occlusion

737918--Tibial, soleus, gastrocnemius occlusion

73792-Deep venous incompetence

73793-surfacevenous occlusions

73794- Cartography

737941- Superficial veins mapping

737942- Deep veins mapping

737943- Mapping of venous malformations

7 -*Instrumental diagnosis of venous insufficiency*

The diagnosis sought and retained varies according to the pathophysiological model of the disease.

The same image, the same figure, the same measurement obtained by the same instruments in the same patient are interpreted differently according to the different explanatory theories of the same disease.

This also explains the different, even opposing, therapeutic approaches.

This is the case for venous insufficiency, depending on whether one considers that:

-Varicose veins are the cause or the consequence of reflux,

-All reflux is pathogenic or not

-Calibre of the veins (ectasia and morphological stenosis) is the decisive criterion or not of its pathology,

-Veno-venous shunts are the central physiopathological phenomenon of the so-called "essential" varicose veins and most of other aetiologies.

This is also the reason for different instrumental investigation methods and protocols, according to the consideration and knowledge of hemodynamic aspects over morphological aspects. The venous disease classification CEAP is an example of prevailing "morphology" on "hemodynamic".

Yet, the rational and efficiency of the treatment cannot be symptomatic, based on clinical severity but on its cause, thus on the damages of the venous system that impairs the hemodynamic condition. The rational treatment of haemorrhage is not defined by transfusion but the stop of the bleeding.

Whatever their clinical aspects, they relate to an excess of transmural pressure TMP and the treatment should consist of the reducing it. This hemodynamic prevalence is proved by clinical evidence. For example, changes of venous pressure, as lower extremities elevation collapses "miraculously" the varicose veins and heals venous ulcers if maintained long enough Ref: C Franceschi - Venous hemodynamics, knowledge, and miracles. Journal of Theoretical and Applied Vascular Research (page 39) - JTAVR 2019;4(2)***.***

To achieve this goal, Trendelenburg maintained this collapse when standing up by blocking the reflux at the groin with his finger, which led him to ligate the saphenous veins in order to maintain this stop, and successfully treat the venous ulcers so far treated by weeks long recumbent stays. The effect of the reduction of one of the two parameters of the TMP, i.e., the intravenous pressure excess was proved.

The second parameter is the explicated by positive effect of the increase of the extravenous pressure, as experimented and proved by Brody 150 years ago, discovering the "rubber bands.

Today, technology has improved the means to understand and assess much better the cause for the TMP excess and to elect its best treatment. Despite these evidences, it is still more morphology oriented than hemodynamic.

This different diagnosis approach leads to different therapeutic strategies: destructive versus conservative.

The hemodynamic approach looks for the hemodynamic cause of venous insufficiency (dynamic non-fractionation of the gravitational hydrostatic pressure FDPHG, open vicarious shunts OVS, open deviated shunt ODS, closed shunts CS.

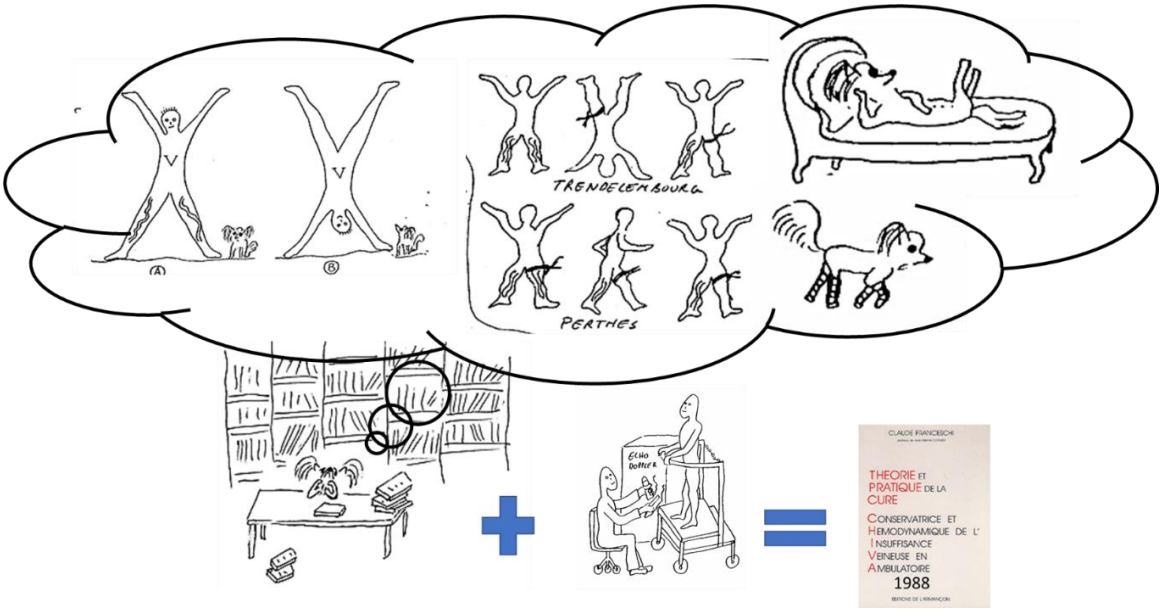
The Echo-Doppler is the central instrument, almost always sufficient, if it is used and interpreted according to the criteria of the hemodynamic approach

It alone allows the study of flows and their pathological value according to their location and their variations according to the various dynamic tests (Paranà, Valsalva) which mimic the physiological behaviour of the heart thoraco-abdominal and valvulo-muscular pumps.

Moreover, it is <u>not invasive</u> and can be repeated as many times as necessary.

The price of these advantages is the <u>necessary expertise both in venous hemodynamic and in the practitioner practice of the device</u>

This method will be discussed in detail after reviewing and criticizing the various other methods.



71- *Invasive Methods*

711-Phlebography

<u>Phlebography cannot evaluate the hemodynamic significance</u> by the calibre of the stenosis and the importance of the collaterals alone. <u>It confirms the severity of a stenosis or occlusion by the collaterals (OVS) but cannot evaluate the loss of load or the degree of hemodynamic compensation.</u>

The patient being <u>immobile in dorsal decubitus</u> position, it can show the reflux only under Valsalva (not Paranà). In addition, <u>Valsalva</u> in this position <u>can be positive in absence of true valve incompetence</u>, for the reason I explained in chapter 5.

It <u>may fail to recognize</u> the <u>occlusion</u> of a venous collateral, <u>one of the two branches of a double superficial femoral</u> and the thrombosis of the plantar veins as I was able to describe it thanks to the echodoppler.

It can also show artefactual stenoses and occlusions which are only postural, i.e. caused only by the supine position, as in the pseudo MTS demonstrated by postural echodoppler:

complete occlusion of the left iliac vein in the supine position, "cured" by the semi-sitting position.

Ref: ***Ref:1-*** Paolo Zamboni, Claude Franceschi, Roberto Del Frate.The overtreatment of illusoryMay Thurner syndrome Veins and Lymphatics 2019; volume 8:8020. 2-VIDEO: Pseudo MTS : https://www.youtube.com/watch?v=h931XXo2hdk&t=23s

3-***V***an Vuuren TM, Kurstjens RLM,Wittens CHA, et al. lllusory angiographic signs of significant lliac vein compression in healthy volunteers. Eur.J Vasc Endovasc Surg 2018;56:874

One can imagine how many patients have been mistakenly treated with these pseudo-MTS

712-Catheter pressure measurements.

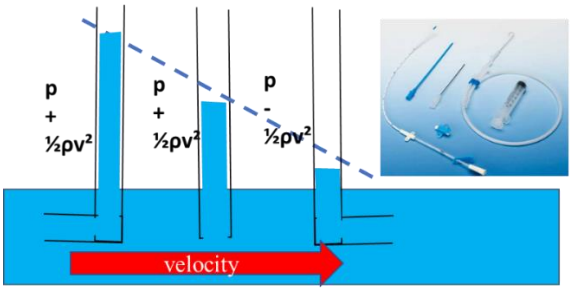
These pressure measurements have been the spearhead of research *since the second half of the twentieth century, because they have already shown the differences in venous pressure according to pathologies by valvular incompetence and/or obstruction.*

They are consistent with the hemodynamic concepts of dynamic fractioning of the gravitational hydrostatic pressure DFGHSP.

However, they ***do not consider the veno-venous shunt effects*** *suspected by Trendelenburg and highlighted by the Doppler effect and the results of the CHIVA cure.*

Its interpretation must consider *the orientation of the pressure sensor with respect to the direction of venous flow (see Pitot tubes).*

Today, measurement of posterior tibial venous pressure at the ankle by Doppler allows this invasive method to be avoided and reserved for pre- and post-procedure evaluations of venous revascularizations.



Venous pressure with the catheter
Total pressure Pt = p + ½ρv² + ρgh
When the catheter faces the flow, it measures the Total Pressure p + ½ρv²
When it is perpendicular to the flow, it measures the only lateral static pressure p.
When it is in the direction of the flow, the pressure is equal to p- ½ρv²

713-Endovenous ultrasound

Endovenous ultrasound ***does not provide hemodynamic assessment****. Yet it measures the calibres and shows endovascular anomalies, such as MTS synechiae, but it doesn't assess the flow but.* ***Moreover****, the measurement of calibres* ***depends too much on the posture*** *of the*

patient (for example the Pseudo MTS of decubitus) to be considered in the hemodynamic diagnosis and the resulting therapeutic indication.

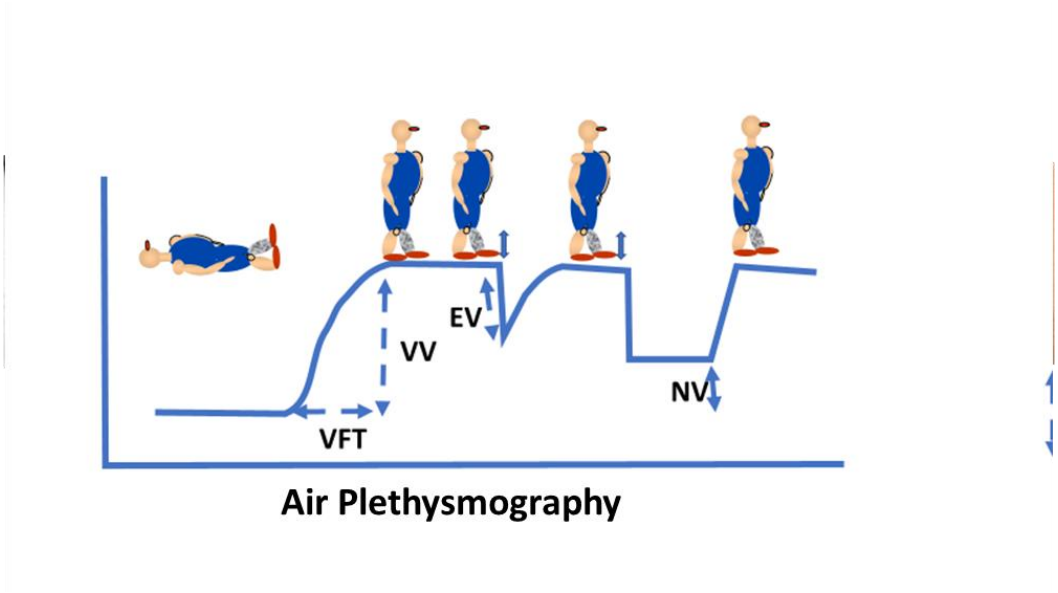
72-Non-invasive methods

721-MRI angiography

***Venous MRI angiography shows the veins but not sufficiently the hemodynamic** despite the possibility of measuring flow rates, but only at rest and in the supine position. It is especially useful for evaluating the location and extension of venous malformations before and after treatment. However, pre and perioperative echo doppler is more accurate in marking their location, particularly in the extremities (Chapter 8 VM treatment))*

722-Air plethysmography (APG).

This is a global hemodynamic method which quantifies the variations in volume of the leg according to normal or pathological hemodynamic conditions depending on the posture and the activity of the valvulo- muscular pump.



***To assess reflux,** an air-filled cuff, connected to a pressure/volume meter, is placed around the leg. It measures the passive filling time after moving from the supine position with the leg raised to the standing position with the leg resting on the opposite side. The 90% filling time is a volume flow index (VFI). Then, a single contraction of the calf to raise the heel reduces the volume by the value of the ejected volume (EV). Then, the same movement is repeated 10 times, which related to the Previous filling volume VV measures the ejection fraction EF = EV/VV. 100.*

The residual volume obtained (Residual Volume RV) *reported to the resting volume VV, is VV, is the fraction of residual volume RVF = RV/VV.). Logically, and in accordance with the hemodynamic concepts that we have explained previously, as well as with the data from invasive pressure measurements, VFI decreases and RVF increases in proportion to the degree of valvulo-muscular incompetence, that is, to the defect in dynamic fractionation of the gravitational hydrostatic pressure DFGHSP that we have defined and explained previously.*

To assess obstructions*, the patient remains lying down. An inflatable cuff with pressure gauge, is added to the thigh and then inflated to 70 mmHg. When the leg volume reaches a plateau, the cuff is quickly deflated. The volume evacuated during the first second is related to the Previous volume and measures the outflow fraction (OF). OF decreases in proportion to the hemodynamic value (resistance) of the obstruction.*

These global measurements are rivalled by echodoppler, which allows precise individualization of refluxed and occluded veins, as well as direct measurement of posterior tibial venous pressure at the ankle in mmHg. *They can be useful for more objective pre- and post-treatment evaluation and are less treatment dependent.*

In conclusion, this method does not allow the evaluation of the topography and/or type of veno-venous shunts which are of primary importance for CHIVA hemodynamic therapeutic strategy.

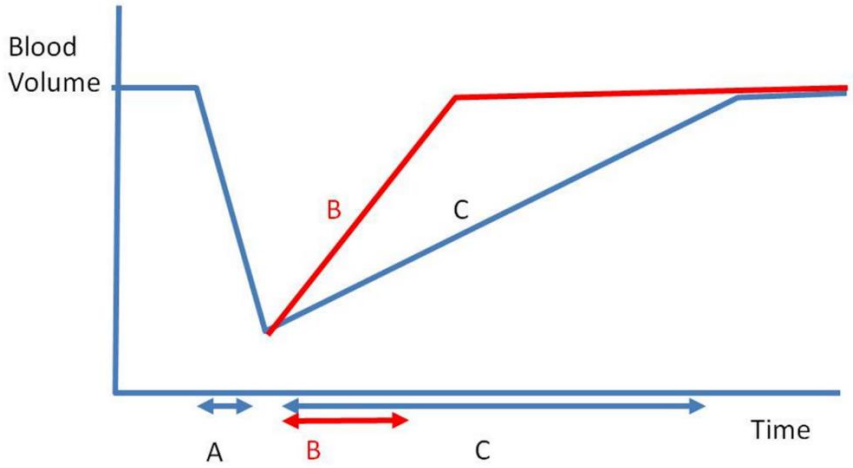
Nevertheless, It can be used for studies of the overall hemodynamic results of various treatments.

723-Strain gauge plethysmography (SPG)

SPG is equivalent to APG, *from which it differs by the measuring equipment. Indeed, SPG uses the electronic measurement of the elongation of a wire in a particular placed on leg, of which it measures the circumference variations instead of the volume measured by APG.*

724- *Infrared plethysmography (IRP)*

Ref: Claude Franceschi. Who knows the rationale of the refilling time measured by plethysmography? Veins and Lymphatics 2018; volume 7:7199



The Plethysmograph emits infrared radiation and receives back those that are not absorbed by haemoglobin. This allows the measurement of blood volume variations under the probe of the device*. The small synchronous variations of the pulse correspond to variations of blood volume brought by the arteries. The non-synchronous variations, which are much larger, represent the venous blood volume****.***

In phlebology, the probe is placed on the skin at the level of the middle 1/3 - lower 1/3 of the internal face of the *leg in a seated subject. The patient is asked to perform several flexions-extensions of the foot to empty this area of the maximum amount of venous blood by muscular pumping. Then the complete TR filling time is measured after pumping has stopped. Classically, we deduce that the shorter the time, the greater the reflux, i.e. the faster the zone explored is filled.*

<u>In fact, the filling time TR can be shortened by two combined effects</u>.

*The area incompletely emptied by pumping due to valvular incompetence, fills the faster the more flow or reflux volume remains available at the end of pumping. The normal RT after several dorsiflexions, flexed foot extensions is greater than 20 seconds. This means that filling is "slow" because the filling rate is low regardless of the direction of flow. This explains why the destruction of superficial veins reduces this flow rate, especially when it is radical (longer time after stripping vs. crossectomy (Cestmir Recek)). Indeed, stripping or any other destructive superficial endovenous technique alters skin drainage, which is responsible for reactive neo-angiogenesis, matting, telangiectasias and varicose recurrence****.***

<u>A "good plethysmographic result" (TR> 20 seconds) is therefore not necessarily a good functional result.</u> CHIVA treatment (fractioning of the column and disconnection of the shunts), removes the reflux overload without removing its physiological drainage part although the flow remains reversed. *The filling time is lengthened, but less than after stripping or endovenous ablative procedures, because it is more functional. On the other hand, a time of less than 20 seconds, reflects pump failure and/or overload by CS closed shunt reflux or ODS by-pass.*

<u>Thus, the TR must be revisited in its functional meaning</u>. Moreover, the concept "reflux = pathology" is contradicted by the normalized TR after the CHIVA cure. In fact, the CHIVA cure eliminates hemodynamic overloads and re-establishes the physiological drainage hierarchy despite the direction of the flow which remains reversed. Moreover, it is not the inversion of the direction of the flow that is responsible for the inflammation but only its flow/pressure. ***Ref:*** Paolo Zamboni, MD and al. Oscillatory flow suppression improves inflammation in chronic venous disease. journal of surgical research _ s e p t e m b e r s e p t e m b e r 2 0 1 6 (2 0 5) 2 3 8e2 4 5.

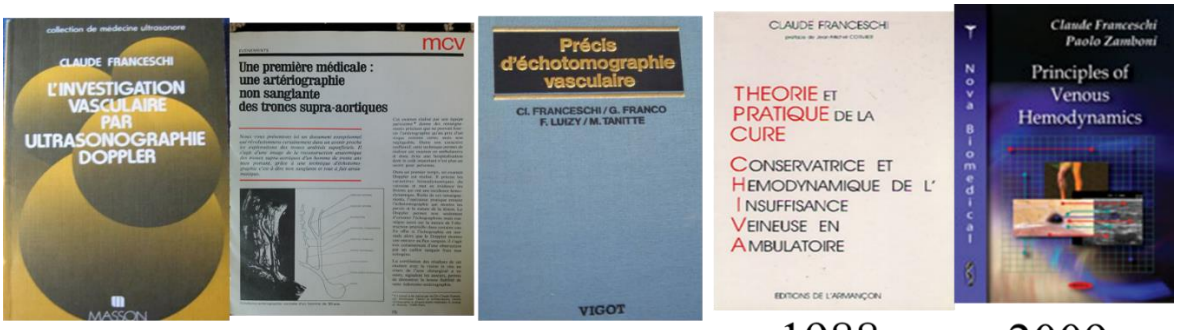
725- *Hemodynamic and topographic Doppler.*

<u>I emphasize "hemodynamic and topographic" because this method is practiced and interpreted too differently by individuals, schools, and countries.</u>

I was fortunate to be a pioneer in echodoppler technology and application, which gave me a better understanding of arterial and venous hemodynamic.

Unfortunately, the ***<u>diagnostic treasures of this method are still hidden to many vascular specialists and their teams</u>****.*

Once again, a good knowledge of venous hemodynamic and pathophysiology is required to perform a quality Doppler ultrasound and a good knowledge of the subtleties of the Doppler ultrasound is required to understand its vascular application possibilities.



1977 **First Doppler semiology**

1981 **First carotide Echography**

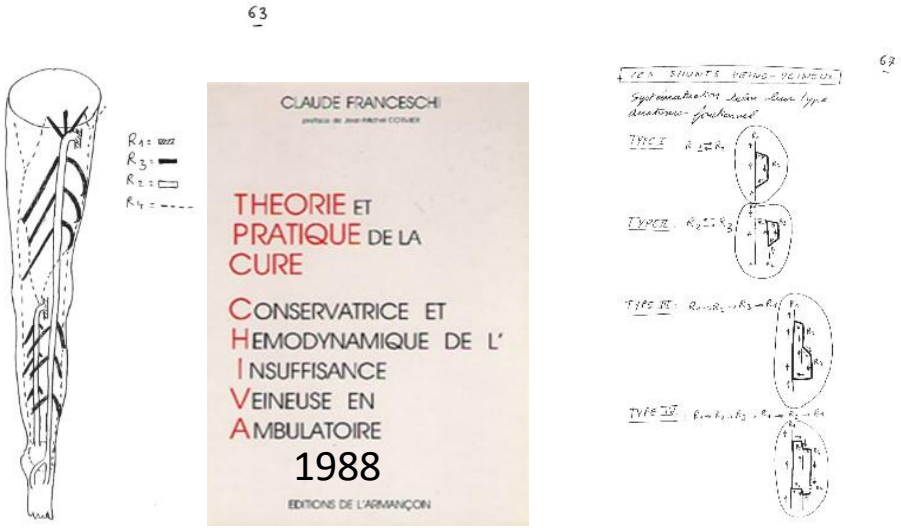
1986 **First Vascular Echo Imaging**

1988

2009

The anatomy of the venous system, particularly the superficial system, is highly variable and does not prejudge its pathology.

<u>The hemodynamic classification into N1, N2, N3 networks, open vicarious shunts OVS, open deviated shunts ODS and closed shunts CS, define the veins by their pathophysiological function</u>**, regardless of their anatomy.**



Réseaux Shunts

.

We can say that the <u>veins are not always where we look for them</u>, but they are always where we found them thanks to the echodoppler.

This allowed me to focus on the hemodynamic anomalies and the search for their causes (escape points, routes, and re-entries). This is how I found the pelvic escape points. It was by ultrasound tracing the descending flow (normal directions) but Valsalva + (pathological) of the tributaries of the saphenofemoral junction that I was able to define these escape points anatomically and functionally. ***Ref:*** 1. Franceschi C, Bahnini A. Points de fuite pelviens viscéraux et varices des membres inférieurs. Phlébologie 2004;57:37-42.2. Franceschi C, Bahnini A. Treatment of lower extremity venous insufficiency due to pelvic escape points in women. Ann Vasc Surg 2005;19:284-8. 3. Franceschi C. Anatomie fonctionnelle et diagnostic des points de fuite bulboclitoridiens chez la femme (point C). J Mal Vasc 2008;33:42.

Anatomists were inspired by the classification of veins (hemodynamic) into anatomical-functional networks that I proposed in 1988. ***Ref***: Théorie et pratique de la cure conservatrice et hémodynamique de l'insuffisance veineuse en ambulatoire [CHIVA] Editions de l' Armancon 1988 ISBN-10: 2906594067 ISBN-13: 978-2906594067.***They confirmed on the cadaver what was evident in ultrasound and translated R1, R2, R3 networks (R for reseaux in French) into English N1, N2, N3 for networks.***

The echodoppler allowed me to follow the normal and abnormal flows according to the activity of the cardiac, thoraco-abdominal (Valsalva) and valvulo-muscular pumps (initially by compression-relaxation which I replaced by the more physiological Paranà manoeuvre. I named this last one after the city in Argentina where I Presented it for the first time. **Ref:**1. Franceschi C. Mesures et interprétation des flux veineux lors des manœuvres de stimulation. Compressions manuelles et manœuvre de Paranà`. Indice dynamique de reflux (IDR) et indice de Psatakis. J Mal Vasc 1997;22:91–5. 2 Ermini, F Passariello, M Cappelli, C Franceschi - Experimental validation of the Paraná manoeuvre compared to the squeezing test Journal of Theoretical and Applied Vascular Research (PAge 97) - JTAVR 2017;2(2):97-105.

The need to examine the functioning of the venous system ***not only in the supine position, but especially in the standing position,*** *became obvious to me when I saw clinically how the calibre of the veins and varicose veins changed radically according to the position (already described at the end of the 19th century by Trendelenburg) and the walk (Perthes, assistant to Trendelenburg).*

These simple data make it clear that venous disease, particularly varicose veins, is ***above all a disease of posture which worsens in the standing position and especially when walking.***

The behaviour of the flows, origin, path, destination according to the positions and activity of the pumps, led me to understand *that the varicose veins and other signs and symptoms were not the cause but the result of a hemodynamic disorder due to valvular incompetence and/or resistance to the flows (venous obstacles, cardiac or thoraco-abdominal failure****).***

The effect of this disorder is an excess of venous transmural pressure *which dilates the veins and opposes tissue drainage (trophic disorders, ulcers).*

Specific dynamic manoeuvres performed in a rigorous manner under echodoppler *control are essential for the diagnosis of the physiopathological configuration of venous insufficiency specific to each patient.*

They allow the normal and pathological hemodynamic effects of thoracoabdominal (Valsalva) and valvulomuscular (compression-relaxation and especially Paranà) pumps to be evaluated by Doppler. *They evaluate the degree of deep and superficial valvular incompetence and the type of veno-venous shunt that they activate. They are completed as much as necessary by the Perthes test and the Doppler measurement of venous pressure at the ankle.*

Ultrasound imaging locates the veins and their environment.

The combination of Doppler flow and ultrasound imaging results in a topographic and hemodynamic map specific to each patient.

This mapping is the essential document for diagnosis and therapeutic strategy.

Its realization requires a ***thorough knowledge of hemodynamic physiopathology, anatomy, and the technology of echodoppler equipment. It must provide all the hemodynamic elements (types of shunts)*** *useful for the therapeutic strategy, but also anatomo-topographic elements useful for the therapeutic technique (approach and modes of disconnection according to the anatomical characteristics of the escape points to be disconnected).*

7251- *Device settings*

The current echodopplers (Doppler Ultrasound Scanner DUS) are all of sufficient quality.

Not all pre-setting's from different manufacturers are suitable for venous exploration as they should be for adequate exploration of the venous system.

They are often filtered and pre-processed to obtain a beautiful image to the detriment of the information necessary for diagnosis.

Therefore, it is necessary to carry out its own pre-processing, using as little filtering as possible.

It is not the most "beautiful" image that makes the best diagnosis, but it is the truest, even if it is "ugly". *There is an anthropological hunting reflex among doctors, as among other human beings, which considers the "beautiful diagnosis" as a "beautiful, slaughtered animal", even if it means "arranging" it so that it appears even more beautiful though less true.*

72511-Probes and frequencies

The mechanical rotating probes at low frequency (1 to 2 Mhz) did not allow to see the peripheral arteries and veins. I patented a water bag (1981) which added to these probes allowed for the first time to explore them and to write the first book of vascular echotomography (1986) and the book CHIVA (1988)).

Single- and multi-frequency linear probes must adapt the frequency to the depth of the vein examined, i.e., reduce the frequency with depth.

72512-Dynamics and contrast

Veins require high contrast, i.e., a reduction in "dynamics" and gain adjustment*, until clearly differentiated walls are seen from the circulating lumen and surrounding tissue.*

72513-Doppler

The Doppler effect measures the direction and speed of blood flow

Johann Christian Doppler explained in 1842 that the change in colour that we perceive from the stars varies from red to blue, therefore depending on the wavelength, when they move away or approach us.

This principle remains true with the sound. The sound of the train is perceived more acute when it approaches and more serious when it moves away.

Echodoppler uses US Ultrasound. The Doppler signal is the measurement of the difference ΔF = F1-F2, between the frequency F1 of the transmitted US beam and the frequency F2 of the echo returned by the target.

It gives the direction of the flow which moves away when F2 is lower than F1 (F1-F2 positive) and which moves closer when F2 is higher than F1(F1-F2 negative).

F1-F2 = 0 when the angle of incidence of the beam is perpendicular to the flux (90°) and maximum when the angle is zero (0°).

$\Delta F = F1-F2=2V.F1.\ Cos\ \Theta/C$

ΔF = Frequency beat =F1-F2

F1= frequency of the US beam emitted to the blood

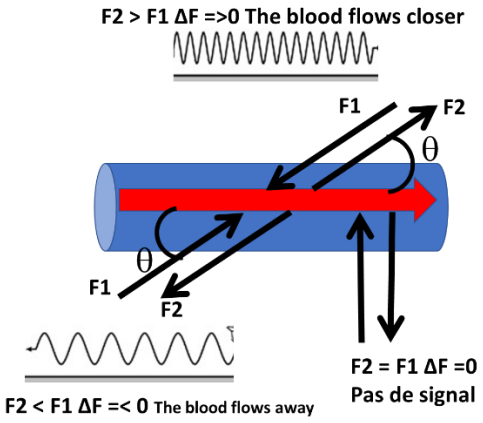
F2= frequency of the US echo beam returned by the blood to the probe

V= actual velocity of the blood

C= Sound speed

Θ = Angle of incidence formed by US beam of frequency F1 emitted by the probe and the direction of blood travel.

ΔF = 2V. F1. Cos Θ /c It is understood here that the increase in the Doppler angle is accompanied by a decrease in ΔF (the value of cos approaches zero) which is completely cancelled out when the angle reaches 90° (cos90° = 0). The calculation of the circulatory velocity therefore requires knowledge of the Doppler angle. To calculate this speed, the formula becomes: V = ΔF. c / 2F1. CosΘ



F1-F2 = 0 when the angle of incidence of the beam is perpendicular to the flow (90°) and maximum when the angle is zero (0°) .
ΔF =F1-F2=2V.F1.Cosθ /C
ΔF = frequency
=F1-F2 < 20 KHz extended by the doctor
F1= frequency of the US beam emitted to the blood
F2= frequency of the US echo beam returned by the blood to the probe
V= real speed of the blood
q = angle of incidence formed by the US beam of frequency F1 emitted by the probe and the direction of the blood movement.
ΔF = 2V. F1. Cos θ /c It is understood here that the increase in the Doppler angle is accompanied by a decrease in ΔF (the value of cosq approaches zero) which is totally cancelled out when the angle reaches 90° (cos90° = 0). The calculation of the circulatory velocity therefore requires knowledge of the Doppler angle. To calculate this speed, the formula becomes : V = ΔF. c / 2F1. Cos θ

725131-Continuous wave Doppler CW

The continuous Doppler emits through a piezoelectric element and receives through another one the echo of an ultrasound beam emitted and received simultaneously.

It was implanted at its beginning (more than 50 years ago) in pencil probes, blind because they did not give an image of the tissues nor the depth of the vessels, but its ear was very fine to listen to the "Doppler beat range" of the speed of the flows. It is still used for arterial and venous pressure measurements. ***Despite its better sensitivity than the Pulsed Doppler, it is no longer present in the majority of echodoppler*** *devices except in the low frequency Phased Array imaging probes, intended for cardiology****.***

The Doppler present in the imaging probes is now only the Pulsed Doppler in its forms of speed curves and colour imaging.

725132-Pulsed Doppler

The Pulsed wave Doppler (PWD) transmits and receives by the same piezoelectric element a beam of ultrasound emitted and received by "packs" of waves.

This allows to know ***the depth of the flow, to localize it in the image and to analyze it by segments of chosen depth called "sampler volume".***

The pulse repetition frequency (PRF) limits the sensitivity *according to the depth and the speed of the blood flow. In other words, a* ***flow will be less measurable the deeper and faster it is. That limits the sensitivity and the possibilities.***

The "steering" is a virtual electronic "orientation" of the angle of emission/reception *of the beam in relation to the probe. In general, it is adjustable by the user from 0 to 45°.*

The "angle" is made of 2 sides. The side made of the angle of the mission-reception forms is automatically recognized by the machine. The second side, *formed by the direction of the flow* ***is not automatically recognized. It must be marked manually by the user using the machine's "angle" function****. Only then can the actual speed of the blood be calculated by the machine and displayed on the screen.*

C = average speed of Propagation of ultrasound in soft tissue, i.e. 1540 m

The measured Velocity is the actual velocity of the blood when the angle of incidence of the US beam on the flow direction = 0°. If CosΘ=1, ΔF = 2V. F1 /c

In practice, it is rare that we can give this orientation = 0 to the US beam, even with the "steering". When the angle increases, Cos Θ= goes from 1 to 0.

It is therefore necessary to correct the frequency beat ΔF by cos Θ to obtain the real speed of the flow. Therefore, the machine's computer can give the real speed only if it is given the direction of the flow by activating its "Angle" function.

*But, for technical reasons, this calculation is less reliable when approaching 90° (cos Θ = 0) because F2 is then too close to F1. Therefore it is recommended to orientate the incidence of the beam as best as possible, manually and/or by "steering" to obtain the best ΔF signal possible****.***

As the Doppler signal is better when the angle of incidence of the beam on the vein is reduced, the steering orientation is set as steep as possible on longitudinal sections. On transverse sections, the probe is tilted as much as possible in relation to the direction of flow of the vein.

The High Pass Filter *eliminates the low frequencies to "erase" the electronic noise and give a "clean" Doppler image.* ***But this prevents listening to low frequencies (low speeds) and certain turbulences that can be useful for diagnosis****. It is therefore necessary to* ***remove the high Pass filter and accept imperfect Doppler images!***

The pulsed Doppler is a succession of US F1 emitted waves. The Doppler signal is formed when the F2 echo received between two F1 emissions is different from F1, i.e., when the target moves.

The depth and position of the target is recognized by calculating the time elapsed between the F1 emission and the F2 return in relation to the speed of the US in the tissue.

The sensitivity and resolution of the signal varies according to the flow velocity, the repetition frequency of the ultrasound pulses PRF and the frequency of the emitted ultrasound.

The Pulse repetition frequency (PRF) is the frequency at which the ultrasound beam is repeated, and it is therefore necessary to leave a sufficient time (period) between two F1s for f2 to reach the probe before the next F1 is emitted. *As the period increases with the depth of the target, the PRF must be reduced accordingly. Moreover, according to Shannon's law, the PRF must be at least twice the frequency of the Doppler signal. If this law is not respected, we observe an "aliasing" with folding of the spectrum.*

It is therefore necessary to increase the PRF with the frequency of F1, which limits the possibility of measuring very high speeds, especially as the vessel is deep. *Shannon's law explains the "stroboscopic" effect which, in the cinema, shows the wheels of the carriages turning backwards from the direction of travel, when the image frequency is too low compared to the speed of rotation of the wheels.*

The speed is measurable point by point along the beam (multigate) *with an acquisition of a determined number of points variables according to the machines, of the order of 1 to 15 mm that we call* ***"sample volume".***

Contrary to what is often taught, *the sample volume must cover the maximum calibre of the vein, or even exceed it.*

725133- *Colour Doppler*

Colour Doppler is a colour (coding) marking of all moving points detected by pulsed Doppler in a selected area called "colour box".

The colours signal the speed and direction of the flows without quantifying them.

It allows to quickly locate the flows in the field of the scanning plane. It presents the advantages and limitations of pulsed Doppler.

Each flow visualized with the colour Doppler must be quantified by the Pulsed Doppler *to assess the value of a reflux, normal, partial, segmental, total or closed shunt as I explained and defined them previously.*

725134-Power Doppler

The Power Doppler is the same signal processing as the Colour Doppler, but it displays the power and not the frequency of the Doppler signal.

This does not allow to measure the direction and speed of the flow, but only the energy of the flow movement.

It allows a better filling of the vessel and a better identification of small vessels with slow flow and depends less on the angle of incidence of the beam.

725135-B Flow

B Flow is not based on the Doppler effect.

It is a processing by temporal subtraction of the energy of echoes of successive images, therefore without risk of overflow and limitations according to the speeds.

It does not quantify the speeds nor their direction.

An added Doppler coding allows to show the direction of the flow.

Patented by a manufacturer, it is not available on all machines on the market.

725136-In practice

Low PRF colour Doppler is used as a first line to detect circulating veins but does not allow the hemodynamic characteristics of these flows to be specified.

It must be followed by pulsed Doppler.

Because, only pulsed Doppler allows evaluation of physiological diastolic reflux and the different types of pathological reflux (total, partial, segmental and shunts), knowledge of which is essential for diagnosis and therapeutic strategy.

A "pathologic" Colour Doppler reflux is often physiological on the Pulse Doppler

73-Dynamic manoeuvres are the key to diagnosis and therapeutic strategy.

The manoeuvres must reproduce as closely as possible the hemodynamic conditions of the venous system according to the posture and activity of the pumps. *Manoeuvres that aim to reproduce unusual movements are no less interesting.*

They are only possible with the echodoppler.

Indeed, it is thanks to the echodoppler that I was able to better understand the fundamental hemodynamic aspects of venous physiopathology.

This also explains why these concepts are difficult to accept by those who do not personally practice echodoppler.

They indicate echodoppler performed by ultrasonographers who do not have sufficient knowledge of pathophysiology and who are constrained by doctors to simplistic protocols.

<u>The conditions are optimal when it is the practitioner-therapist who performs the echodoppler.</u>

731-Compression-relaxation

Compression-relaxation of the thighs and calves *is useful only when the patient is lying or sitting,* ***i.e., when the Paranà manoeuvre is not possible.***

732-The Parana manoeuvre

The Paraná manoeuvre is practicable in the standing patient. It is more effective and closer to physiology than the compression-Relaxation manoeuvre, i.e., to what happens during walking.

Paranà is the name of the city in Argentina where I first presented this method. **Ref:**1. Franceschi C. Mesures et interprétation des flux veineux lors des manœuvres de stimulation. Compressions manuelles et manœuvre de Paranà`. Indice dynamique de reflux (IDR) et indice de Psatakis. J Mal Vasc 1997;22:91–5.

7321- *Paranà Method*

The Paranà manoeuvre consists in giving a light impulse or attraction to the patient's waist.

It triggers a Proprioceptive reflex that contracts the muscles of the lower limb, particularly the calf muscles, in a quasi-isometric manner.

It is much more effective in terms of systolic and diastolic flow rates than compression-Relaxation

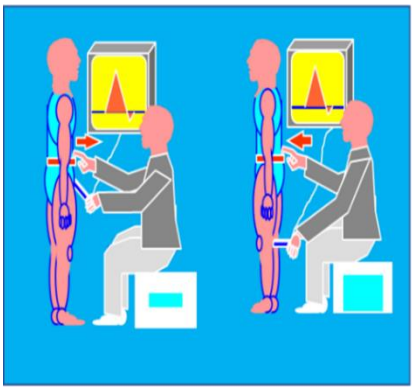
It is also more reproducible because it does not depend on the examiner's manual compression force.

It is applicable from top to bottom at all deep and superficial levels, *except for plantar and pelvic veins.*

In the competent popliteal veins, *Paranà mobilized 40% more blood volume than compression-release*

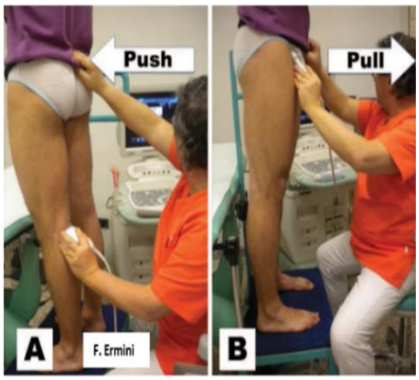
The diastolic phase of Parana compared to compression-relaxation *lasted more than 3 times longer in the saphenofemoral junction, more than 2 times longer in the incompetent GSV trunk, and more than 3 times longer in the perforating vein re-entry.*

Ref: Ermini, F Passariello, M Cappelli, C Franceschi - Experimental validation of the Paraná manoeuvre compared to the squeezing test Journal of Theoretical and Applied Vascular Research (PAge 97) - JTAVR 2017;2(2):97-105.



Paranà Maneuver

Paranà River



733- *The Valsalva manoeuvre*

The Valsalva manoeuvre can be performed in any position (Supine and standing).

It can be positive in the supine position when the valves are continuous and lead to a false diagnosis of incontinence (phlebography). Indeed, Valsalva first fills the veins because they are not very dilated in this position, before being able to close the valves. Therefore, it must be repeated in the standing position.

7331-Method of the Valsalva manoeuvre

The Valsalva manoeuvre is obtained by blocking forced expiration, which increases thoracic and abdominal pressure against the venous proximal system.

It is performed physiologically *by heavy lifting, defecation, and childbirth efforts.*

The patient may be asked to hold the breath and "push" as if they were to defecate*. Both this question and the answer can be tricky.*

The clogged straw*. I successfully use a simple method that everyone can understand****. I ask the patient to blow as if to unblock it, through a straw of which I have previously clogged one end.***

The correct execution of the manoeuvre MUST be verified.

How can this be done?

*By checking that the flow of the femoral vein resumes just after the systolic thrust stops****. This verification is particularly important when exploring the veins of the inguinal fossa.***

Its effectiveness decreases distally.

7332- Interpretation of the effects of Positive Valsalva (V+)..

It is said "positive" (Valsalva+ or V+) when it activates a flow or reflux during the systolic thrust (forced systole of the thoracoabdominal pump)

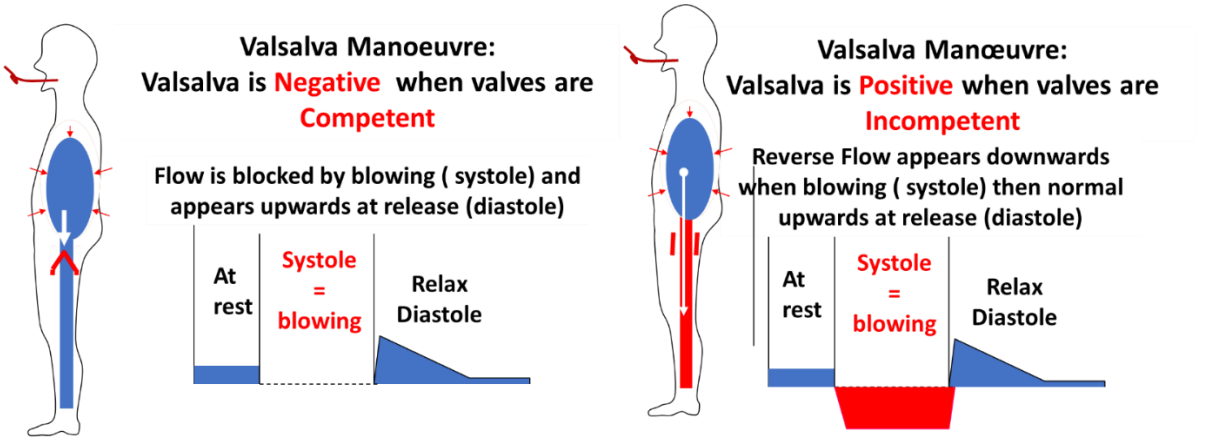
It is said to be negative (V-) when it does not activate the flows during the thrust systolic (forced systole of the thoracoabdominal pump).

<u>It reverses the pressure gradient towards the feet</u>. It first increases the pressure in the deep veins N1.

This higher pressure in N1 than in N2 or N3 leads to superficial reflux *N1>N2, N1>N3, or N1 N2>N3 in case of incompetence of junctions or perforators R>N2 or N2>N3.*

Therefore ***Valsalva is positive in closed shunts CS*** *overloaded by N1 (CS type I, III, IV, V or VI) according to the incompetence of the valves of the perforators, the junctions Saphéno-Femoral SFJ and Saphenopopliteal SPJ or the pelvic escape points* ***and negative*** *when the connection N1>N2 or N1>N3 is competent, as* ***in open deviated shunts ODS and Shunts 0****.*

<u>It alone allows the presence of a pelvic escape point</u> *discharging into a descending tributary of the arch of the great saphenous vein to be confirmed.*



Valsalva Manœuvre:
Particularly easy and reliable when performed by blowing into a blocked straw

7333- Interpretation of the effects of negative Valsalva (V-).

Valsalva is said to be "negative" (V-) when it does not activate flows during systolic thrust (forced systole of the thoracoabdominal pump).

On the other hand, the Valsalva manoeuvre does not cause reflux in shunts that are not supplied by N1. Thus, <u>it is negative in the case of open deviated shunts (type II shunts N2>N3) and S0 (type 0 shunt).</u>

It is therefore a good test for differentiating between these different shunts *<u>because their identification is decisive for the therapeutic</u> strategy and*

postoperative controls, particularly post CHIVA which transforms CS in Shunt 0 or undisconnected ODS left behind.

7334- *Interpretation of diastolic effects of valvulo-muscular pumps and Paranà manoeuvre*

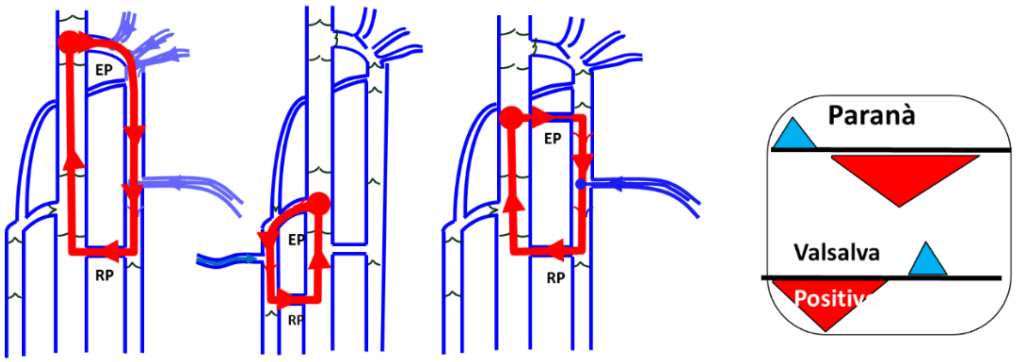
The Paranà manoeuvre is specific of the hemodynamic anomaly when it causes a diastolic or systolic reflux.

But the pathologic relevance of these flux and reflux can be assessed only when the escape points EP, pathways and re-entry points RP are identified by scanning up and down along the stream evoked by the manoeuvre.

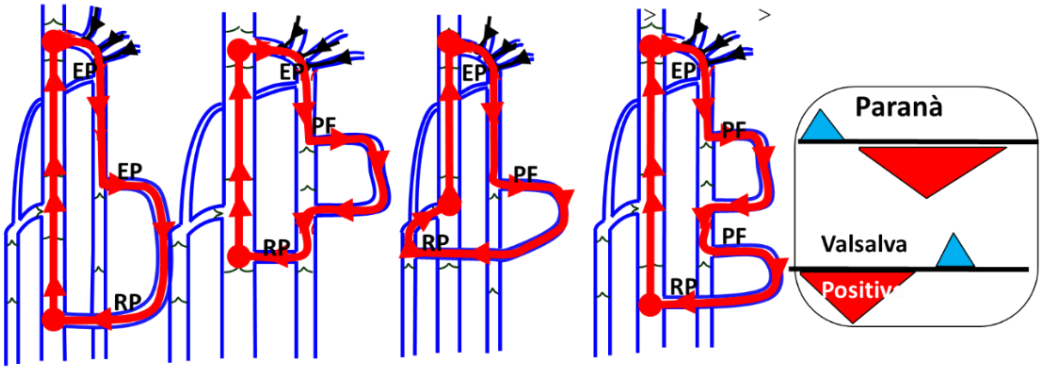
When the reflux is accompanied by an escape point with reversal of the drainage hierarchy*, it is a closed shunt CS overloaded by N1 (CS type I, III, IV, V or VI) or an open deviated shunt ODS, not overloaded by N1, but only by N2 (ODS shunt type II).*

In the absence of an escape point contrary to the drainage hierarchy*, the reflux is a simple flow reversal without pathological incidence called Shunt 0 S0.*

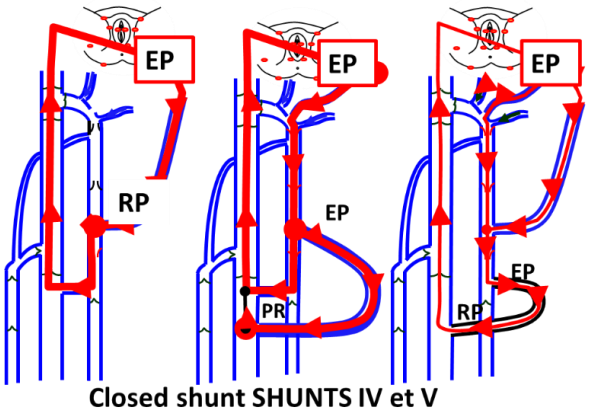
The differentiation between CS, ODS and SO must be confirmed by the Valsalva manoeuvre *as explained below.*

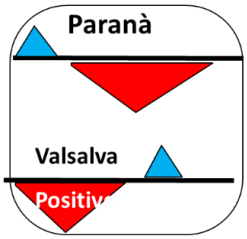


Closed Shunt SHUNTS I

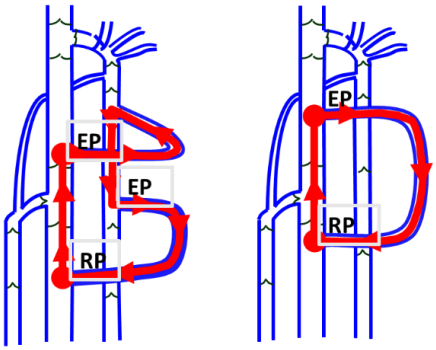


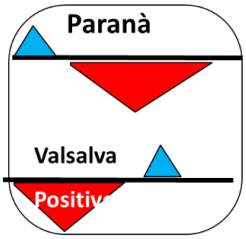
Closed Shunt SF SHUNTS III



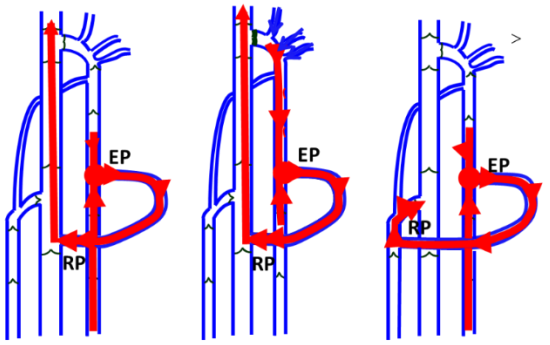


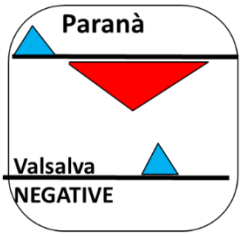
Closed shunt SHUNTS IV et V



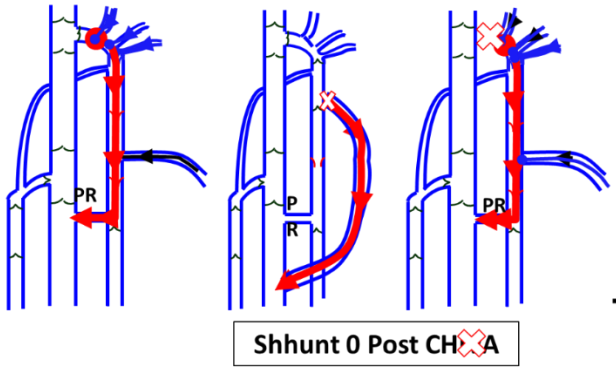


Closed shunt SHUNTS VI





Open deviated shunts ODS SHUNTS II



Shhunt 0 Post CHOA

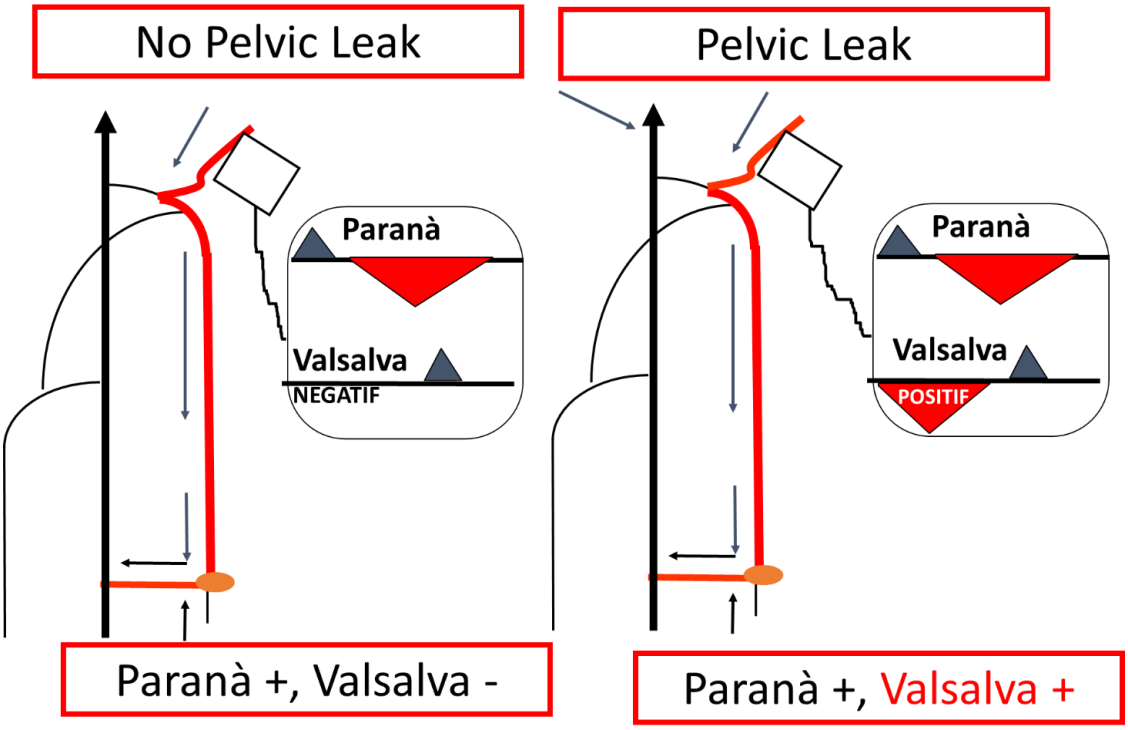
Shunts 0. NO escape point (no overloading flow)

7335- *Interpretation of the flows of the descending tributaries of the arch of the great saphenous vein and pelvic leaks*.

The Paranà manoeuvre (or compression-relaxation) cannot confirm pelvic reflux *on the direction of flow of the tributaries of the saphenofemoral junction.*

In fact, the diastolic flow of the tributaries of the saphenofemoral junction remains in a normal direction (without reflux) even when it is overloaded by reflux from a pelvic escape point.

This defect is corrected by the Valsalva manoeuvre *which, as we shall see below, is positive only if there is a pelvic escape point N1>N3.*



7336- *Interpretation of valvulo-muscular pump systolic flow.*

The Paranà manoeuvre (or compression-relaxation) is not specific *when it results in the normal direction systolic flow of an open vicarious shunt OVS.*

In this case, it is ***necessary to look for the systolic reflux from the escape point that feeds it****, most often through a leg perforator and/or the saphenopopliteal junction in case of femoral obstruction, but also the saphenofemoral junction in case of iliac obstruction.*

7337- Shunt I+II vs SHUNT III differentiation test: Paranà.

***Shunt I+II vs SHUNT III differentiation is crucial for the CHIVA strategy*.**

The Paranà manoeuvre is associated with manual or tourniquet block of the N3 tributary of the great saphenous vein N2 (N1>N2>N3>N1) to see if it is a type III shunt (without N2 intermediate re-entry perforator) or type I + II (with great saphenous vein trunk N2, intermediate re-entry perforator) because the CHIVA therapeutic strategies are different.

***In the case of a type III shunt**, the reflux of the overlying trunk of the great saphenous vein N2 is eliminated because there is no intermediate truncal re-entry perforator.*

***In case of type I + II shunt**, the reflux of the overlying trunk of the great saphenous vein N2 is maintained thanks to an intermediate GSV trunk re-entry perforator that can be visualized.*

***However, this method can be defective** when the re-entry perforator is not activated by the muscle pump on which it depends. This is sometimes the case in some patients when the thigh trunk perforator is not activated by the Parana manoeuvre or the calf compression-relaxation.*

Therefore, when the Paranà manoeuvre is negative, it must always be completed,** under the same conditions of compression of the refluxing tributary, **by the Valsalva manoeuvre, which does not depend on the diastolic activity of the valvulo-muscular re-entry pumps.

7338- *Shunt I+II vs SHUNT III differentiation test: Valsalva.*

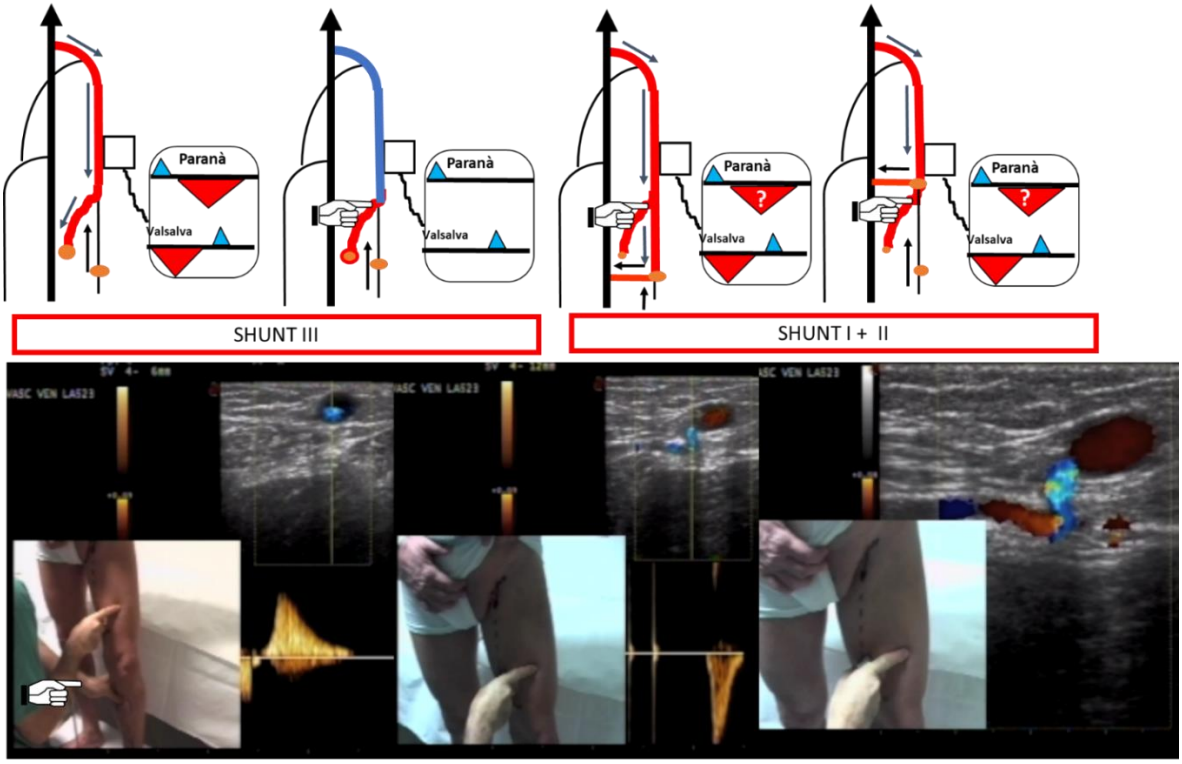
Like the Paranà manoeuvre, the Valsalva manoeuvre is associated with manual block or tourniquet of the N3 refluxing tributary of the great saphenous vein N2 to determine whether it is a type III or a type I + II shunt, for which the CHIVA therapeutic strategies are different.

***In case of a type III shunt,** the V+ reflux of the overlying trunk of the great saphenous vein N2 is suppressed because there is no intermediate truncal re-entry perforator.*

***In case of type I+ II shunt**, V+ reflux from the overlying trunk of the great saphenous vein N2 is maintained thanks to an intermediate re-entry perforator on the thigh trunk N2 which is visualized by colour Doppler.*

This method must always complement/verify the absence of diastolic Paranà reflux

Indeed, in some patients, the thigh trunk perforator is not activated by either the Paranà manoeuvre or calf compression-relaxation.



Differential diagnosis SHUNT III vs SHUNT II+I

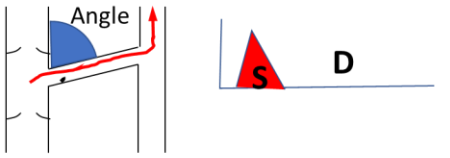
Effective manual compression of N3: if diastolic reflux -Paranà or compression-release of the N2 trunk of the Great Saphenous Vein persists, a re-entrant perforator must exist on N2. When the perforator drains into the femoral, the Paranà or compression-release maneuver may not show it. The Valsalva maneuver is essential to show it.

7339-Perforators

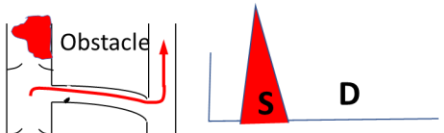
Perforator refluxes and calibre meanings according to echodoppler

C;Franceschi, R.Delfrate, M.Cappelli

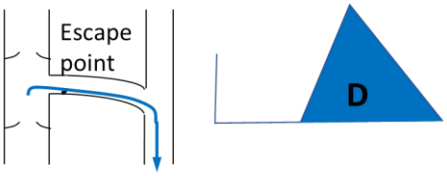
- **During the valvomuscular pump activation (Paranà, Squeezing-relaxation)**
  - Systolic reflux of perforator forming an acute upward angle with the deep vein without deep downstream obstacle is due to incompetence. It is not pathogenic.



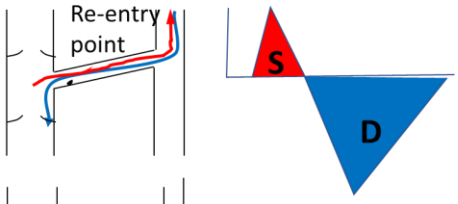
  - Systolic reflux due to deep obstacle is pathologic but not pathogenic because compensatory , so to be respected . It is the escape point of an open vicarious shunt.



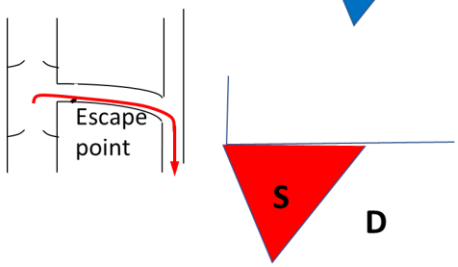
  - Diastolic reflux is pathogenic: escape point of a closed shunt.



  - Biphasic perforators (systolic reflux followed, by an inward re-entry diastolic flow) are not pathogenic if the diastolic flow prevails over the systolic reflux. It is usually the re-entry perforator of a shunt (Closed shunt CS, Open Deviated Shunt ODS, Shunt 0)



- **During Valsalva maneuvre**
  - Systolic Vasalva reflux is always pathogenic



- **Most of the time, large calibre doesn't mean reflux but overloaded to be respected re-entry**

734- *The venous tourniquet. Perthes' test.*

According to Perthes (> 120 years ago), tourniquets mimic surgical ligations on the dilated great saphenous vein.

It checks whether the underlying varicose veins collapse during walking.

-***Collapse indicates*** *that the deep system is functioning properly.*

-***Non-collapse indicates*** *that the deep system is not functioning properly.*

I propose to apply this principle to all levels of superficial veins that are being considered for disconnection based on the ultrasound data.

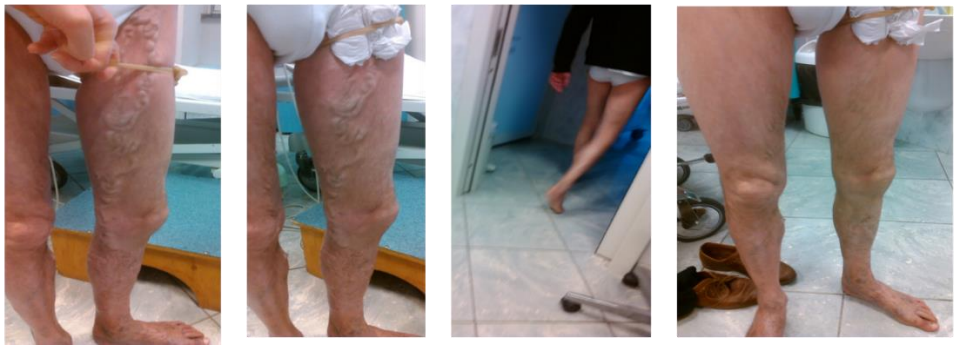
The non-collapse of the varicose veins must lead us to look for the precise cause.

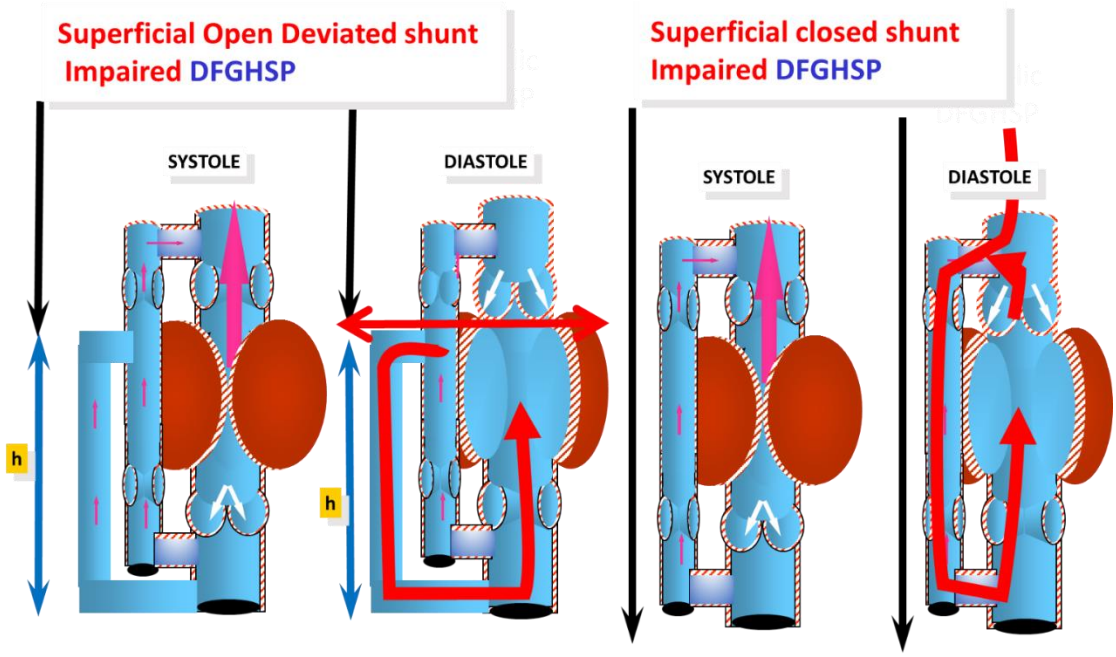
-An insufficiently tight tourniquet.

-Or a competitive deep reflux, when the deep reflux is massive and fills the muscle pumps more rapidly so that the varicose veins do not have time to empty.

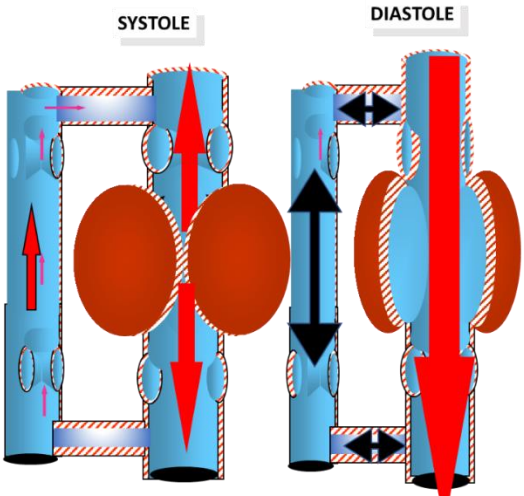
-Or a deep venous obstacle, for which the varicose veins are the compensatory route (Open Shunt Vicarious -OVS), further increasing their calibre and tension.

Clinical maneuvers:Perthes Manoeuver checks the deep venous system status according to the varicose veins collapse during walking with a thigh tourniquet and estimates the clinical outcoms of CHIVA disconnection without any venous ablation





Deep competitive reflux



Competitive deep reflux. In case of deep and superficial valvular incontinence, deep reflux dominates and prevents, when it is major, the reflux of the greater saphenous vein, even though it is varicose.
No reflux of the great saphenous vein on Doppler and the Perthes test does not result in its collapse

735- *Venous pressure Doppler measurement of TMP*

It is falsely scientific to dispute the value of measuring venous pressure with a sphygmomanometer cuff over the ankle and an echodoppler probe over the posterior tibial vein. Otherwise, the arterial pressure measurement at the ankle wouldn't be reliable!

Ref: 1- Bartolo M. Phlebodopplertensiometry, a non invasive method for measuring venous pressure. Folia Angiol. 1975; 25:199-203. 2-M. Bartolo. Non-invasive Venous pressure Measurements in Different Venous Diseases Angiology. Journal of vascular Diseases November 1983 .2_ Venous pressure Doppler: https://www.youtube.com/watch?v=SwLu3Z_tz3w

Taken venous pressure in the supine position,

It measures the only residual pressure *because the gravitational hydrostatic pressure is almost zero in this position.*

It allows to evaluate the hemodynamic value of the downstream venous resistances *(venous obstacles and/or deficit of the abdomino-thoracic and cardiac pumps).*

It is therefore very valuable for establishing a therapeutic strategy*, particularly when the relative importance of obstacles must be eliminated or confirmed in the context of post phlebitis disease where they are associated with valvular incompetence. In fact, valvular incompetence does not increase venous pressure in the supine position*

It is essential when it allows to affirm that the collaterals are *sufficient to compensate the obstacle when the venous pressure is normal.*

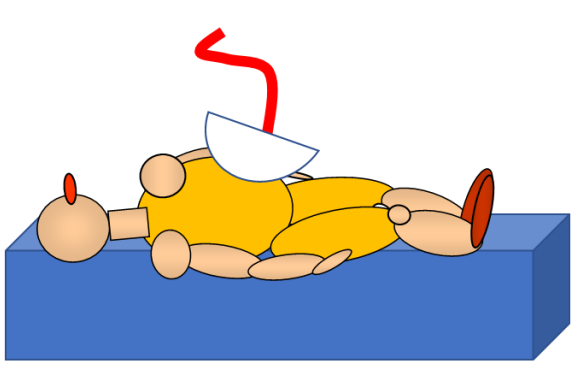
It must be compared with that of the contralateral lower limb *because of a possible excess of pressure caused by a decubitus position that is not perfectly horizontal, or cardiac insufficiency, or even compression of the vena cava by an obese abdomen.*

736- ***Positions for the echodoppler examination.***

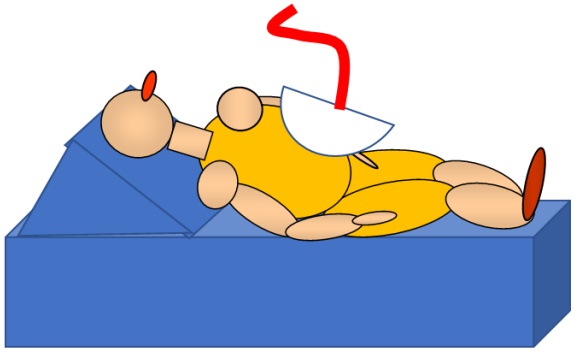
The patient's position must change according to the anatomic and hemodynamic diagnosis needs.

7361-Diagnosis of pelvic occlusions and incompetence.

73611- Recumbent and half-seated position



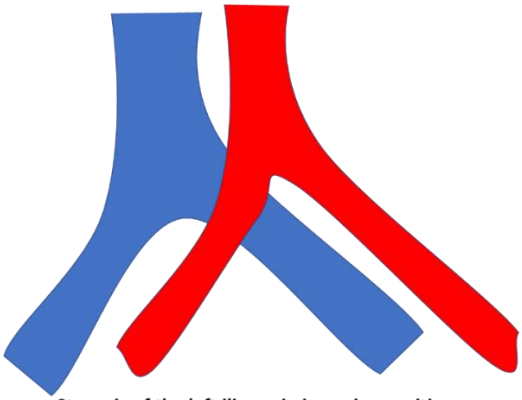
Pelvis and abdomen: Occlusion and stenosis of iliac and vena cava. Lumbar and azygos collaterals. Arterio-mesenteric clamp, left iliac vein clamp.



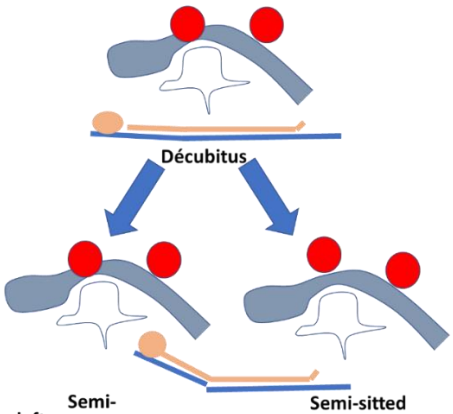
Pelvis and abdomen: If renal vein stenosis by aorto-mesenteric clamp or left iliac vein occlusion in decubitus, check in semi-sitting potion if it is not postural stenosis, usual artifact of MRI, and Phlebography.

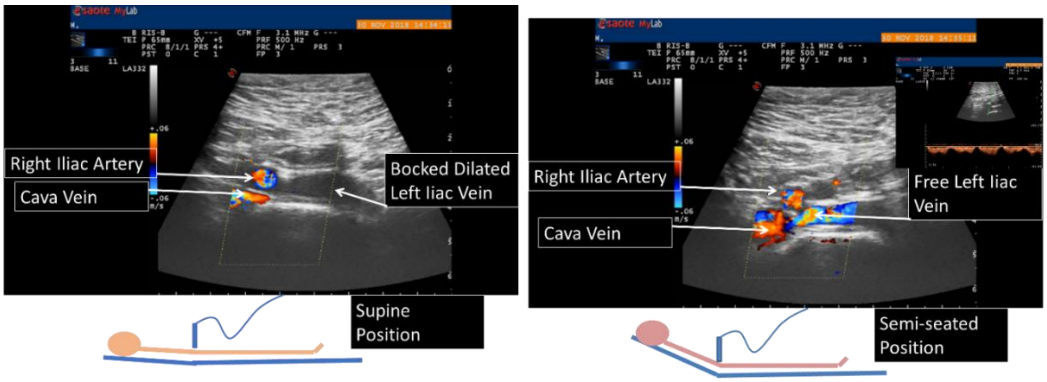
736111-Diagnosis of May Thurner syndrome (or Cockett) Nutcracker syndrome NTS

<u>In the case of horizontal supine occlusion of the left iliac vein, the appearance of iliac flow in the half-sitting position corrects the diagnosis of true MTS to pseudo-MTS</u>



Stenosis of the left iliac vein in supine position.
In the semi-seated position: If significant stenosis of the left iliac vein persists = true MTS,
If it becomes free = pseudo MTS





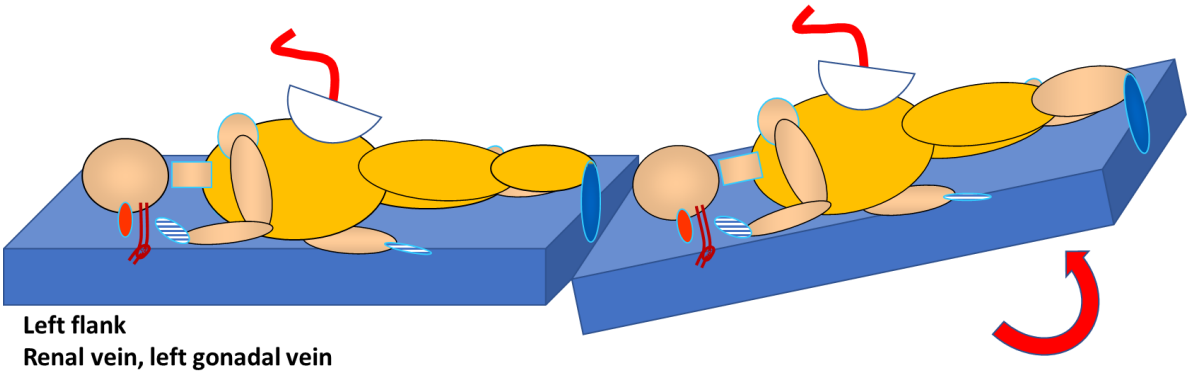
May Thurner Syndrome at phlebography but
Pseudo May Thurner Syndrome on Echodoppler: only postural

Ref: Paolo Zamboni, Claude Franceschi, Roberto Del Frate.The overtreatment of illusory May Thurner syndrome Veins and Lymphatics 2019; volume 8:8020.

Phlebography and echodoppler *performed only in the* ***horizontal position*** *may provide an* ***overdiagnosis*** *indicate an* ***overtreatment stenting in pseudo-MTS. This may explain the finding of "illusory" MTS assessed by horizontal supine phlebography in young asymptomatic subjects.Ref:*** van Vuuren TM, Kurstjens RLM,Wittens CHA, et al. Illusory angiographic signs of significant Iliac vein compression in healthy volunteers. Eur.J Vasc Endovasc Surg 2018;56:874-9.

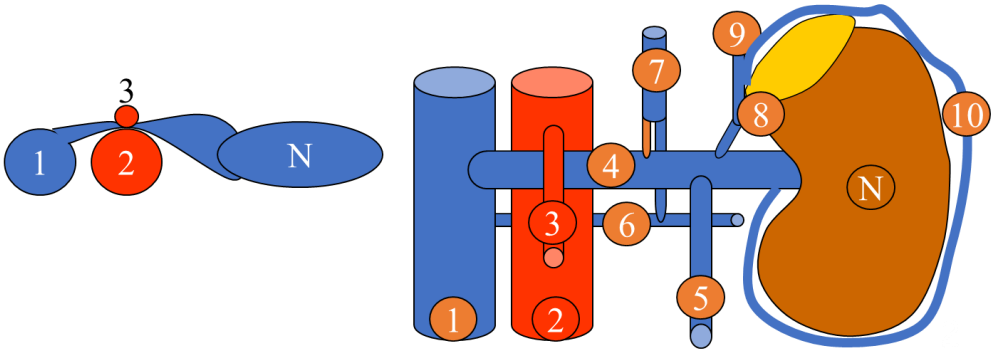
VIDEO: Pseudo MTS : https://www.youtube.com/watch?v=h931XXo2hdk&t=23s

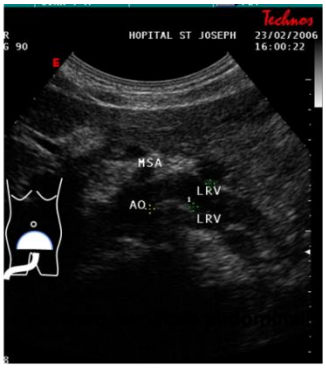
In the case of horizontal supine occlusion of the left renal vein by the aorto-mesenteric clamp, the appearance of flow in the semi-seated position corrects the diagnosis of true NTS into postural NTS

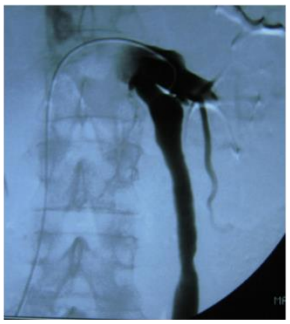


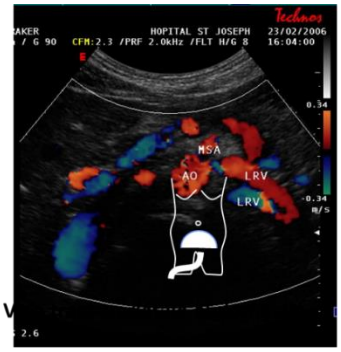
Left flank
Renal vein, left gonadal vein
Varicocele, Nutcarcker: Trendelenburg test to check if the resting gonadal reflux modulated by breathing persists. If yes: NTS. If no, no NTS.

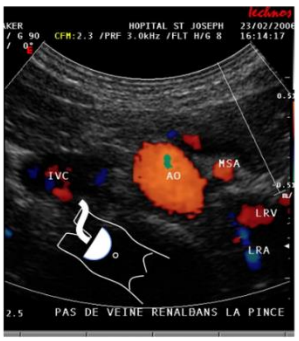
Compression of the left renal vein (4) against the Aorta (2) by the superior mesenteric artery (3) can generate a dangerous hemodynamic stenosis for the left kidney (N) due to a drainage deficit and a vicarious overload of the hemi-azygos (7), lumbar (6) adrenal (8) and phrenic (9) veins, the perirenal venous circle (10) (hemorrhagic risk) and the genital vein (5) whose reflux and pressure generate a left varicocele (spermatic in men and ovarian in women) sometimes responsible for a pelvic congestion syndrome.









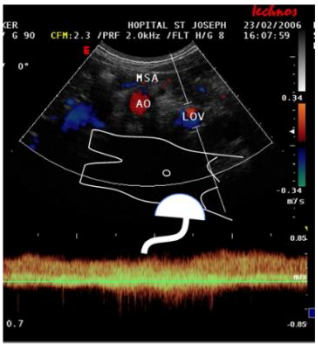


Trans-abdominal scan in Trendelenburg position.

Aorto-Mesenteric clip.
Venous flow = 0

No reno-azygo-lombar compensation

PENMANENT left ovaric vein reflux in Trendelenburg position (head lower than the feet).



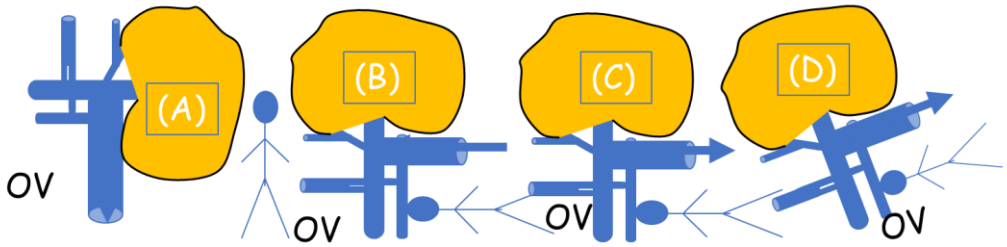
Trans-abdominal scan in Trendelenburg position.

Nutcracker Syndrome: Total Aorto-Mesenteric clip
Single bypass through the left ovarian vein

How to assess the risk? Measure the pressure in the renal vein, spontaneously and during the occlusion of the refluxing ovarian vein. By echodoppler: the reflux in the ovarian vein (VO)

-perceived in standing, in the sitting and half-sitting position and modulated by respiration, it can be due to the force of gravity alone without needing residual renal venous pressure (A). It is normalized in decubitus (B)

-If it is perceived as permanent and in decubitus, it can no longer be related to the force of gravity but to a saving vicarious effect (C). This test is made more sensitive by tilting the patient into Trendelenburg where the reflux is seen to persist (D). This pressure can be approximated by measuring the inclination necessary to stop this reflux.



7361112- Indirect diagnosis of iliac and cava obstacles and incompetence:

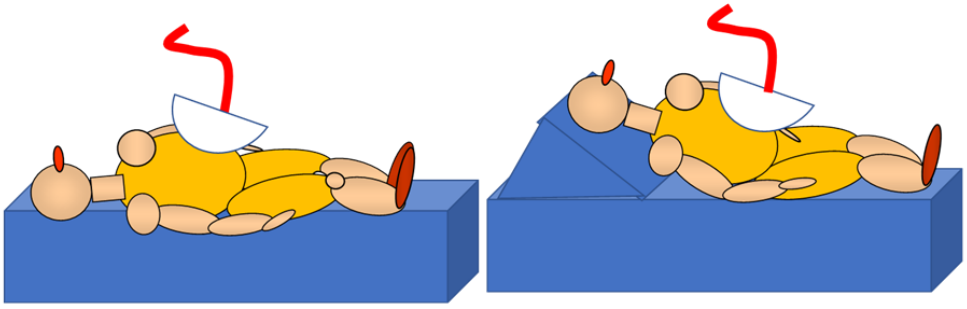
-non-breathing modulation of the femoral flow in supine position

-Indirect diagnosis of iliofemoral incompetence by Valsalva in upright position

73612-- *Right side horizontal lying position.*

Examination of the <u>left gonadal vein (ovarian and testicular).</u>

If the reflux of the gonadal vein (varicocele) persists *in the Trendelenburg position (inclination of the examination table with the feet higher than the head),* ***it means that it is <u>an open vicarious shunt compensating</u> for a Nutcracker Syndrome NTS.***



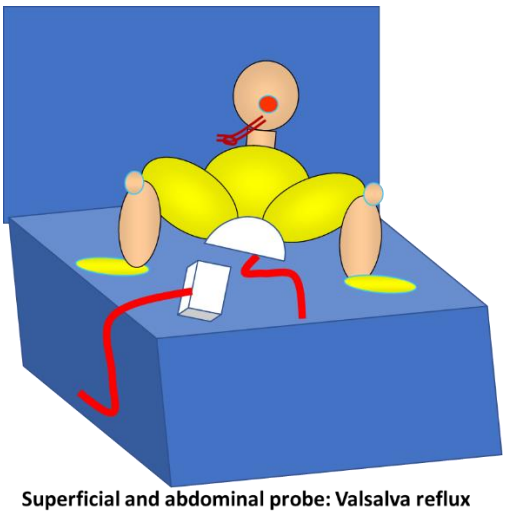
Pelvis and abdomen: Occlusion and stenosis of iliac and vena cava. Lumbar and azygos collaterals. Arterio-mesenteric clamp, left iliac vein clamp.

Pelvis and abdomen: If renal vein stenosis by aorto-mesenteric clamp or left iliac vein occlusion in decubitus, check in semi-sitting potion if it is not postural stenosis, usual artifact of MRI, and Phlebography.

73613--Gynaecological position

Examination with a trans-perineal (not trans-vaginal or trans-rectal) abdominal probe of hypogastric veins and their tributaries, including Valsalva reflux through the pudendal veins into Alcock's canal but also reflux around the ovaries.

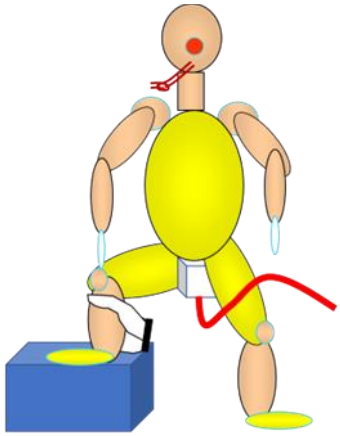
The trans-vaginal scan is useless *as it does not allow the origin, route and termination of the refluxes to be determined.*



Superficial and abdominal probe: Valsalva reflux of incontinence of superficial and deep perineal veins, pelvic veins, hypogastric veins, pudendal veins, Varicoceles and Perineal and Clitoral leak points

73614-Standing position, with an elevated leg.

One foot on a stool 40-50 cm high. Examination of the perineal and clitoral escape points under the Valsalva manoeuvre. Reflux by compression of the calf is not sufficient to confirm reflux of the pelvic veins. This is particularly the case when, after embolization of the pelvic veins or varicocele, there is still a negative Valsalva reflux.



Perineal leak point
Valsalva, Compression-release

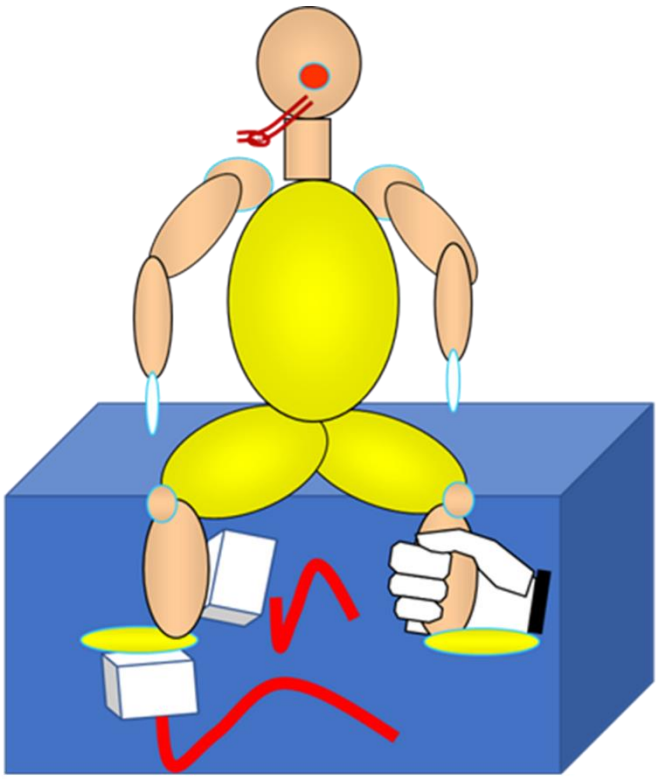
7362--- *Diagnosis of iliofemoral incompetence and leg vein occlusions.*

-Except in cases of suspected recent phlebitis, *which requires caution and examination in the supine position, the first and* ***most instructive position is the standing position.***

-<u>To avoid any risk of a serious fall</u>, I prefer not to have the patient stand on a stepladder.

-I examine the patient standing on a 20 cm high platform at the edge of the examination bed.

-To examine the <u>feet and ankles directly</u>, the patient sits with his or *her* ***legs dangling off the examination bed.***



Tibial, sural and plantar veins.
Phlebitis, reflux.
Compression-relaxation
Valsalva

73621-Standing position:

Dynamic tests:

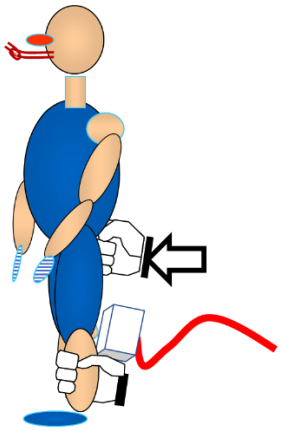
1-Parana manoeuvre,

2-Valsalva manoeuvre

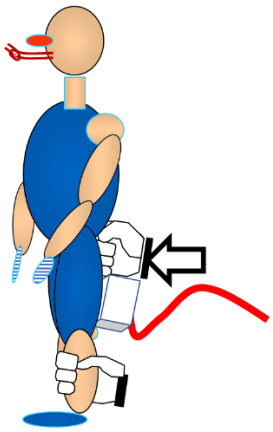
3-Shunt III and Shunt I+II differentiation test.

4-Perthes test

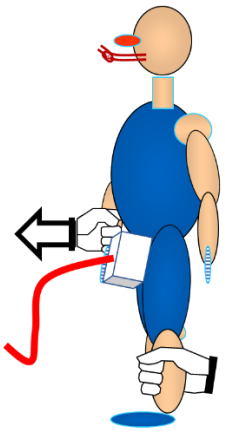
Preoperative markings



Popliteal fossa
Parana, Compression-relaxation
Valsalva, Thrombosis, reflux Popliteal veins, Small Saphenous vein;
Giacomini. Gastrocnemius, sural.
Popliteal cyst, muscular rupture (medial gastrocnemius) hematoma.



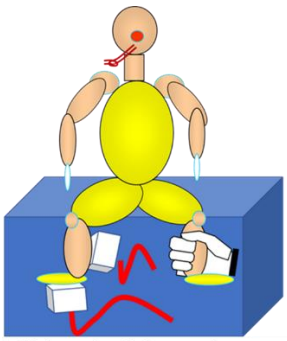
Posterior aspect of the thigh
Parana, Compression-relaxation (deep femoral)
Valsalva, Femoral Veins, Giacomini
Sciatic Vein. Leak points Perineal PP, Gluteal Superior and inferiror.



Inguinal area
Parana, Compression-relaxation
Valsalva, Thrombosis, reflux Popliteal veins, Great Saphenous vein;
Descending tributaries, Pelvic leakage: Obturator point, Inguinal point, Clitoral point

73622- *Sitting position*

compression-relaxation of the calf and foot.



Tibial, sural and plantar veins.
Phlebitis, reflux.
Compression-relaxation
Valsalva

73623-Recumbent position

Direct diagnosis of thrombosis is made by compressing the veins with the probe. Total or partial non-compression indicates total or partial venous thrombosis. This compression is only reliable if the muscles are relaxed, i.e. in the supine position.

In order to be identified, the veins must also be dilated, i.e., there must be a minimum gravitational hydrostatic pressure. The ideal position is therefore the half-seated supine patient.

The exploration is refined at the level of the calf and the soles of the feet, with the patient sitting with legs hanging over the edge of the examination bed.

737---Echodoppler examination: hemodynamic signs

7371- Supine and half-seated examination:

73711-Probe compression tests of veins of the sole of the foot, calf, popliteal fossa, and thigh.

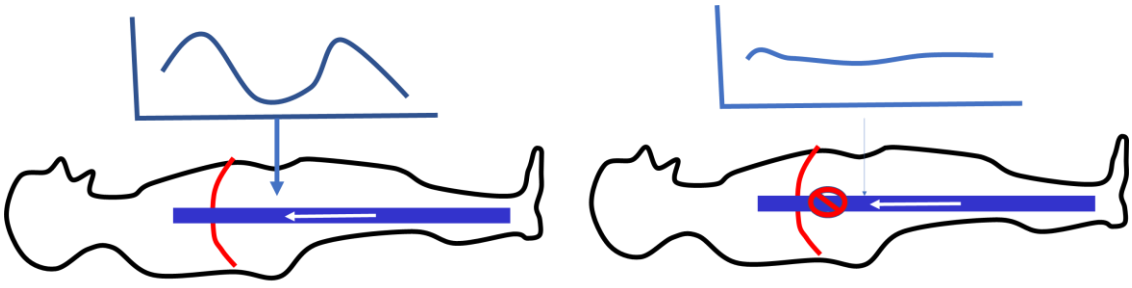
Non-compressibility + absence of flow on colour Doppler = complete occlusion.

Partial compressibility + colour Doppler flow = partial thrombosis.

73712--Femoral vein flow modulated by respiration, increases during expiration and decreases during inspiration, both at rest and during exercise.

If it is demodulated, at rest, there is a significant downstream hemodynamic obstacle at rest.

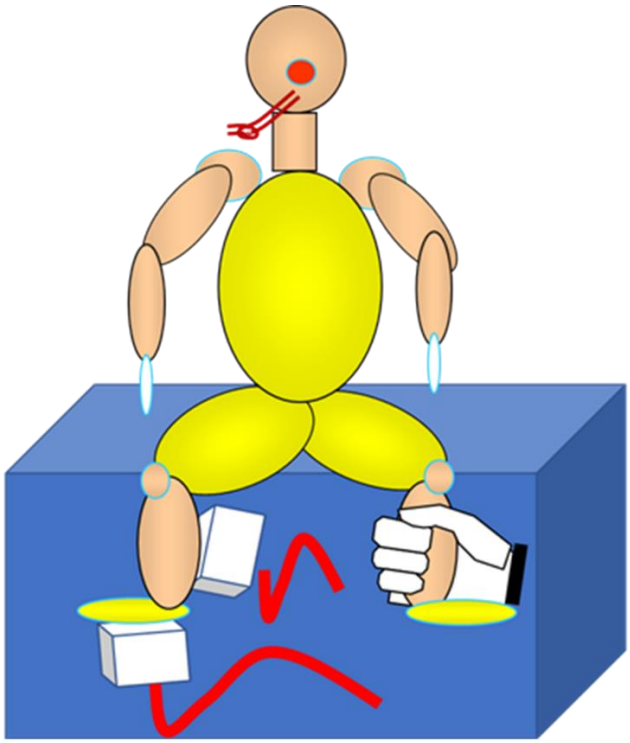
If it is demodulated only after 15 pedal movements, the obstacle is significant only at stress.



Iliocaval obstruction: Loss of respiratory modulation of femoral flow on Doppler

73713-- ***Reflux into the common femoral vein during Valsalva manoeuvre shows common iliofemoral incompetence with prolonged reflux into the great saphenous vein and/or deep thigh veins.***

7372--Sitting on the edge of the examination bed



Tibial, sural and plantar veins.
Phlebitis, reflux.
Compression-relaxation
Valsalva

73721- *Probe compression tests of the veins of the sole of the foot of the foot, calf.*

Non-compressibility + absence of flow on colour Doppler = *complete occlusion.*

Partial compressibility + colour Doppler flow = *partial thrombosis.*

73722-Flow and reflux of the Tibial, Fibular, Soleus and gastrocnemius veins *according to* ***compression relaxation of the foot and calf***

7373-Standing examination:

73731-Popliteal vein:

737311-Popliteal vein and gastrocnemius.

At rest and Paranà manoeuvre:

-Total, partial, or segmental flow and reflux of the popliteal vein.

-Flow and reflux of the gastrocnemius veins.

Note that popliteal reflux does not mean N1 deep incompetence, when *a segment of popliteal vein located between competent upstream and downstream valves refluxes during Parana diastole into the escape point (saphenofemoral junction or popliteal perforator) of a closed shunt.*

It is normal for this reflux to disappear when the escape point is disconnected.

737312-Great and small saphenous veins.

At rest and Parana manoeuvre.

Assesses the systolic and diastolic flows and refluxes of the *saphenopopliteal junction, small saphenous vein, Giacomini's vein and possible perforator of the popliteal fossa.*

Locates the *frequent junctions of the small saphenous vein and the gastrocnemius vein, which form a common trunk* ***which then drains into the popliteal vein.***

Be aware of reflux from the saphenopopliteal junction.

- Systolic reflux *when it is an open shunt escape point vicariant to the OVS.*

- Diastolic reflux *when it is a closed shunt escape point CS.*

-Systolic and diastolic reflux *when OVS and CS have the same escape point:* ***Mixed shunt MS.***

737313-Check for the presence of popliteal cyst which can be a cause of pain and oedema.

73732- Groin area:

- Rest,

- Parana test

-Valsalva test:

Total, partial or segmental iliofemoral flow and reflux.

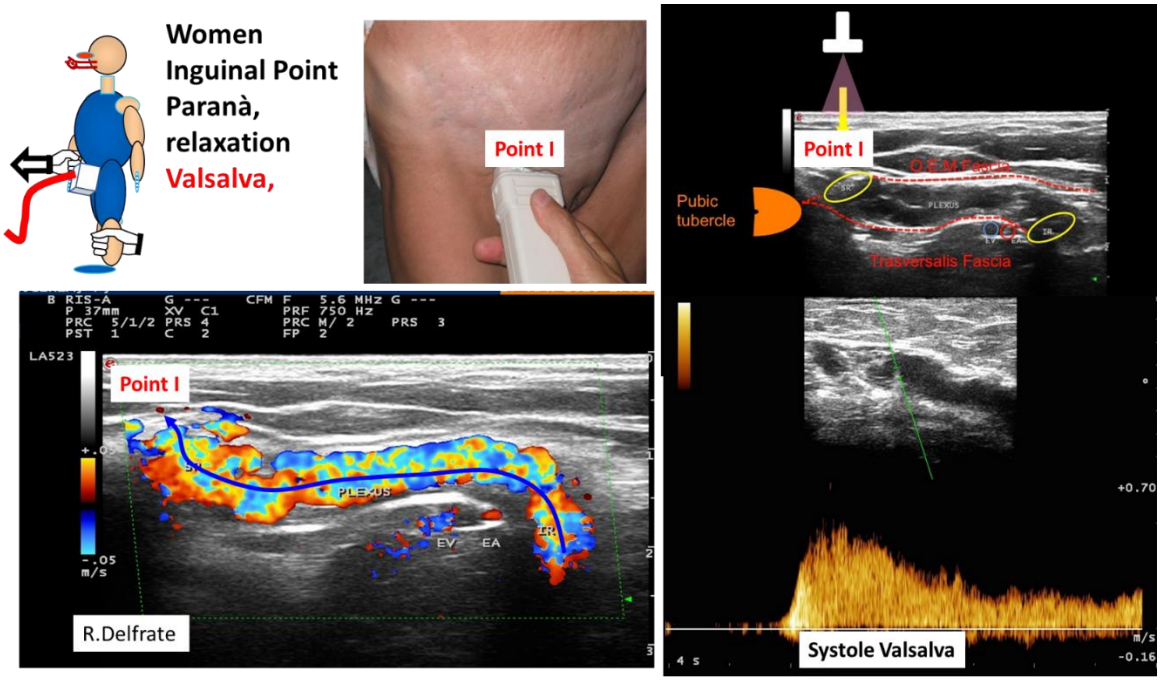
737321-Systolic and diastolic flow and reflux *of the saphenofemoral junction, great saphenous vein****, tributaries of the arch of the great saphenous vein by the Paranà manoeuvre.***

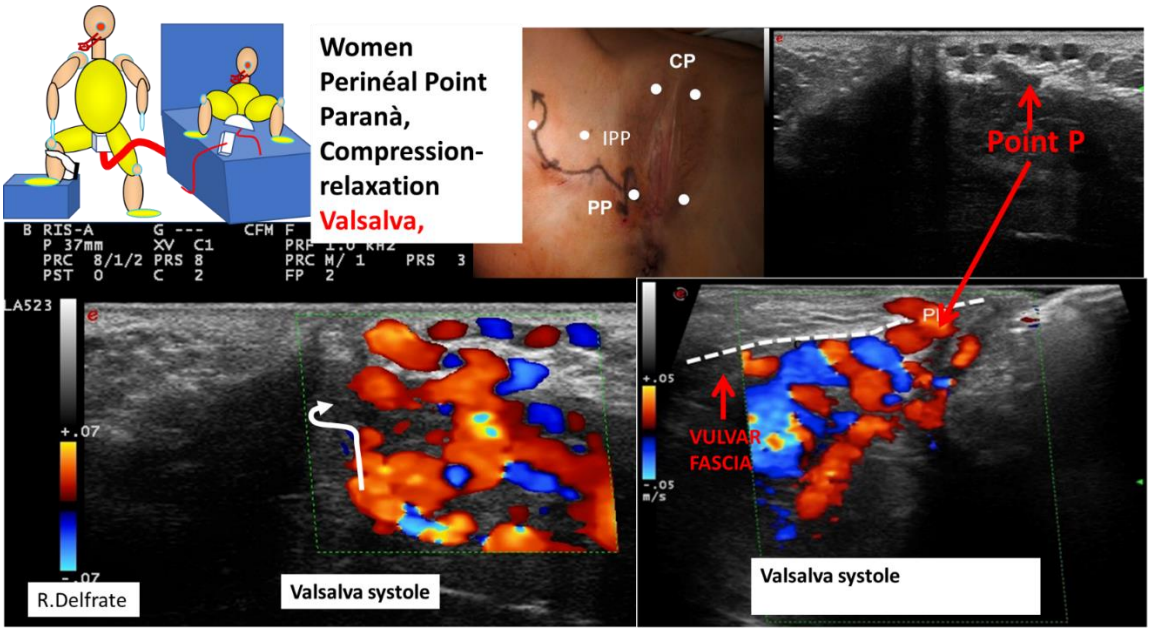
Confirm Paranà Reflux of the Femoral-Popliteal junction by

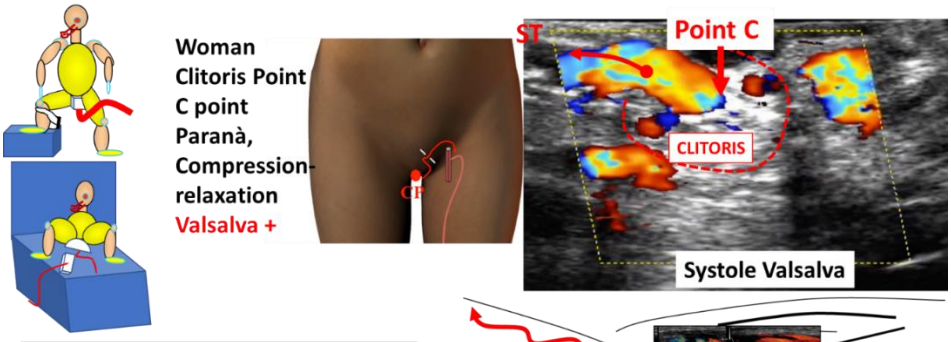
Valsalva + which confirms reflux from the femoral vein.

737322-Reflux from pelvic visceral escape points

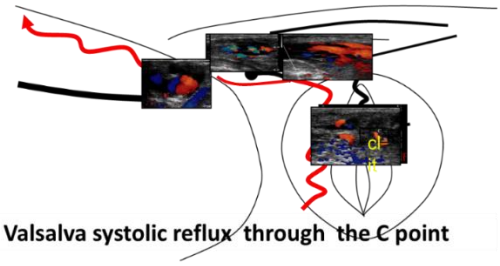
Inguinal point (I-point), Perineal point (P-point), Clitoral point (C-point)







Plexus ven. Commnicans (Clit. Et Bulbus vest.): Valsalva reflux Internal Pudendal V. into the Antarior Saphena via dans la Saphène Antérieures via Plexus Ven. Communicans (Clit. et Bulbus vest.), Veine Dorsal du Clitoris ane external Pudendale V.



Valsalva systolic reflux through the C point

<u>These points are particular because they have long been misunderstood for 2 reasons:</u>

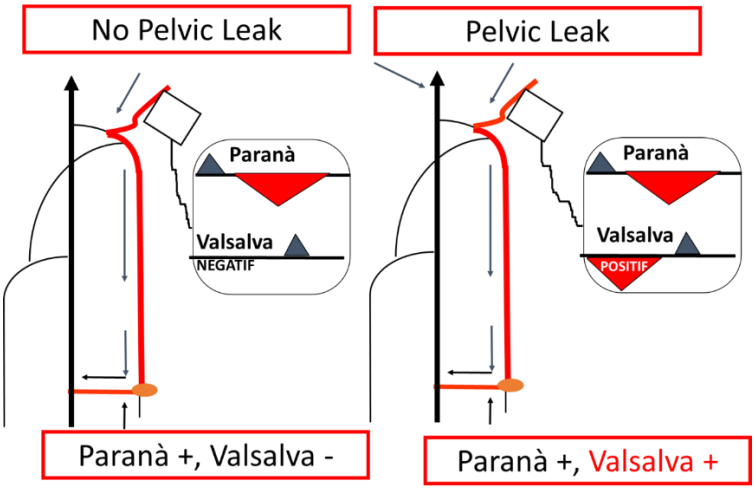
-Firstly, they drain through the tributaries of the saphenous vein without reversing the direction of their flow (no reflux).

-Secondly, they can also drain through one of the tributaries of the contralateral junction via suprapubic and subpubic anastomoses.

They are much more frequent in mono or multiparous women than in men.

<u>The normal flow of the tributaries of the saphenous vein does not change direction when it is overloaded by the pelvic escape points (no reflux!) during the diastole of the Parana manoeuvre or the release of the compression</u>.

<u>The Valsalva manoeuvre alone can make a difference.</u> -In the absence of pelvic reflux, *the flow is stopped by the Valsalva thrust* ***(Valsalva -)*** *and reappears with release* ***-In the case of pelvic reflux****, the flow appears during the push* ***(Valsalva +).***



***To identify the visceral escape points**, we go upstream **along the Valsalva + veins** which* *leads to*

Obturator point (O point), or Perineal point (P point), or Clitoral point (C point).

Sometimes the pelvic escape point may be contralateral.

The Obturator vein (O Point) *refluxes into the Great Saphenous vein usually very close to the saphenofemoral junction.*

The Internal Pudendal vein (P Point) refluxes through the perineal and/or the posterior labial vein through the superficial perineal fascia at the junction of the ¾ anterior and ¼ posterior of the Vulvo-perineal fold. The reflux can be transmitted to the great saphenous arch, but more frequently lower, into the thigh trunk. It can also descent along the posterior face of the thigh, down to the popliteal fossa.

The vein of the Round Ligament of Uterus Inguinal point refluxes through the superficial ring of the inguinal canal (I point) into the great saphenous arch tributaries.

The Internal pudendal vein refluxes into the dorsal clitoral vein at the basis of the clitoris (C point). Then in the Great saphenous vein through the External Pudendal vein.

Pelvic escape points are much more frequent in mono or multiparous women.

In men, the equivalent of the Clitoral point C point can be found at the level of the dorsal vein of the penis, and the equivalent of the Inguinal point I point fed by a varicocele.

***The pelvic parietal escape points** are the Obturator point **O point** and the superior Gluteal point **SG Point** and inferior Gluteal point **IG Point**, can be found in both men and women.*

***The Superior Gluteal SGV and Inferior Gluteal IGV escape points** most often reflux in the context of a venous malformation.*

They often feed the Sciatic Vein which then drains into the small Saphenous Vein.

In practice, a Valsalva + flow of normal or reversed direction *in a superficial vein, descending tributary of the saphenofemoral junction, Grande Saphenous vein, Anterior Saphenous vein, any superficial flow, of normal or reversed direction by the Valsalva systolic thrust **must lead to a search for the escape point which feeds it, homolateral, but also contralateral.***

737323-*Great saphenous vein GSV*

The anatomy of the GSV N2, ideally of regular calibre, located in a duplication of the fascia (Bailly's Egyptian eye) is not always present.

The sapheno-femoral unction SFJ

may be double,

and the trunk may be more or less hypoplastic over variable areas. This hypoplasia must be recognized because they must be considered in the treatment of type III shunts in particular.

The measurement of the calibre of the arch saphenous vein is useless.

It is more rational to measure its trunk at 10 cm under the saphenous femoral junction SFJ which is of interest before and after treatment to assess the effect of disconnection of closed shunts. This calibre, which is often increased by a diastolic overload of a closed shunt, regresses after disconnection of the escape point. Ref: Mendoza E., Diameter reduction of the great saphenous vein and the common femoralvein after CHIVA Long–term results, Phlebologie, 2013, 42: pp. 65–69***.***

However, large saphenous veins may be functionally normal!

7373231-Normal hemodynamics of the Great saphenous vein GSV

3732311-The Paranà Manoeuvre activates both the calf and sole pumps (Léjars' pump)

The "normal" systolic flow is usually that provided by the Léjars pump when the leg and thigh perforators are satisfied. This is evidenced by the disappearance of this flow if a tourniquet is applied to the ankle. This explains why it may not appear when the foot support is modified (hollow foot and/or flat foot). Video: https://youtu.be/ktZAYBX9Km4

It also appears in cases of incompetence of the leg perforators, without much pathological significance when it is isolated or associated with a re-entry flow of closed shunt CS or open deviated shunts ODS. It is pathological when it is the escape point of an open vicarious shunt OVS.

73732312-Manual compression of the calf activates systolic saphenous flow in a non-physiological way by direct artificial compression of the tributaries and the saphenous trunk of the leg.

It may also activate systolic reflux of the incompetent perforators which drain into the great saphenous vein.

Manual compression of the foot reproduces the systole of the Léjars pump.

73732313-N3 tributary veins of the great Saphenous vein N2.

The tributaries drain a superficial territory called the "phlebosome".

The flow of the tributaries*, whether anterograde or refluxing****, is pathological or not depending on its source, destination, and content.***

<u>It is not pathological when</u> it is Parana refluent in diastole but Valsalva negative *and not fed by the saphenous trunk N2 (especially after disconnection of a shunt N2>N3) it makes a shunt 0.*

<u>It is pathological when</u> *it is fed only by the saphenous trunk (Open Shunt by ODS N2>N3, type II shunt) i.e.,* ***when it is Parana refluent in diastole but not at Valsalva (Valsalva negative).***

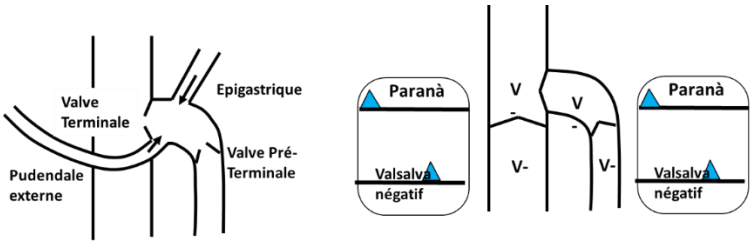
<u>It is pathological when it is Valsalva positive</u>*, fed via the saphenous trunk by a N1>N2 escape point (saphenofemoral junction or pelvic escape point). It is a Shunt III, IV, V (N1>N2>N3>N1). This is also the case of a closed type VI shunt N1>N3>N1*

7373232-The hemodynamic of the saphenous trunk.

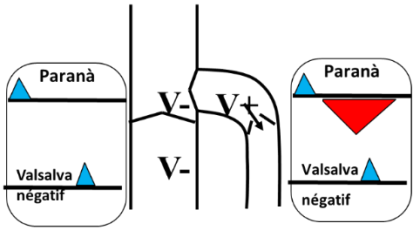
The <u>Terminal Valve TV</u> is the one located closest to the saphenofemoral junction.

The <u>pre-terminal PTV</u> is the valve located behind the latter.

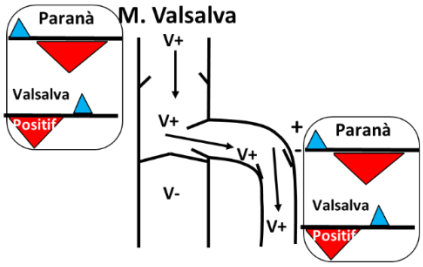
This anatomical <u>distinction is hemodynamically and strategically important</u>, *depending on whether one or the other or both are incompetent****.***



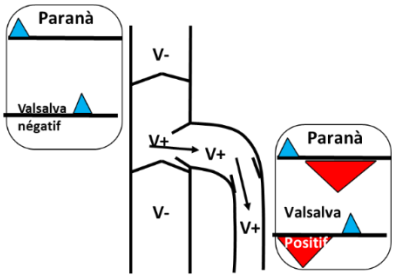
No reflux



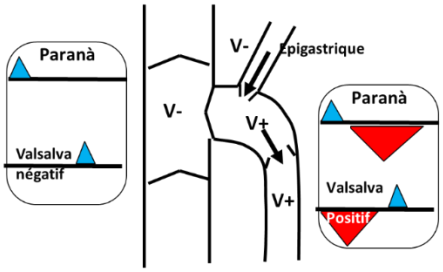
Pre-Terminal Valve incompetence.
No overloading reflux



Terminal , Pre-Terminal Valve incompetence
Femoral V competent.
Great N1 overloading reflux

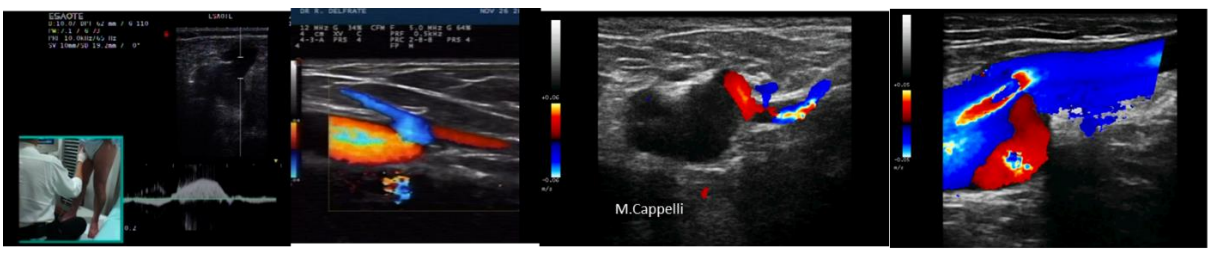


Terminal , Pre-Terminal Valve incompetence
Femoral V competent.
Small N1 overloading reflux

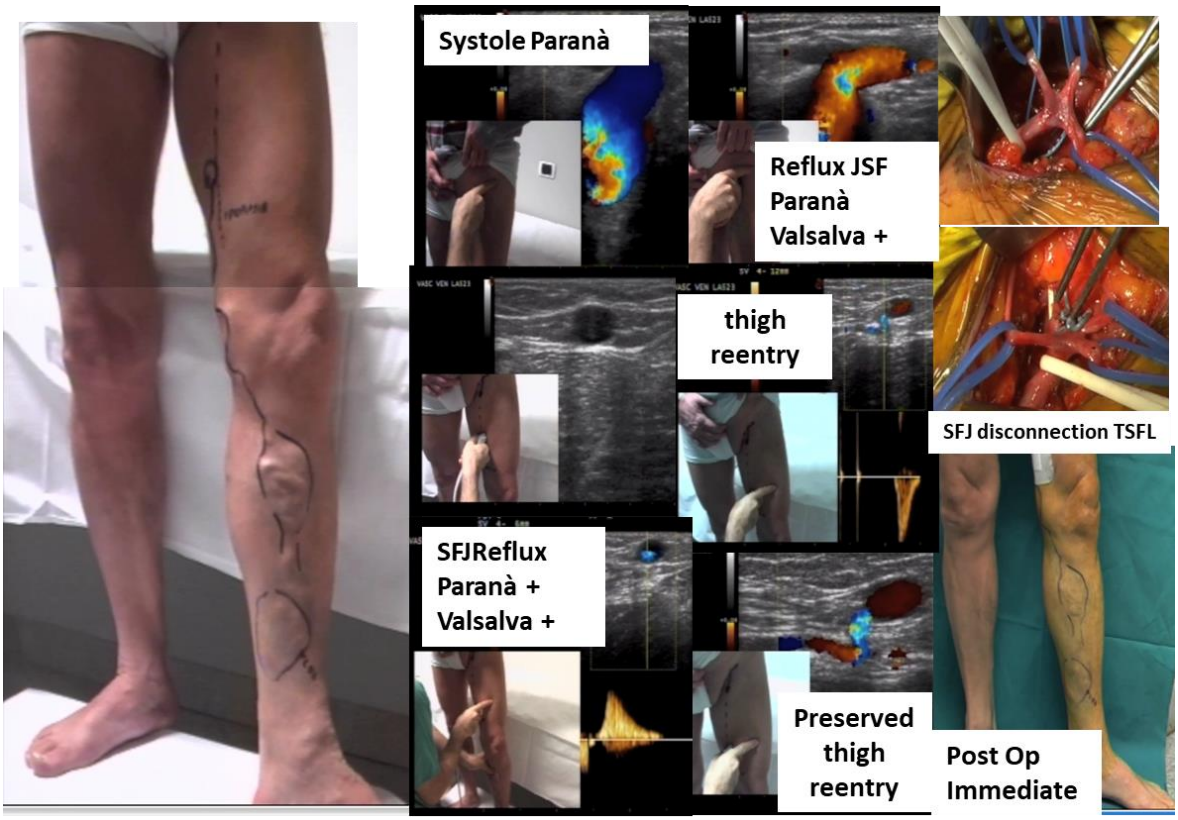


Terminal valve comptent. Pre-treminal valve incompetent.
Reflux overloaded by pelvic leak ,

Safenofemoral junction



GSV terminal valve reflux



These <u>distinctions are diagnosed by the combination of the Parana and Valsalva manoeuvres.</u>

It should be noted from now on <u>that not every retrograde flow called reflux is pathogenic and not every anterograde flow is normal</u>. What should define <u>the pathological or non-pathological character of a flow is not its direction alone, but its origin, destination, and content.</u>

Diastolic Paranà reflux alone does not allow the flow to be qualified hemodynamically or functionally. Indeed, it does not allow us to confirm or deny an N1>N2 femoral or pelvi-femoral escape point.

Only a Valsalva+ can confirm the presence of an N1>N2 escape point.

Diastolic reflux N1>N2 at Saphenous femoral junction SFJ.

This is the escape point of a closed CS shunt. It is Valsalva positive at the saphenofemoral junction SFJ (<u>incompetent Terminal</u> Valve TV).

<u>By raising the colour PRF, trans-valvular reflux can be visualized. It varies from a thin stream through a small hole in a valve to massive reflux.</u>

Diastolic reflux N1>N2 Pelvi-saphenous reflux.

This is the escape point of a closed CS shunt.

It is negative Valsalva V- at the level of the saphenofemoral junction (competent terminal valve)

and positive Valsalva V+ upstream (incompetent pre-terminal valve).

The pelvic escape point should be sought (point O, I; P, C) by following only the positive Valsalva tributaries.

Diastolic reflux N2 without reflux N1>N2.

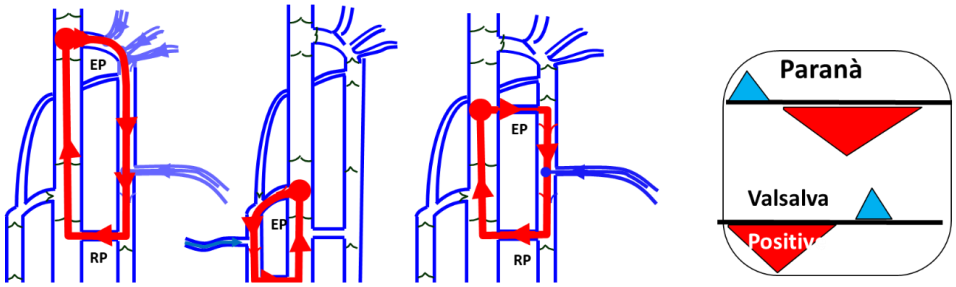
Paranà diastolic reflux but negative Valsalva. It is a simple retrograde flow, not overloaded and not pathogenic

Because of a competent terminal valve TV,

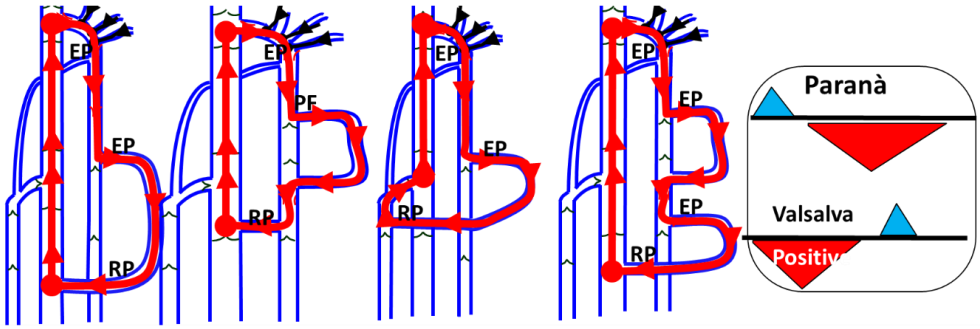
Or after disconnection of the SFJ escape point of a closed femoral-saphenous or pelvic-saphenous shunt.

Although benign, this reflux is still too often considered as pathological and treated as such by those who do not understand venous physiopathology.

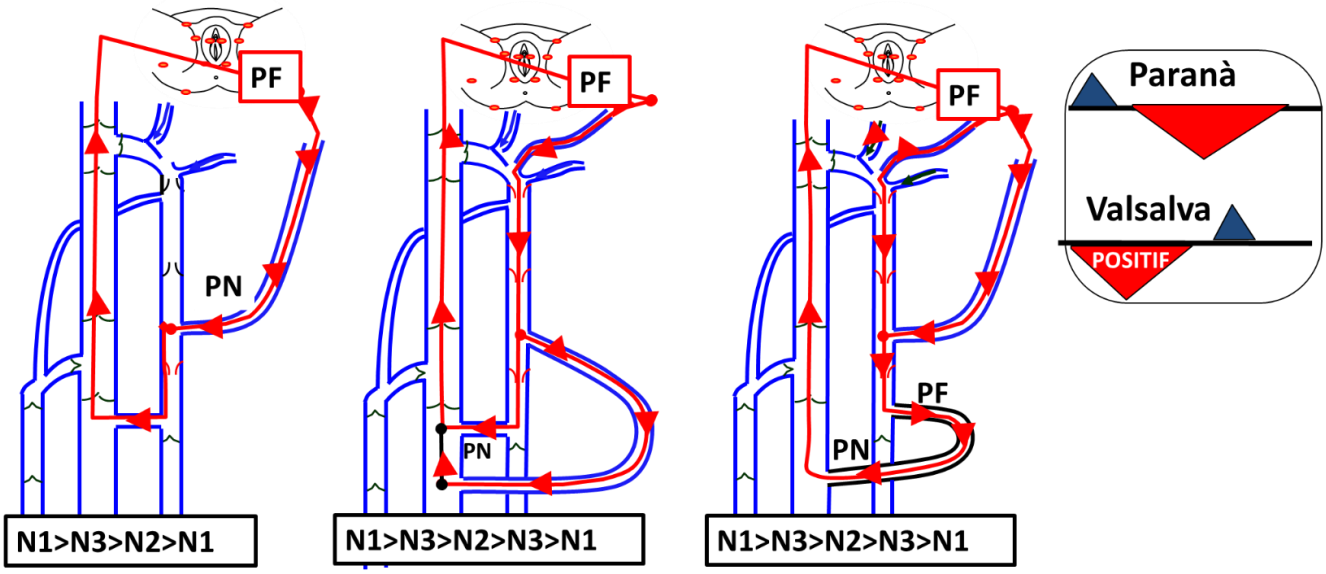
This N2 diastolic reflux without N1>N2 reflux may also simply be the result of a deviation of the flow of a segment of saphenous vein between 2 competent valves, due to a small resistance. This small resistance may be an underlying re-entry perforator or an incompetent N3 tributary (open deviated shunts Type II) Valsalva negative because there is no N1>N2 or N1>N3 incompetence which can transmit the Valsalva systolic thrust).



Closed SHUNT CS. Recirculation. SHUNT Type I N1>N2>N1: Overloaded by N1 3 examples EP= Escape Point Junction Saphéno-femoaral junction. Sapheno-popliteal junction. Thigh perforator. RP= Reentry point

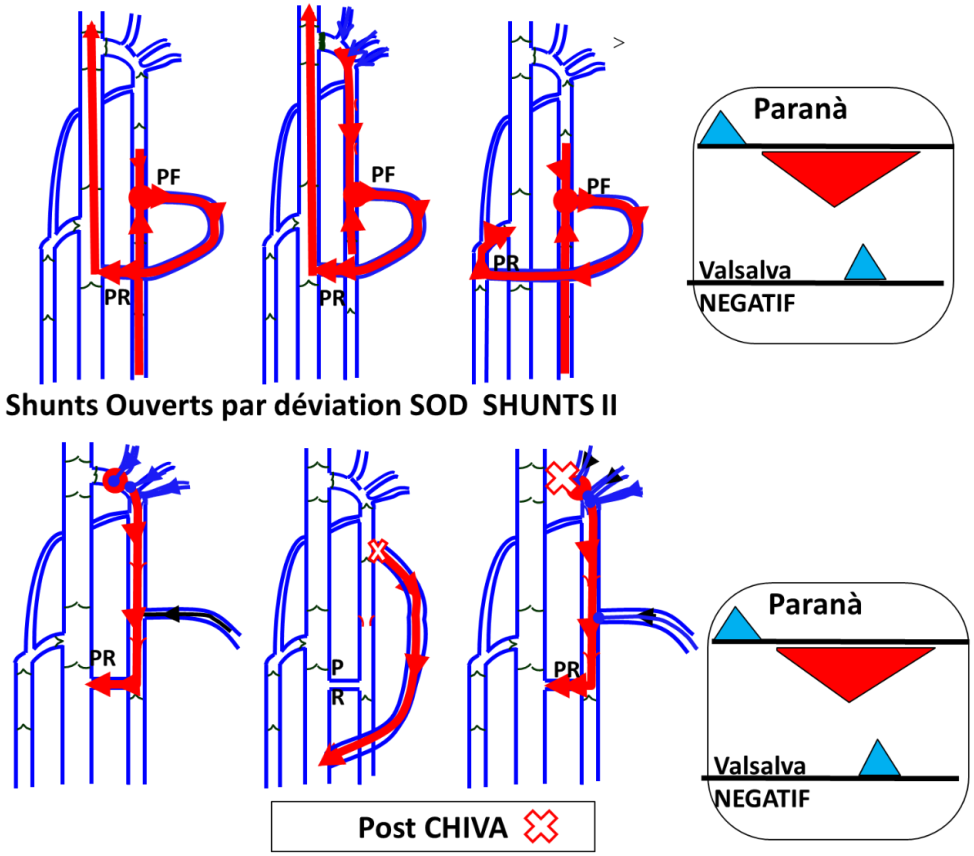


Closed SHUNT CS. Recirculation. SHUNT Type III N1>N2>N3>N1: Overloaded by N1 . 4 examples EP= Escape Point Junction Saphéno-femoaral junction. Sapheno-popliteal junction. Thigh perforator. RP= Reentry point



The reflux of the Pelvic Leaks Points during the theValvo-Muscular Pump Diastole is at of NOT SPECIFIC

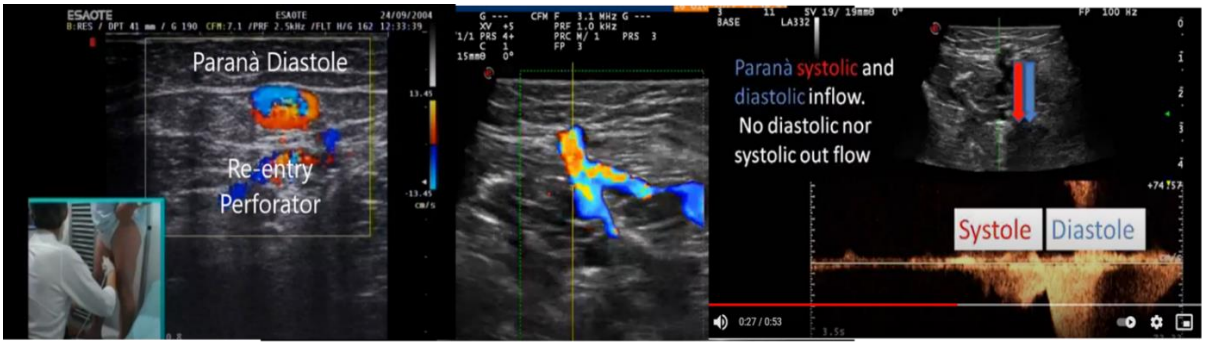
Only Valsalva + is specific



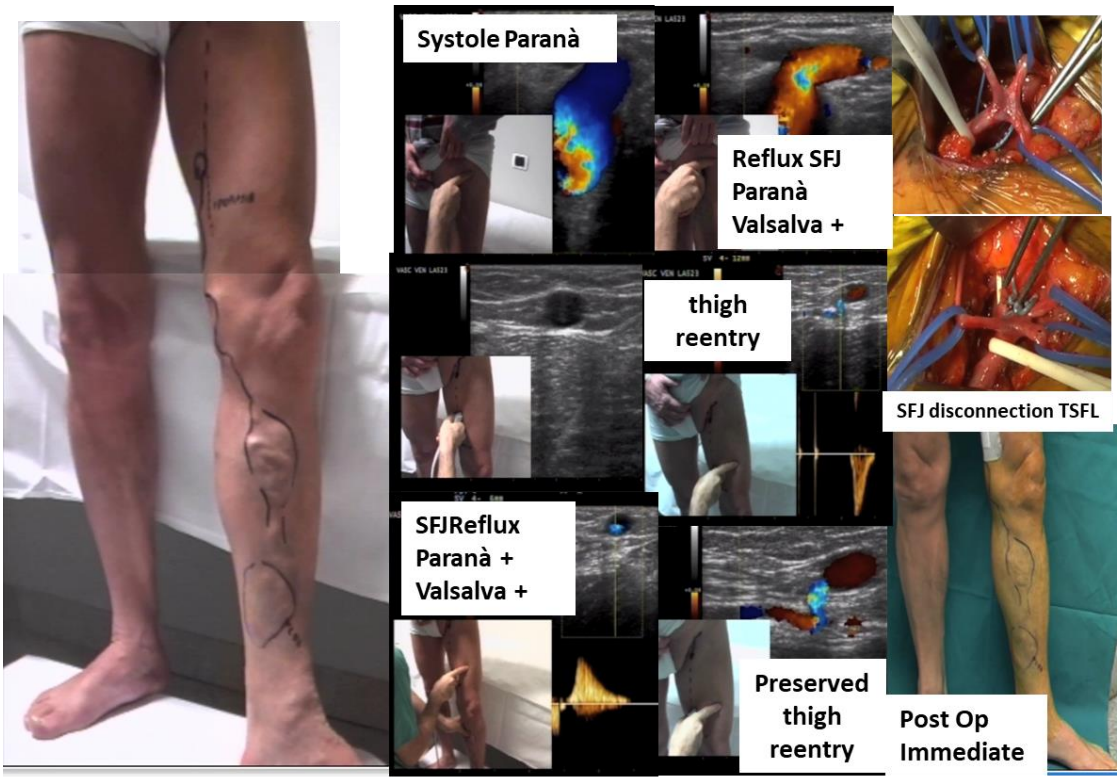
Shunts 0. Pas de point de fuite

7373233- *Re-entry perforators RP of the Great Saphenous Vein*

Re-entry perforators of the Great Saphenous Vein must be recognized in order to map the related shunt, and tailor the treatment.



Perforantes de réentrée Tronc Saphène de cuisse de cuisse (Dodd) de gros calibre mais sans reflux



The lowest re-entry perforator of closed shunts is called the <u>terminal re-entry perforator according to Massimo Cappelli</u>. It is the one that usually carries the greatest volume/pressure of reflux.

<u>The absence of an intermediate truncal re-entry perforator RP between the saphenofemoral escape point and the first refluxing tributary defines type III closed shunts</u>.

<u>This absence or presence of this perforator changes the therapeutic strategy</u>. It should therefore be sought when it is not visible at first glance by the differentiation manoeuvre Closed shunt Type I versus closed shunt Type III.

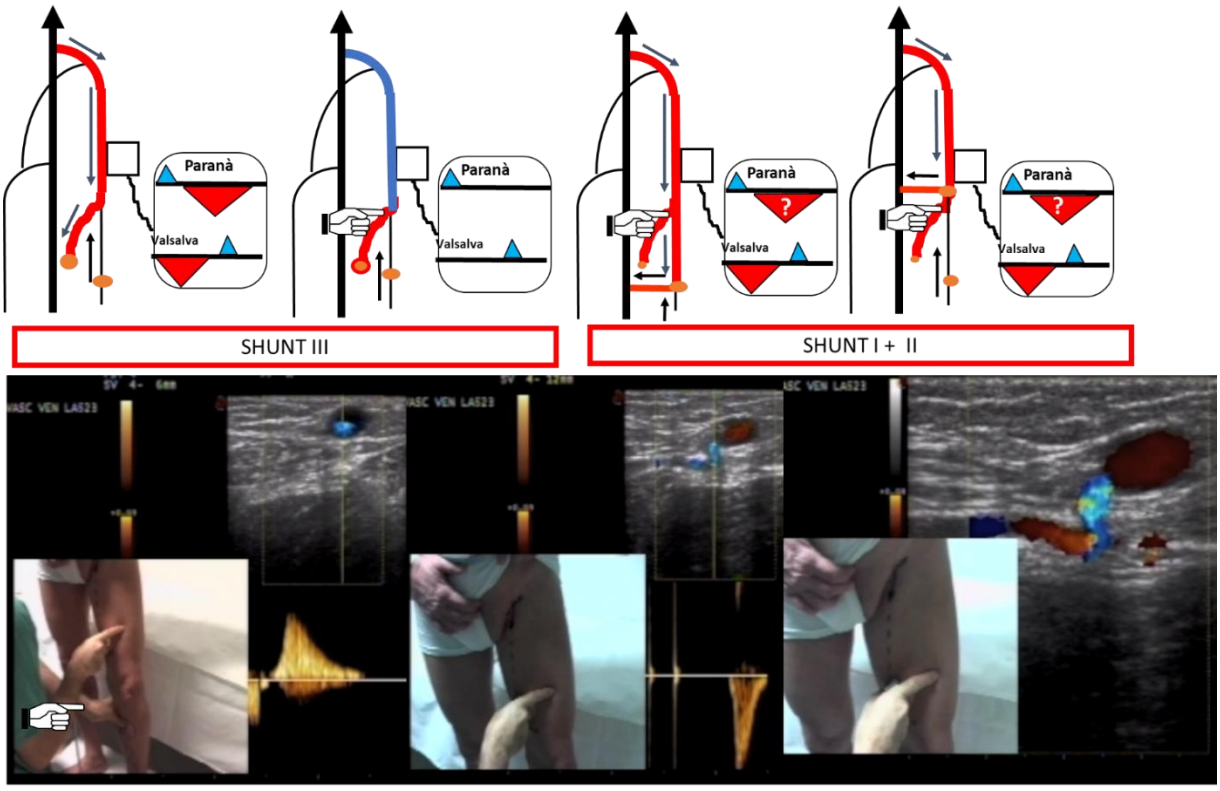
Shunt I+II vs SHUNT III differentiation test.

The Parana manoeuvre is associated with manual compression of the N3 tributary of the N2 great saphenous vein <u>to determine whether it is a type III or type I + II shunt</u>, for which the therapeutic strategies are different.

<u>In the case of a type III shunt, the reflux of the overlying trunk of the great saphenous vein N2 is suppressed</u> because there is no intermediate truncal re-entry perforator.

<u>In the case of a type I+II shunt, reflux from the overlying trunk of the great saphenous vein N2 is NOT suppressed</u> because of an intermediate re-entry perforator which can be visualized.

<u>However, this method may fail when the re-entry perforator is not activated by the muscle pump on which it depends.</u> This is sometimes the case in some patients when the thigh trunk perforator is not activated by the Parana manoeuvre or calf compression-relaxation. <u>Therefore, when the Parana manoeuvre is negative, it must always be completed, under the same conditions of compression of the refluent tributary, by the Valsalva manoeuvre which does not depend on the activity of the muscles.</u>



Differential diagnosis SHUNT III vs SHUNT II+I

Effective manual compression of N3: if diastolic reflux -Paranà or compression-release of the N2 trunk of the Great Saphenous Vein persists, a re-entrant perforator must exist on N2. When the perforator drains into the femoral, the Paranà or compression-release maneuver may not show it. The Valsalva maneuver is essential to show it.

7373234- *<u>Systolic</u> Paranà reflux N1>N2 at <u>Saphenofemoral junction SFJ</u>*

The saphenofemoral junction may be the ***escape point of an open vicarious OVS shunt in relation to a homolateral iliac obstruction.***

It is activated by the Parana manoeuvre and calf compression. It is Valsalva negative.

<u>It drains by reflux of the tributaries of the saphenofemoral junction SFJ either towards the superficial veins of the abdomen or towards the contralateral long saphenofemoral junction.</u>

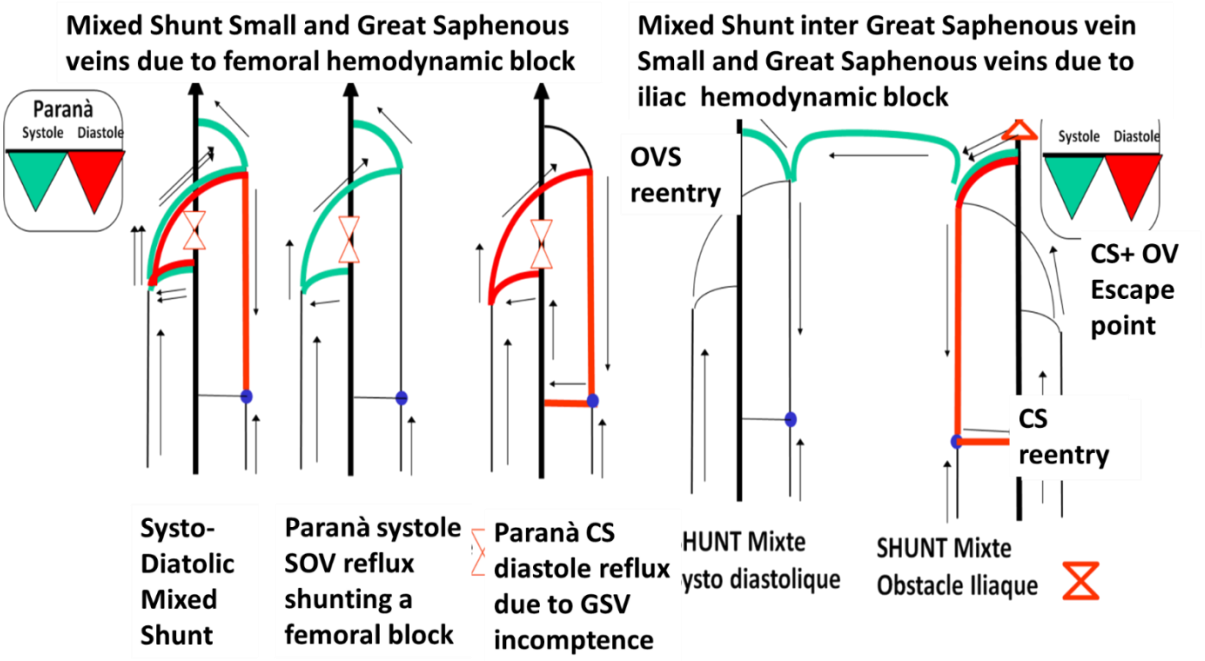
It <u>produces a "spontaneous Palma</u>" *(reference to the venous by-pass between the two saphenofemoral junction proposed by Palma) by reflux into the tributaries of the homolateral saphenofemoral junction and then a normal directional flow but overloaded in the tributaries of the contralateral saphenofemoral junction to join the contralateral femoral vein OVS N1>N2>N3>N2>N1.*

<u>It can be associated with a closed shunt CS N1>N2>N1</u> when the underlying saphenous trunk refluxes in diastole Paranà but also Valsalva +. This OVS-CS combination creates a mixed MS shunt and diastolic reflux

7373235--<u>Systolic</u> Parana reflux N1>N2 at the <u>Saphenopopliteal junction SPJ.</u>

An <u>open vicarious shunt OVS</u>, which compensates <u>a constitutional or acquired obstacle of the superficial femoral vein, made of SPJ systolic escape point reflux an SFJ re-entry</u>.Its course is usually Small saphenous arch, Giacomini's vein, trunk and junction of the Great Saphenous vein then femoral vein OVS N1>N2>N1.

If the segment of the trunk of the great saphenous vein, below its junction with Giacomini's vein, refluxes in diastole, it is a Closed Shunt CS combined with an Open vicarious Shunt OV, which constitutes a Mixed shunt MS.



7373236- Tibio-saphenous Paranà flows and Open Vicarious Shunt OVS

***The systolic reflux of this tibio-saphenous perforator** runs throughout the great saphenous vein to join the femoral vein. OVS N1>N2>N1.*

***Any open vicarious OVS shunt is evidence of an obstacle which it compensates**. The degree of hemodynamic compensation is measured by **Doppler pressure measurement** of the posterior tibial vein. In addition, this measurement allows the respective share of OVS and CS to be assessed and the therapeutic consequences to be drawn when the two types of shunts are associated.*

However, most tibio-saphenous systolic reflux are benign when they are short and/or followed by a greater re-enter diastolic flow.

7373237-Pulsed saphenous flow

The saphenous flow can be pulsed, synchronous with the heart. This pulsation has 3 possible causes.

73732371-Retrograde pulsating flow due to reflux of the tricuspid heart valve.

73732372-Anterograde pulsed flow due to a decrease in arteriolo-capillary resistance: inflammation of the leg tissues or Arteriovenous fistula AVF

73732373-Anterograde pulsed flow by flow resistance: downstream venous obstruction reduces upstream microcirculatory pressure drop so that pressure pulsatility can be transmitted.

737324-Small Saphenous Vein (formerly known as the Short Saphenous Vein).

7373241-Anatomy of the Small Saphenous Vein.

The small saphenous vein presents anatomical variations that can modify therapeutic strategies.

***Its anatomical relationship with the sciatic nerve** must be assessed to avoid any nerve accidents in the event of a surgical approach. The fact that it runs through a fascial split with its nerve should also make us fear a neurological complication in the event of surgery.*

***The SPJ saphenopopliteal junction is usually underneath the popliteal fold,** but it may be much higher or even absent.*

***The saphenopopliteal junction may be indirect via a common trunk** with the medial gastrocnemius vein.*

Its connection with a Giacomini vein is useful to evaluate to decide on its disconnection. *Indeed, disconnection of a closed or open vicarious shunt supplied by the saphenopopliteal junction is preferable just below its junction with Giacomini's vein.*

7373242-The hemodynamic function of the Small Saphenous Vein is particular.

Its flow may not be activated by the Parana manoeuvre *when it does not drain the foot. It is then produced by manual compression.*

The saphenopopliteal junction (SPJ) is frequently an escape point not only of a closed shunt (CS), *but also of a open vicarious shunt (VOS) or even of a mixed shunt (MS) when it combines a CS with a VOS.*

It is also frequently the re-entry point of various shunts*. In particular, it drains shunts fed by the pelvic escape points (Perineal point P, Superior Gluteal point GS and Inferior Gluteal point GI) via Giacomini's vein or the Sciatic vein.*

It is also often a re-entry of closed shunts fed by the saphenofemoral junction, via an anterior tributary of the Great Saphenous vein.

737325-Giacomini's vein

7373251-Anatomy of Giacomini's Vein

Giacomini's vein is a vein of the N2 network which usually joins the arch of the small Saphenous vein at a variable level of the trunk of the Grande Saphenous vein*.*

It presents variations *in calibre, proximal connection, and flow direction,*

but is always connected to the junction of the small Saphenous vein. *It inconstantly presents perforators along its course.*

7373252-Hemodynamic function of the Giacomini's Vein

Giacomini's vein drains the small saphenous vein into the great saphenous vein but *can also drain the latter into the former without pathological incidence.*

It is a compensation route for hemodynamic obstacles of the superficial femoral vein (thrombosis, hypoplasia, constitutional stenosis at the Hunter) *activated by the systole of the calf (Parana, manual compression)*

Its calibre, proximal connection and the presence of perforators on its path are considered in the hemodynamic therapeutic strategy *of open vicarious shunts OVS, closed shunts CS and mixed shunts MS, fed by a saphenous-polarity escape point.*

Here again, the notion of reversed direction = pathological reflux and anterograde flow = normal has no pathophysiological value. *Again, it is the content, source and destination of the flow that determines its normal or pathological character.*

7374-Deep veins of the lower extremities

The frequency of the echodoppler probe must be adapted to the depth of the veins, which varies greatly between patients. *The abdominal probe is often useful in the thigh.*

73741-The examination on the patient lying semi-seated allows:

*-Search for **total and partial thrombosis in the thigh and popliteal fossa**, whether they are totally or partially compressed under the pressure of the probe.*

*-Doppler assessment of orthograde flows of femoral stenoses, and **retrograde flows** of compensatory tributaries (open vicarious shunts OVS) of iliac obstacles, **notably the deep femoral and obturator veins.***

*-Doppler measurement of **posterior tibial vein pressure**, **bilateral and comparative to assess the specific hemodynamic impact of downstream obstacles***

73742-The examination on the seated patient, legs hanging off the examination bed allows

*-Search for **total and partial thrombosis in the leg and foot**, whether they are totally or partially compressed under the pressure of the probe.*

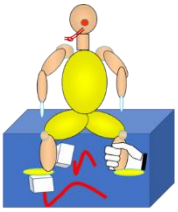
*-Doppler evaluation of the **orthograde flows and refluxes of the tibial, soleus and fibular veins** during the compression-relaxation of the foot and calf.*

*-Identify **open vicarious shunts via the tibial perforators** and then the great saphenous vein.*

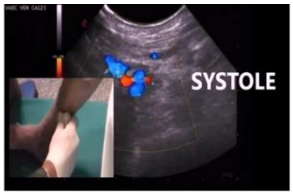
*-Assess **closed CS shunts**:*

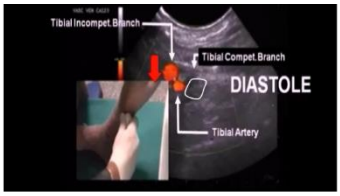
*-Double posterior tibial vein with **only one refluxing.***

*Reflux of **both** posterior tibial veins and **competent fibular veins***



Veines tibiales , surales et plantaires
Phlébites mollet et plantaire et reflux profonds et superficiels : Compression-relâchement , Valsalva





Compression-relaxation: deep Closed shunt made of a double posterior Tibial vein where one of them is incompetent Only the incompetent refluxes during the diastole.

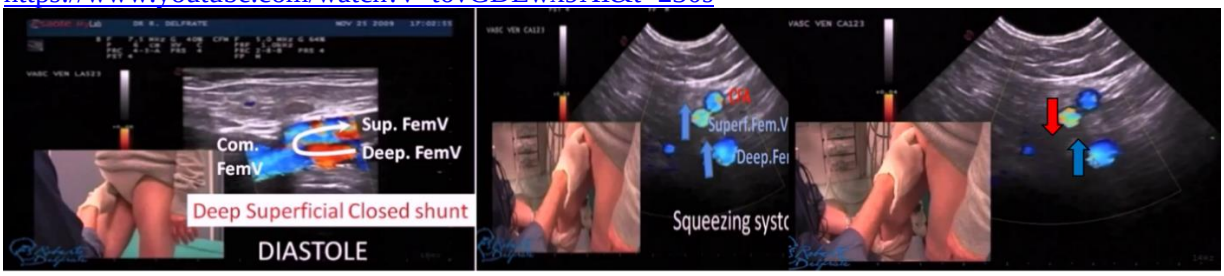
73743- The examination on the standing patient allows

*-evaluate the **degree of total, partial and segmental reflux of the deep veins***

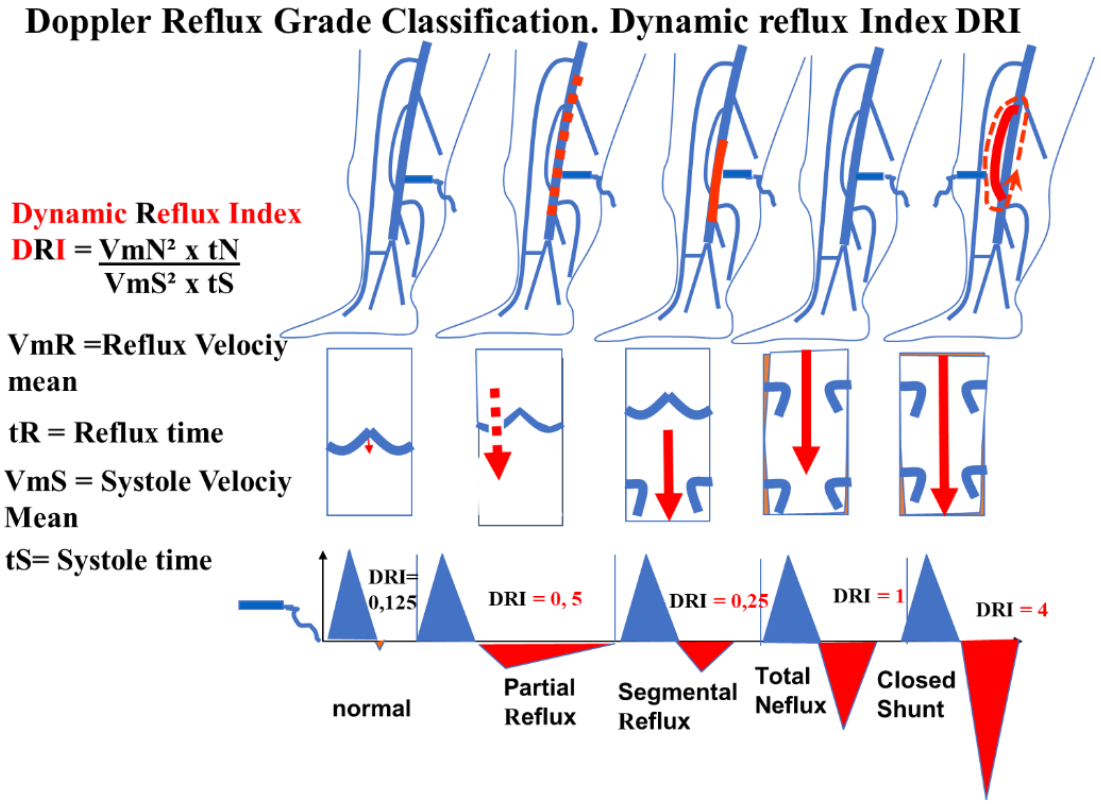
*- evaluate **deep closed shunts: CS***

*-double(duplicated) **superficial femoral vein** where only one is refluxing (Paranà manoeuvre).*

*-reflux of **the superficial femoral veins and anterograde flow of the deep femoral vein** during the Paranà manoeuvre **BUT check the quality of the popliteal-deep femoral vein connection during manual compression of the calf**. Indeed, the **Paranà manoeuvre** may activate the flow of the femoral vein by the systole of the thigh muscles alone and **does not provide information on the Popliteal Vein-Deep Femoral Vein communication which is decisive for the strategy of deep CHIVA**. **Video** Deep CHIVA:*
https://www.youtube.com/watch?v=t6vGDEwx9XI&t=230s



Deep Closed Shunt of the incontinent superficial femoral vein supplied by the deep femoral vein. The compression-relaxation of the calf shows a systolic flow in the deep femoral vein which demonstrates the drainage of the calf through a good Popliteal-Deep Femoral vein connection. This connection is critical for successful CHIVA disconnection of the superficial femoral vein. The diastole shows the reflux of the superficial femoral fed by the deep femoral.



Ref: Franceschi C. Mesures et interprétation des flux veineux lors des manœuvres de stimulation. Compressions manuelles et manœuvre de Paranà`. Indice dynamique de reflux (IDR) et indice de Psatakis. J Mal Vasc 1997;22:91–5

7375: *Venous malformations.*

The echodoppler of venous malformations specific.

The topographical variety and hemodynamic complexity require an examination that can last a long time, which favours loss of consciousness due to vagal reflex following the deflation of the cardiac pump due to the accumulation of blood in the lower extremities.

The examination time in the standing position should be short and limited to the Parana and Valsalva manoeuvres.

The rest of the time is spent in the prone position, ideally inclined at 45°, with manual compressions and Valsalva. The feet

The deep and superficial venous system from the feet to the vena cava should be examined.

Particular attention should be paid to open vicarious shunts which should be respected.

> ***Klippel Trenaunay Weber syndrome is an example, where the marginal vein plays a compensatory role in popliteal and superficial femoral hypoplasia.***
>
> ***I proposed a "compensatory test" that consists of assessing the upstream tension/ dilation of the varicose vein upstream a compression while actioning the calf muscles (pump).***

Varicose veins and venous malformations of the lower limbs: identification by echo-Doppler of varicose veins essential to the venous drainage of the limb

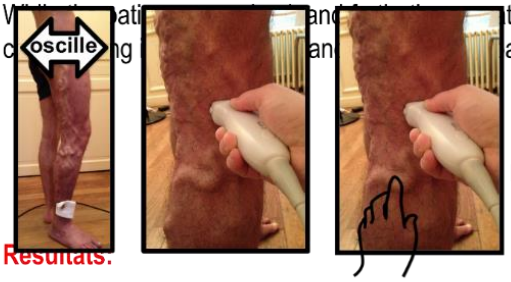
C. Laaengh Massoni, K. Betroune, C. Laurian, N. Paraskevas, A. Bisdorff
Consultation des angiomes Hopital Lariboisière Paris

Retrospective study (2012 and 2013) of 56 patients (58 limbs) explored by echo-Doppler for venous malformation with a varicose component

Objective: to identify the varicose veins essential to the venous drainage of the limb (whose surgical removal or sclerosis would cause an aggravation of the symptomatology by increasing the venous obstructive syndrome)

Method: Probes of 14, 7MHz and 3MHz. Search for agenesis, hypoplasia and incompetence of the deep and superficial trunks of the limb, the iliacs and the ICV.

Compensatory test on varicose veins proposed by C. Franceschi:



tor occludes the varicose vein by
alpation, the upstream pressure

Non suppléante: diminution

Not compensatory: pressure decreases

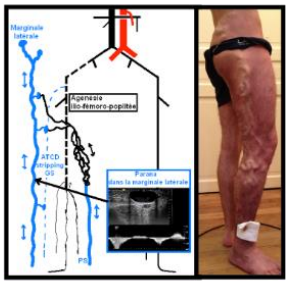
Compensatory: pressure increases

Resultats:

Varices non suppléantes	Varices suppléantes
• 36 patients (37 membres) • 20 F 16 H • 5 à 64 ans, moyenne 29 ans Territoires variqueux: • 17 saphènes • 14 marginales • 6 marginales et saphènes Atteinte profonde: • 32 membres: anévrismes, avalvulations, MV extratronculaires (infiltrant les muscles, les nerfs, interfasciales) • 1 membre: hypoplasie des fibulaires et des tibiales postérieures (tibiales antérieures suppléantes) • 3 sans atteinte profonde	• 20 patients (21 membres) • 9 F 11 H • 8 à 55 ans, moyenne 27 ans Territoires variqueux: • 10 saphènes • 3 marginales • 8 marginales et saphènes Atteinte profonde: hypoplasie ou agénésie d'un ou plusieurs étages • 1 étage: 7 membres (5 jambiers,1 poplité et 1 fémoral) • 2 étages: 8 membres (3 poplité-jambiers, 3 fémoro-jambiers, 2 fémoro-poplités) • 3 étages: 6 membres (4 fémoro-poplité-jambiers,1 ilio-fémoro-poplité et 1 VCI-ilio-fémoral)

35% of marginal vein

37% of Great Saphenous vein
(en l'absence de marginale)



Légende:
Black: deep veins
Blue: superficial veins

Conclusion

One third of limbs with venous malformation with a varicose component have compensatory varicose veins. It is important to perform a complete mapping of the network
a test of compensation to avoid provoking an obstructive syndrome by destroying these paths

A <u>distinction</u> must also be made between

***truncal** malformations which affect transmural pressure, and therefore tissue drainage, and*

***extra-truncal** malformations, which have little or no draining effect (cavernous, capillary, or venous malformations), which do not cause problems of venous insufficiency, but are responsible for iterative thrombosis and chronic pulmonary embolisms.*

These venous malformations are often associated with lymphatic malformations.

***They must be distinguished from arteriovenous malformations**, where the dilation of the veins is not malformities but secondary to the flow/pressure of the AVF, and **whose treatment is more difficult and the less favourable prognosis**.*

***Bilateral and comparative Doppler measurement of posterior tibial vein pressure** to assess the specific hemodynamic impact of downstream obstacles.*

*The echodoppler allows the establishment of **a hemodynamic map,** but also the **identification and precise marking** of non-draining malformations which can be treated surgically.*

7376-Post treatment controls.

Most controls are not hemodynamic, but simply consist of checking whether the endovenous occluded veins are recanalized or whether other varicose veins have developed after stripping.

***The hemodynamic approach is different**. It consists of **understanding the cause of recurrences and its therapeutic consequences**.*

-Recurrent varicose veins without an escape point (negative Valsalva) are compensating draining veins OVS of the occluded or stripped veins.

-Recurrent varicose veins with an escape point are Valsalva positive (V+).

*-**Negative Valsalva (V-) of Paranà positive disconnected trunks and tributaries are signs of a good hemodynamic outcome of the CHIVA cure because the reflux is simply a 0-shunt reflux, correctly draining, not overloaded**.*

***Patients must be warned** that these post CHIVA shunts 0 are normal. Indeed, **many doctors, not knowing the CHIVA cure, consider these pathological** refluxes as a failure of the treatment and propose a destructive procedure!*

7377-Topographic and hemodynamic mapping.

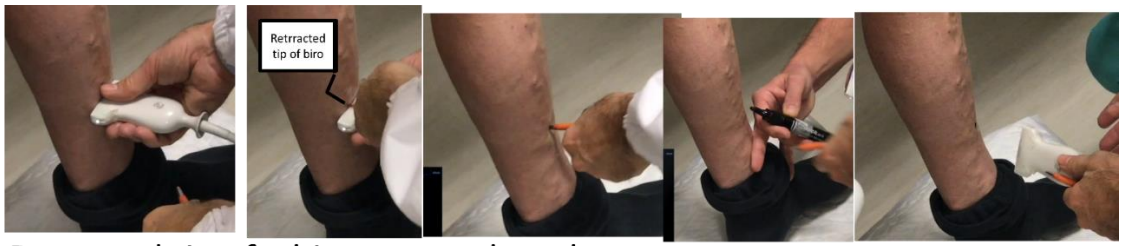
The cartography(mapping) is the essential document *for diagnosis and therapeutic strategy.*

Its realization requires *a thorough knowledge of hemodynamic physiopathology, anatomy, and the technology of echodoppler devices.*

It must provide all the ***hemodynamic features (types of shunts)*** *useful for the therapeutic strategy,* ***but also anatomo-topographic data*** *useful for the therapeutic technique relating to the approach and modes of disconnection of the escape points.*

7378- The marking of the approach points is echo guided and is done under the echodoppler probe with the retracted end of a biro taht which leaves a circle-shaped mark on the skin to be marked with indelebile ink

When pressing the pen on the skin, the tip leaves behind its small circular mark on the skin. The gel is wiped off. The mark is then made with an indelible felt-tip pen



Retracted tip of a biro, pressed on the elected point guided by echo imaging.

Marking on the circular mark made by biro

.

7379-Echodoppler by pathology

73791-Deep Vein occlusions

737911-Nutcraker syndrome NTS or Aorto-mesenteric clamp:

The left renal vein *passes through* ***a clamp formed by the superior mesenteric artery in front and the aorta behind.*** *This clamp tends to* ***close in the supine position and to open in the sitting or standing*** *position. For this reason, it should* ***be re-evaluated in a semi-seated position to avoid unnecessary NTS treatment.***

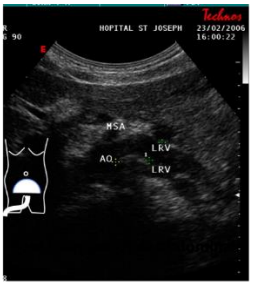
In the case *of the left renal vein* ***occlusion*** *by the aorto-mesenteric clamp,* ***in horizontal supine position, the appearance of flow in the semi-seated position corrects the diagnosis of true NTS to postural NTS***

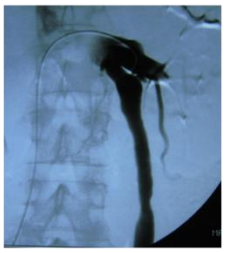
The treatment of the associated varicocele depends on its compensatory function.

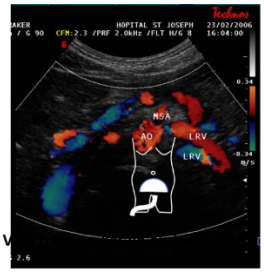
-Supine and anti-Trendelenburg Doppler, *the left renal vein drains into the iliac vein via the retrograde gonadal vein and/or into the azygos vein and/or lumbar veins.*

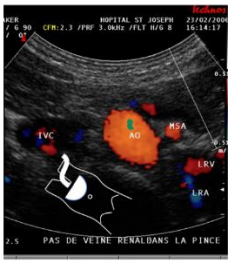
-Gonadal reflux (varicocele) is compensatory, if it persists in the-Trendelenburg position (head lower than the feet) attests for a "true hemodynamic NTS". The consequence is a necessary treatment of the aorto-mesenteric clamp before eventual varicocele embolization.

If it does not persist, the reflux is a simple open shunt ODS reflux and attests for a non-relevant hemodynamic aorto-mesenteric clamp.









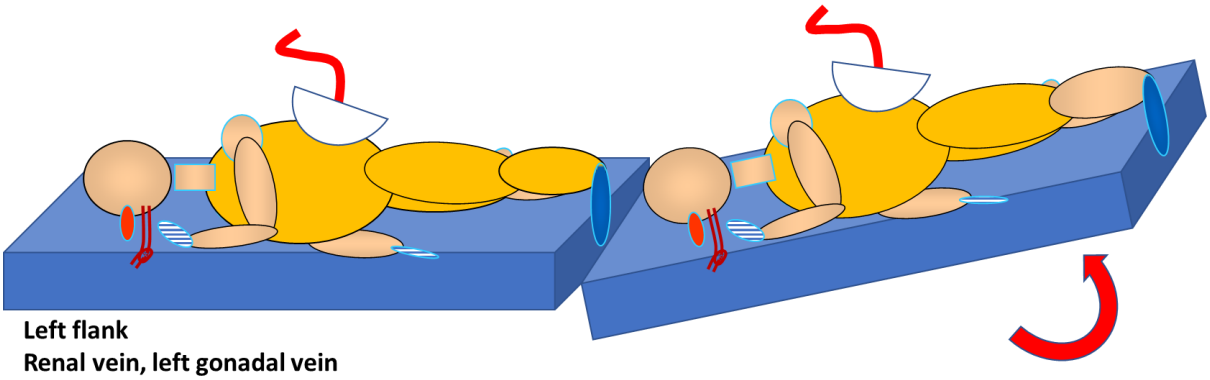
Trans-abdominal scan in Trendelenburg position.

Aorto-Mesenteric clip.
Venous flow = 0

No reno-azygo-lombar compensation
PENMANENT left ovaric vein reflux in Trendelenburg position (head lower than the feet).

Trans-abdominal scan in Trendelenburg position.

Nutcracker Syndrome: Total Aorto-Mesenteric clip
Single bypass through the left ovarian vein



Left flank
Renal vein, left gonadal vein
Varicocele, Nutcarcker: Trendelenburg test to check if the resting gonadal reflux modulated by breathing persists. If yes: NTS. If no, no NTS.

737912- *The iliac and/or cava occlusion,*

Iliac and/or cava occlusion is examined in the *supine and half-seated position.*

<u>It is indirectly recognized by a disappearance of respiratory modulation of the flow</u>

Compensation is *quantified by measuring the Doppler venous pressure at the ankle, at rest and during exercise.*

It is compensated *by various routes.*

The obturator and/or gluteal veins *usually compensate for iliac obstacles via the hypogastric (internal iliac) vein.*

The hypogastric vein *drains into the contralateral* ***hypogastric vein via the pelvic plexuses and/or the gonadal, lumbar and azygos veins.***

Superficial open vicarious shunts OVS:

-Escape points EP: *homolateral saphenofemoral junction SFJ.*

-Re-entry points: *contraleral saphenofemoral junction SFJ*

- via the tributaries of the great saphenous veins (spontaneous Palma).

-Thoracic re-entry points ***via epigastric veins and superficial abdominal varices***

737913-May Thurner MTS syndrome (also known as Cockett's syndrome).

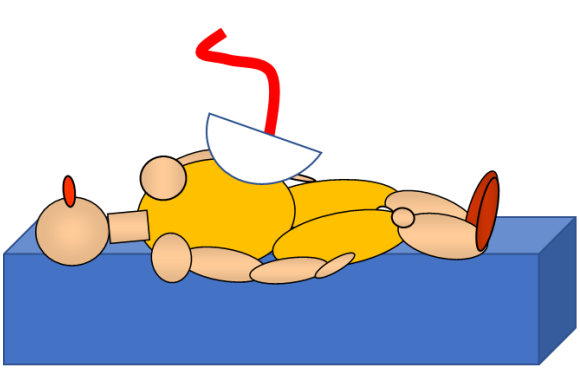
The left iliac vein *passes through a* ***clamp formed by the right iliac artery and the spine.*** *This clamp tends to* ***close when lying supine and to open when sitting or standing.***

A true MTS is a hemodynamically significant stenosis when it persists regardless of posture..

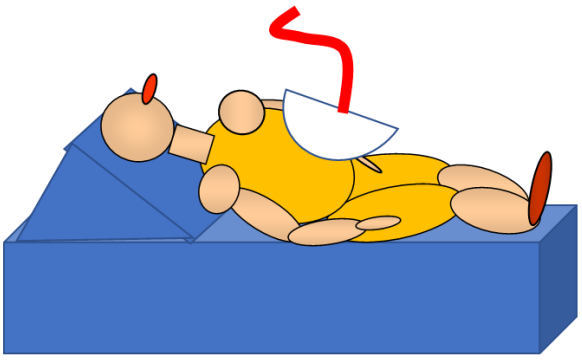
I call pseudo-MTS or postural MTS, *significant stenoses in the supine position which* ***are no longer significant in the half-sitting position.*** ***Ref:*** Paolo Zamboni, Claude Franceschi, Roberto Del Frate.The overtreatment of illusory May Thurner syndrome Veins and Lymphatics 2019; volume 8:8020.

Phlebography and echodoppler *performed only in the* ***horizontal position*** *may provide an* ***overdiagnosis*** *indicate an* ***overtreatment stenting in pseudo-MTS.*** ***This may explain the finding of "illusory" MTS assessed by horizontal supine phlebography in young asymptomatic subjects.Ref:*** van Vuuren TM, Kurstjens RLM,Wittens CHA, et al. lllusory angiographic signs of significant lliac veincompression in healthy volunteers. Eur.J Vasc Endovasc Surg 2018;56:874-9.

Similarly, true MTS, like any other iliac occlusion, *must be hemodynamically evaluated by* ***measuring the upstream venous pressure,*** *to* ***avoid unnecessary stenting of well-compensated obstacles.***



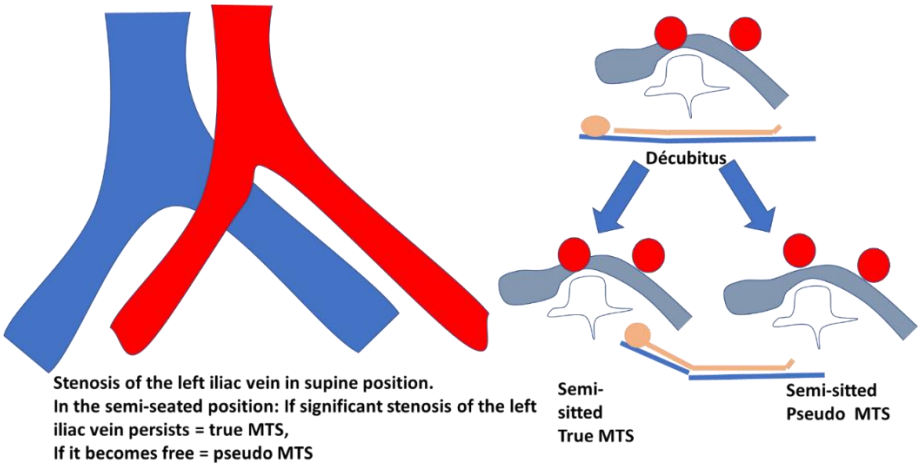
Pelvis and abdomen: Occlusion and stenosis of iliac and vena cava. Lumbar and azygos collaterals. Arterio-mesenteric clamp, left iliac vein clamp.



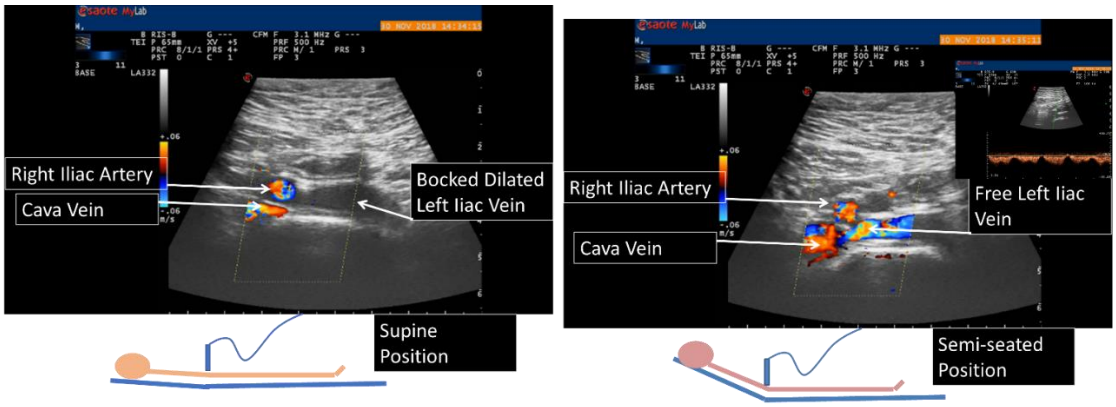
Pelvis and abdomen: If renal vein stenosis by aorto-mesenteric clamp or left iliac vein occlusion in decubitus, check in semi-sitting potion if it is not postural stenosis, usual artifact of MRI, and Phlebography.

Direct diagnosis of May Thurner syndrome MTS (or Cockett) and pseudo-MTS et du Nutcracker syndrome NTS

Similarly, <u>the Nutcracker syndrome NTS may be artefactual in the presence of a varicocele considered to be compensatory for a stenosis of the left renal vein</u>. The disappearance of the reflux of the ovarian vein in the Trendelenburg supine position (head lower than the feet) on the echodoppler proves its absence of compensatory effect, whereas its permanence confirms it.



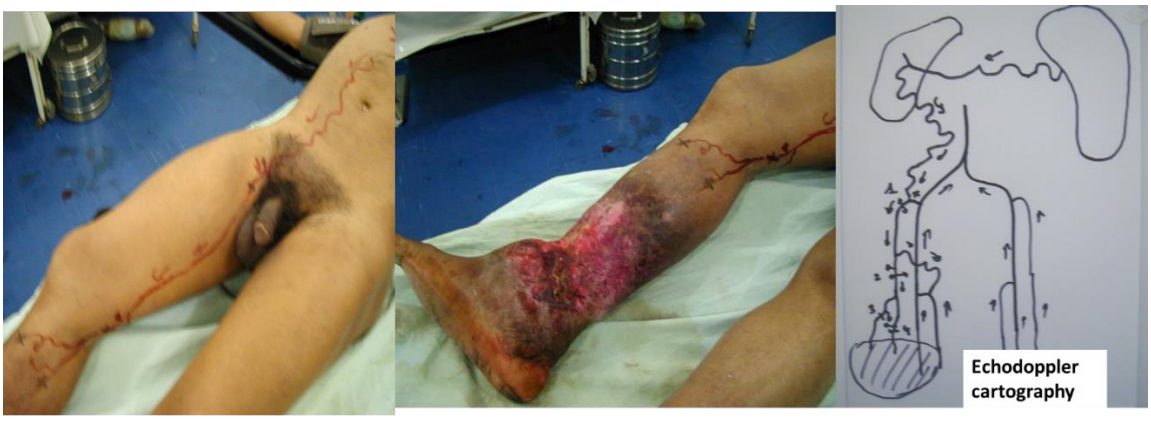
Stenosis of the left iliac vein in supine position.
In the semi-seated position: If significant stenosis of the left iliac vein persists = true MTS,
If it becomes free = pseudo MTS



May Thurner Syndrome at phlebography but
Pseudo May Thurner Syndrome on Echodoppler: only postural

737914-Occlusion of the portal vein

Occlusion of the portal vein can cause an open vicarious shunt (OVS) via the umbilical vein of Cruveilhier (point of escape) and the saphenofemoral junction (point of re-entry). Thanks to the echodoppler, I was able to follow the refluxing superficial vein from the saphenous arch up to the umbilical then to the liver, portal vein and spleen. So, I found the cause of the ulcer partly overloaded by portal blood! See below the mapping. The SVO was associated with a closed Great Saphenous vein shunt, in a mixed shunt. We disconnected the portion of the closed shunt below the SVO re-entry to heal the ulcer while respecting the Cruveilhier compensation.



"Portal ulcer" . Great Saphenous Vein reflux fed via the umbilical vein of Cruvelier by portal blood due to the occluded portal vein (Banty syndrome)

737915--*Common femoral Vein occlusion*

Common femoral occlusion is compensated by the deep femoral vein via the deep tributaries of the hypogastric vein (obturator and/or gluteal veins)

<u>Compensation by the OVS is quantified by measuring Doppler venous pressure</u> at the ankle, at rest and during exercise

737916-Superficial femoral vein occlusion

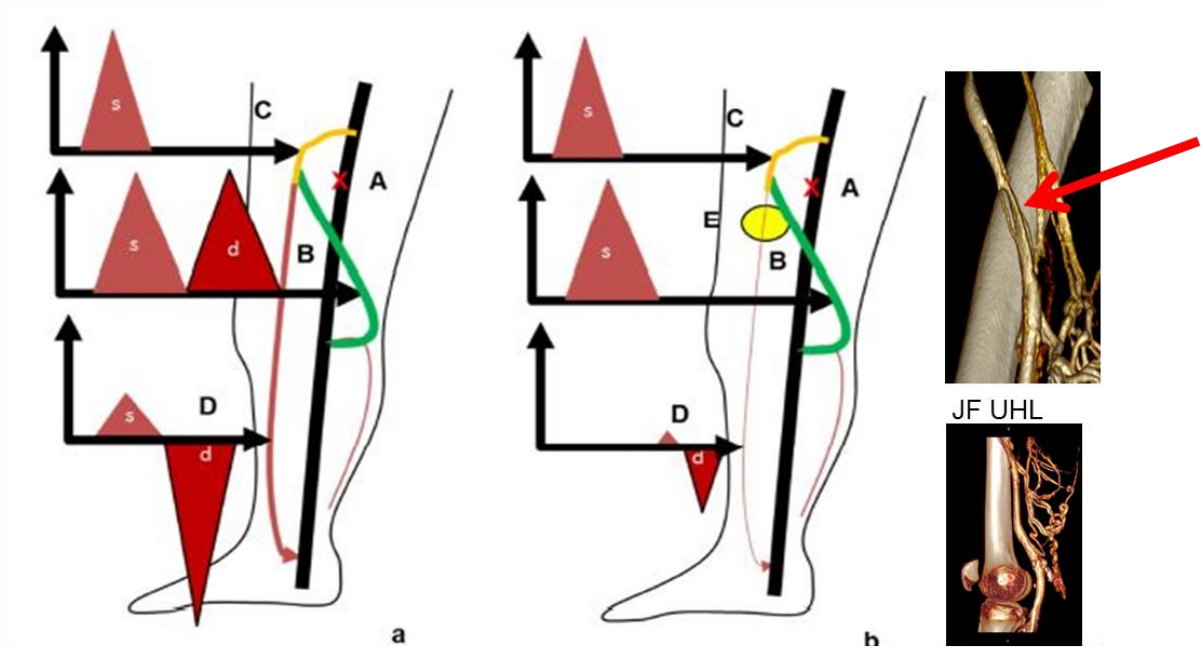
It is compensated by the OVS made of by

-the popliteal vein via the deep femoral vein when the popliteal vein drains via the deep femoral vein and/or

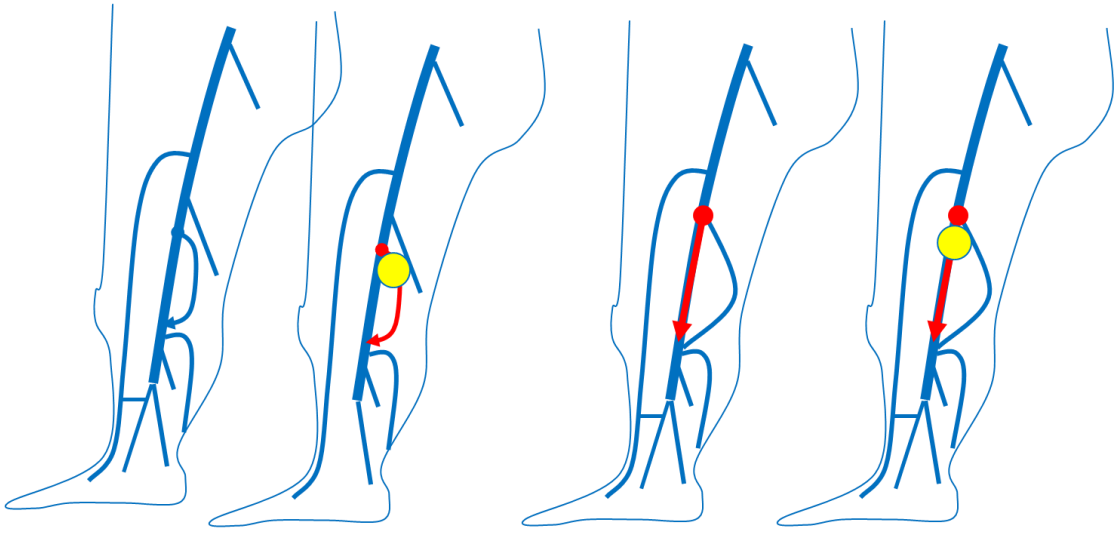
-the great saphenous vein via tibial perforators and/or

-the small saphenous vein via Giacomini's vein

This OVS *compensation* ***<u>is quantified</u>*** *<u>by measuring</u>* ***<u>Doppler venous pressure at the ankle</u>****, <u>at rest and during exercise</u>.*



Obstacle of the Superficial Femoral Vein diagnosed hemodynamically by echodoppler that I called vicarious open shunts and mixed shunts and treated by CHIVA before anatomical confirmation by Dr JF UHL



Double superficial femoral vein with an incontinent collateral is a closed shunt corrected by CHIVA	Incontinent superficial femoral vein and continuous deep femoral vein connected to the popliteal vein is a closed shunt corrected by CHIVA

The variability of the anatomy of the femoral veins is crucial for the treatment of venous insufficiency

737917- *The popliteal occlusion is bypassed by*

-deep collaterals,

-the great saphenous vein via tibial perforators

-the small saphenous vein via Giacomini's vein

737918-Occlusion of the tibial, soleus, gastrocnemius veins ***are usually*** *rapidly compensated* ***for due to their*** *large number and duplication.*

73792-Deep venous incompetence

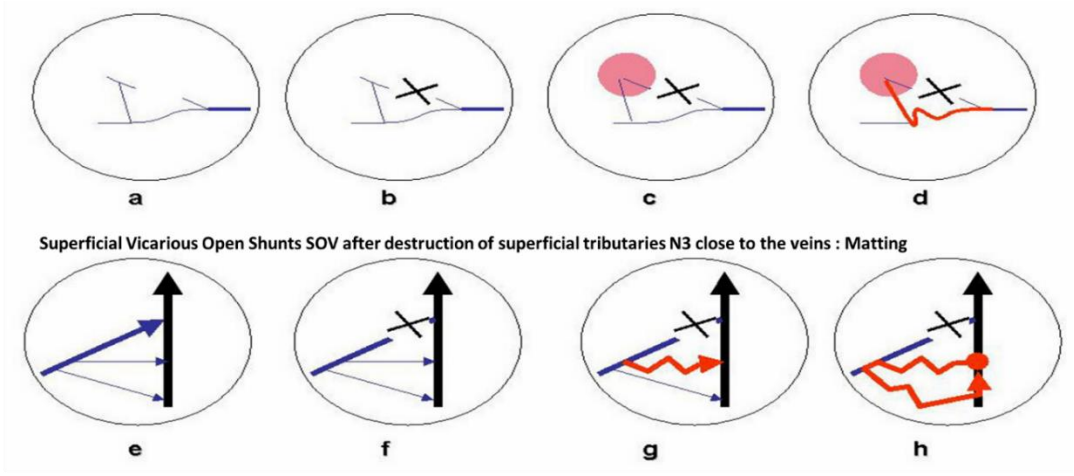
Pelvic, femoropopliteal and leg refluxes may be treated by CHIVA strategy when they combine deep Closed Shunts with or without OVS t VIDEO: deep CHIVA

https://www.youtube.com/watch?v=t6vGDEwx9XI&t=230s

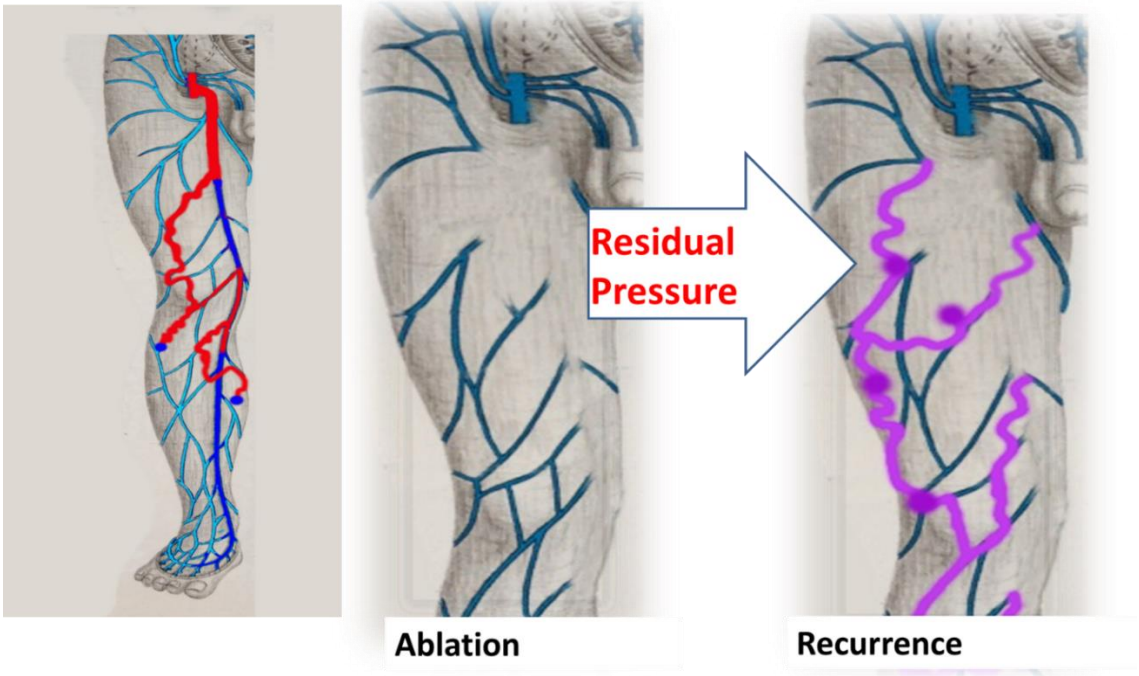
73793-superficialvenous occlusions

Except for thrombosis and by-pass surgery, <u>most occlusions are "therapeutic</u>" (stripping, phlebectomies, laser, glue, sclerosis etc.)

Under the effect of the residual pressure which increases upstream, varicosities, matting and varicose veins develop.



Superficial Vicarious Open Shunts SOV after destruction of superficial tributaries N3 distant from the venules: varicose recurrences



73794- *Mapping (cartography)*

Topographic and hemodynamic mapping is mandatory for CHIVA treatment.

It is simplistic *to considers that any* ***vein carrying a reflux must be sacrificed****, as is still too often the case, despite the* ***high rate of recurrences and the loss of venous capital*** *for subsequent vital by-pass surgery!*

To achieve a ***hemodynamic and conservative treatment with fewer recurrences, such as the CHIVA cure,*** *the mapping must show the* ***escape points, routes and re-entries of the closed CS shunts, open deviated shunts, open vicarious OVS and mixed deep and superficial.***

To do this, the practitioner must have ***knowledge of both hemodynamic and venous pathology.***

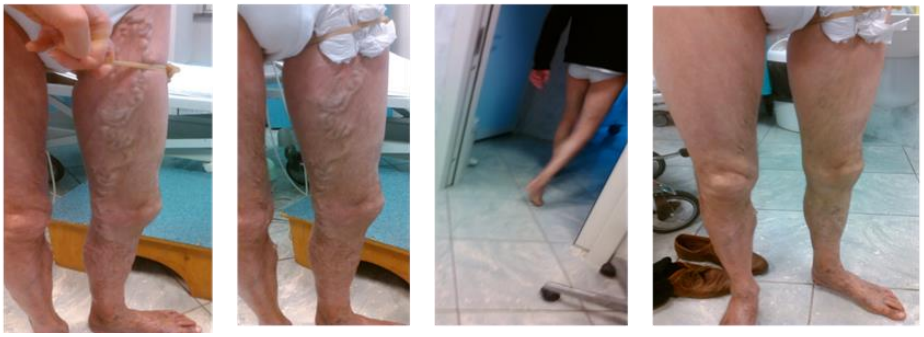
737941- Superficial Mapping

Below, the superficial cartographies (mappings) regard ***the various types of shunts and show the strategic consequences of CHIVA treatment,*** *namely the* ***sites of disconnection and devalvulation.***

It should be noted that the less the disconnections the best medium- long term results. ***Overdoing it (for fear of insufficient collapse of the varicose veins) exposes unfavorable conditions for drainage that cannot be corrected. Thus, the fewer disconnections and the greater the patience of the surgeon and the patient, the better the results of CHIVA.***

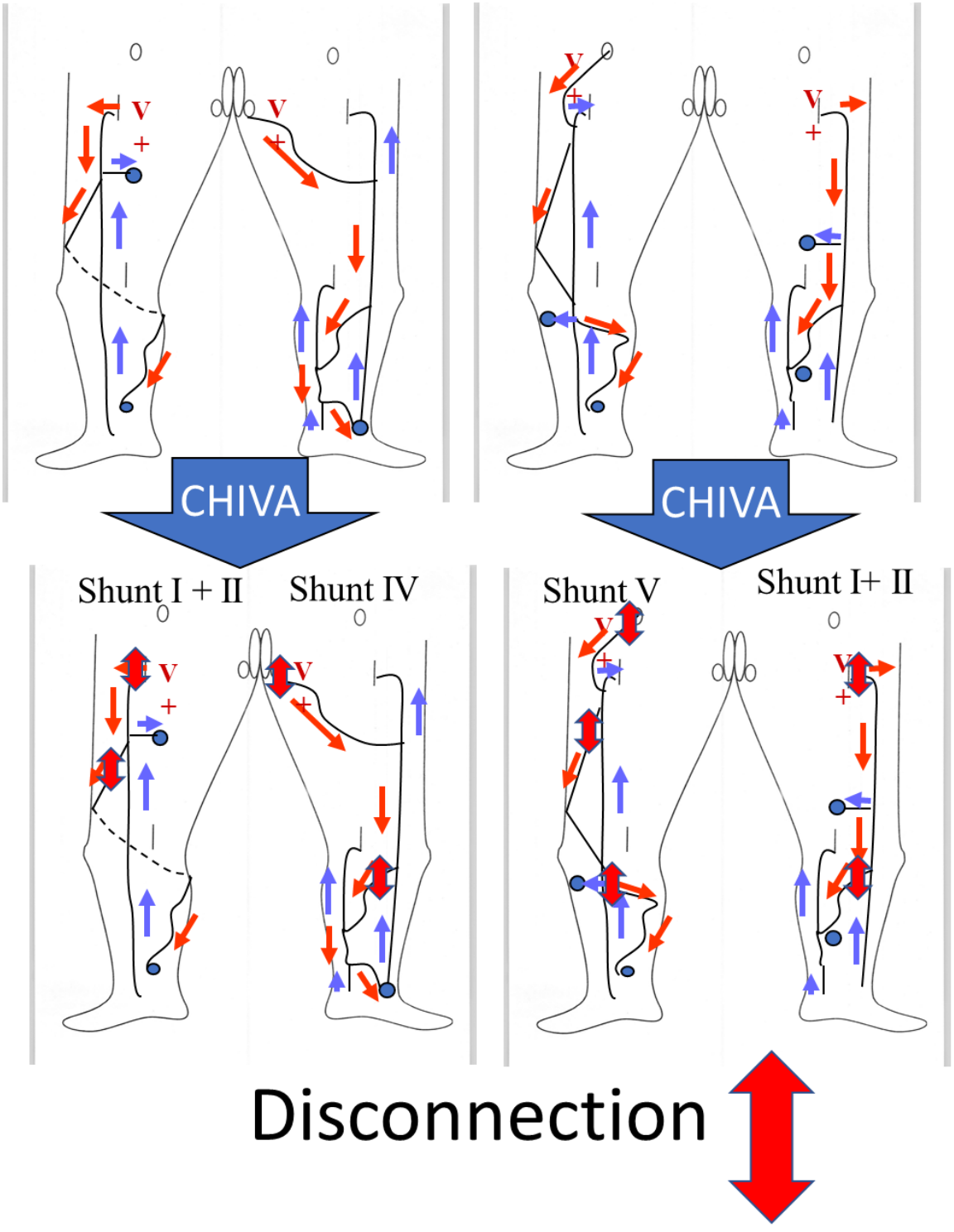
On the other hand, it is easier to complete a disconnection forgotten at first*! Avoid this particularly using the Perthes test that will show "unexpected" dramatic collapse of great clusters of varicose veins below just one point****. The patient notes it as yourself. Just explain him that it is more prudent to risk a lack of disconnection of a single point easy to treat that to perform an excessive one that will not essay at all to correct!*

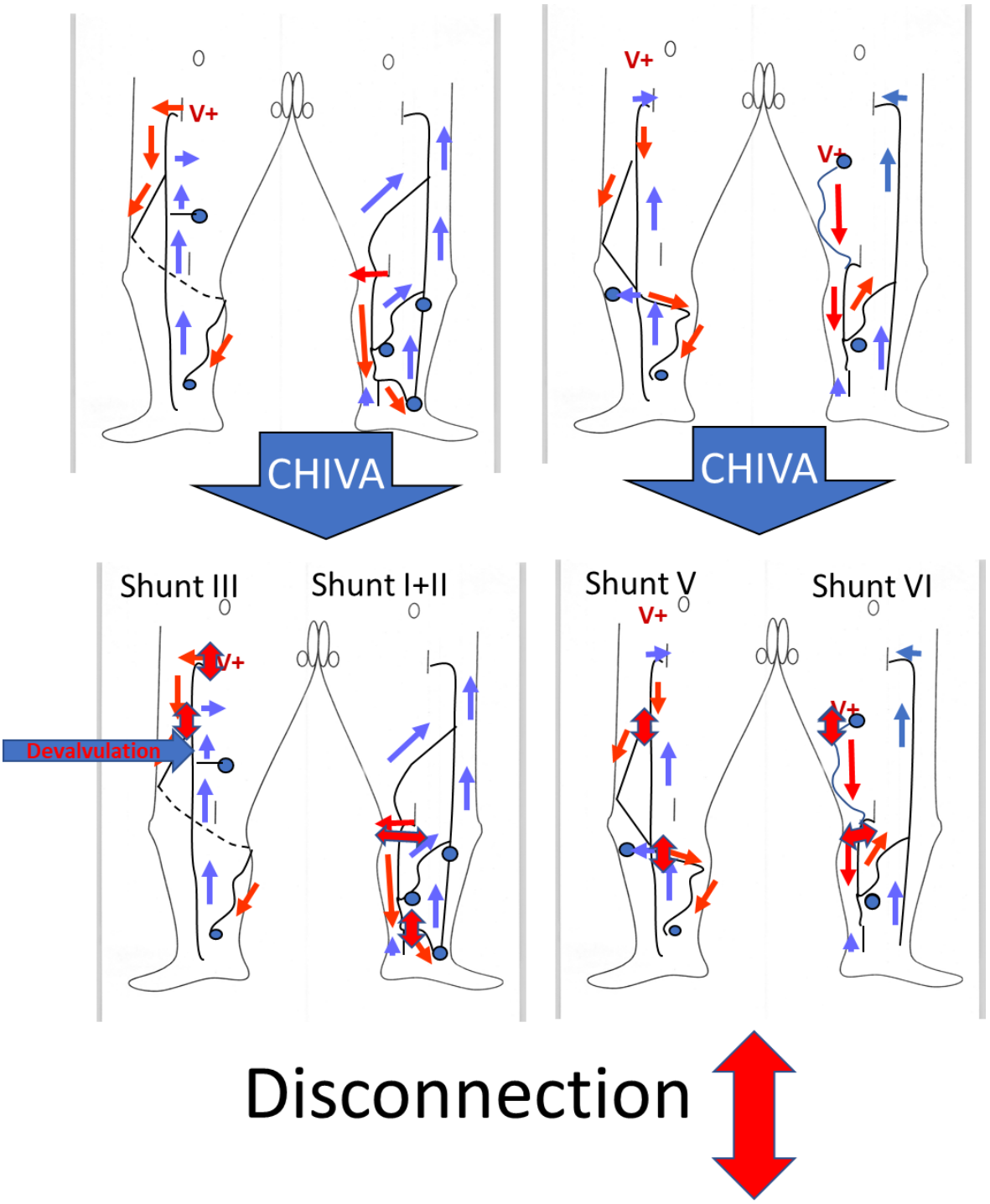
Clinical maneuvers:Perthes Manoeuver checks the deep venous system status according to the varicose veins collapse during walking with a thigh tourniquet and estimates the clinical outcoms of CHIVA disconnection without any venous ablation

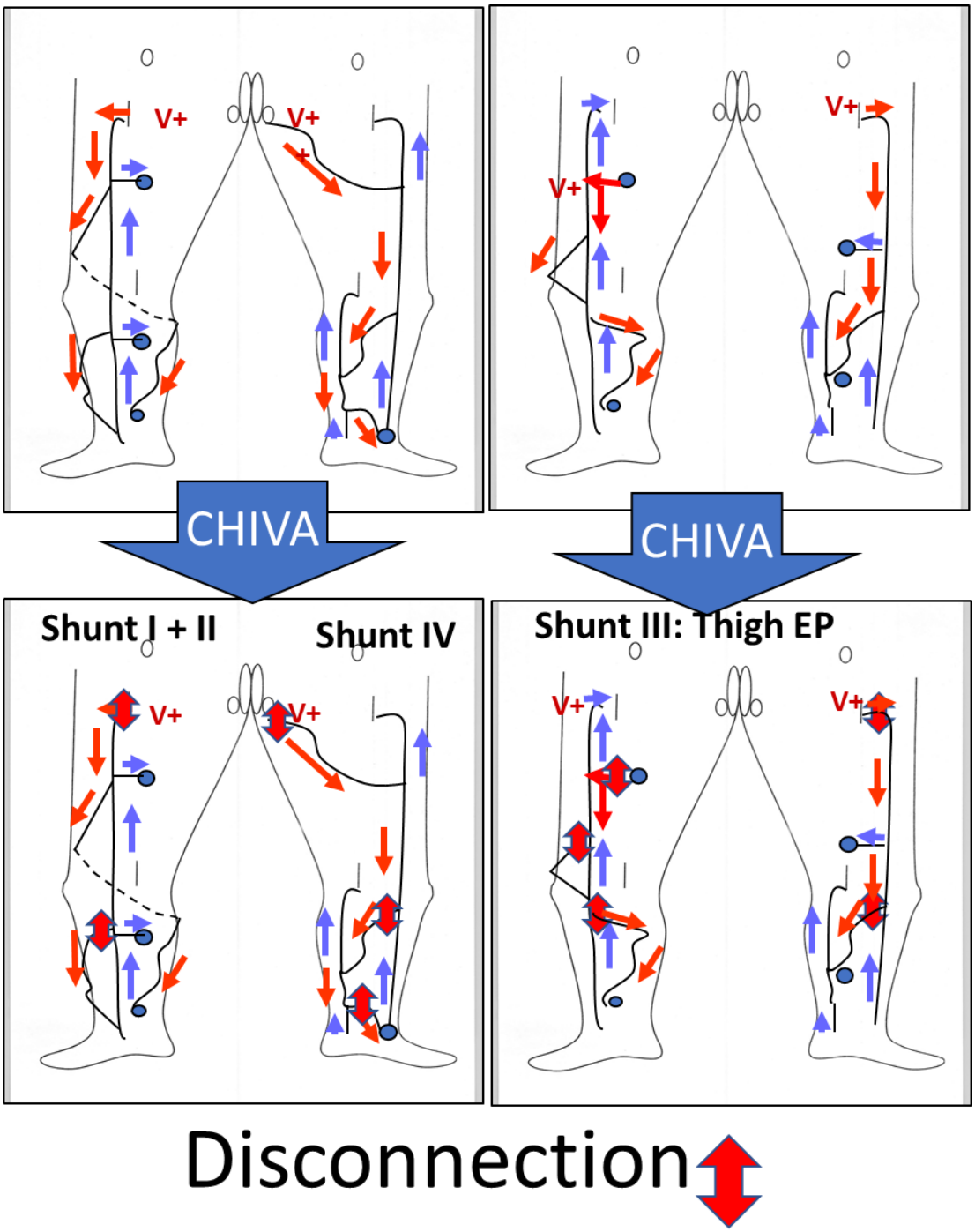


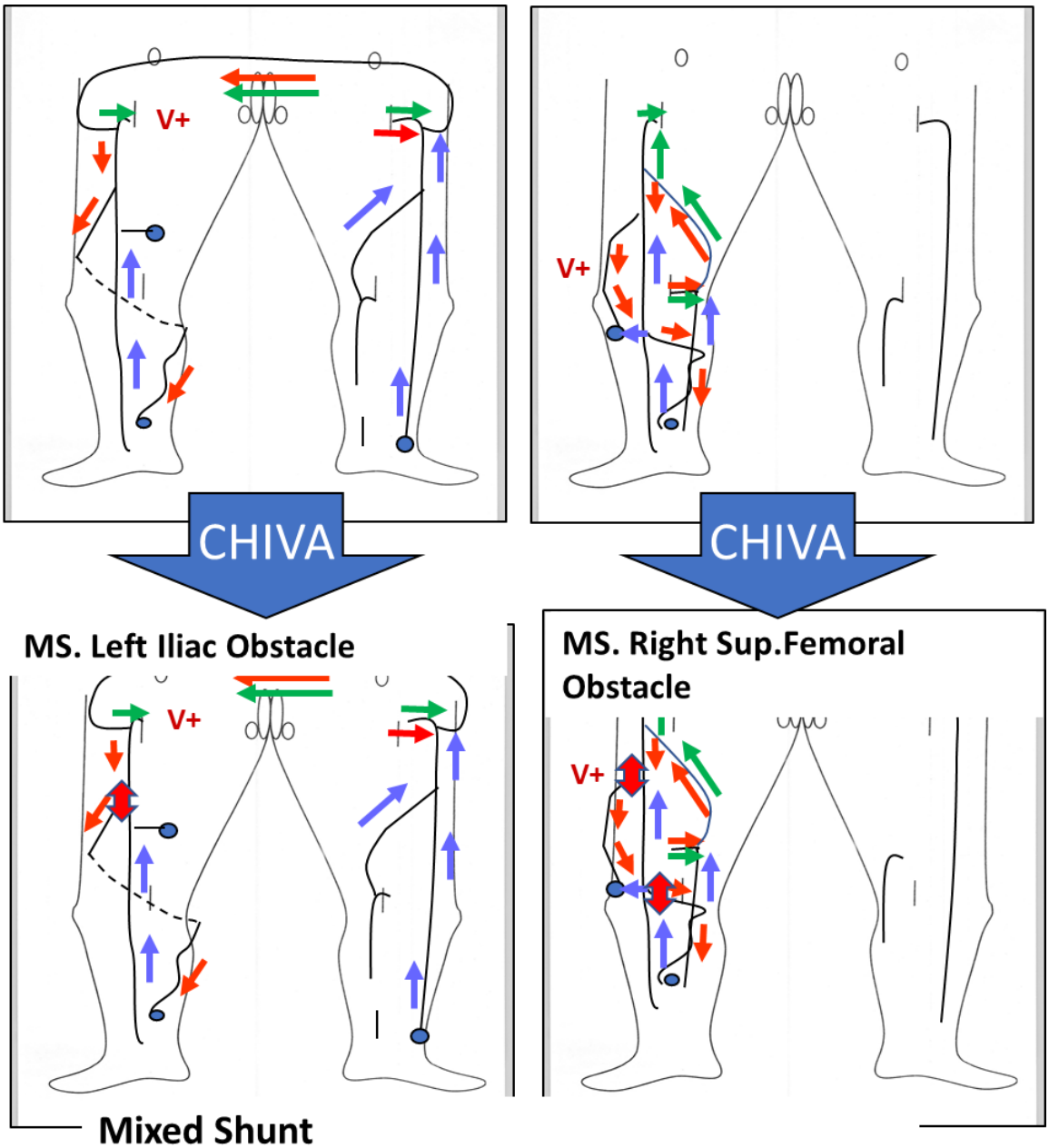
We will see the therapeutic tactics, namely the ***techniques of disconnection and devalvulation essential to avoid recurrence by angiogenesis.***

You will see below various mappings and their specific points to be disconnected. They are a mix of examples provided by CHIVA experts over the world that show the homogeneity of the CHIVA approach.

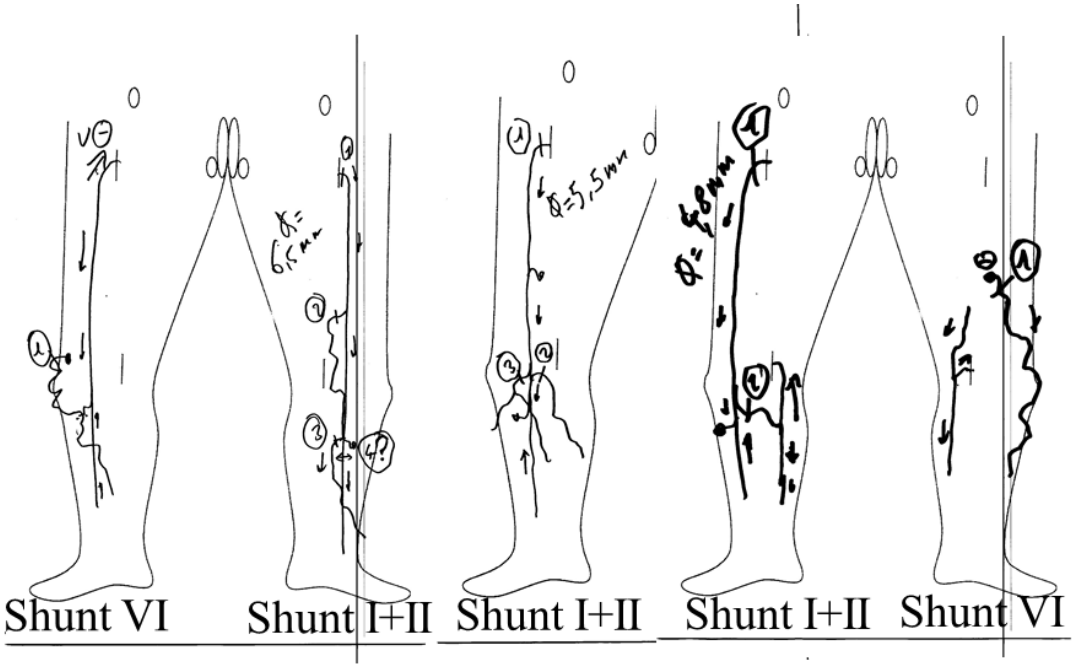

V
+
V
+
CHIVA
V
+
V
+
CHIVA
Shunt I + II
Shunt IV
Shunt V
Shunt I+ II
Disconnection

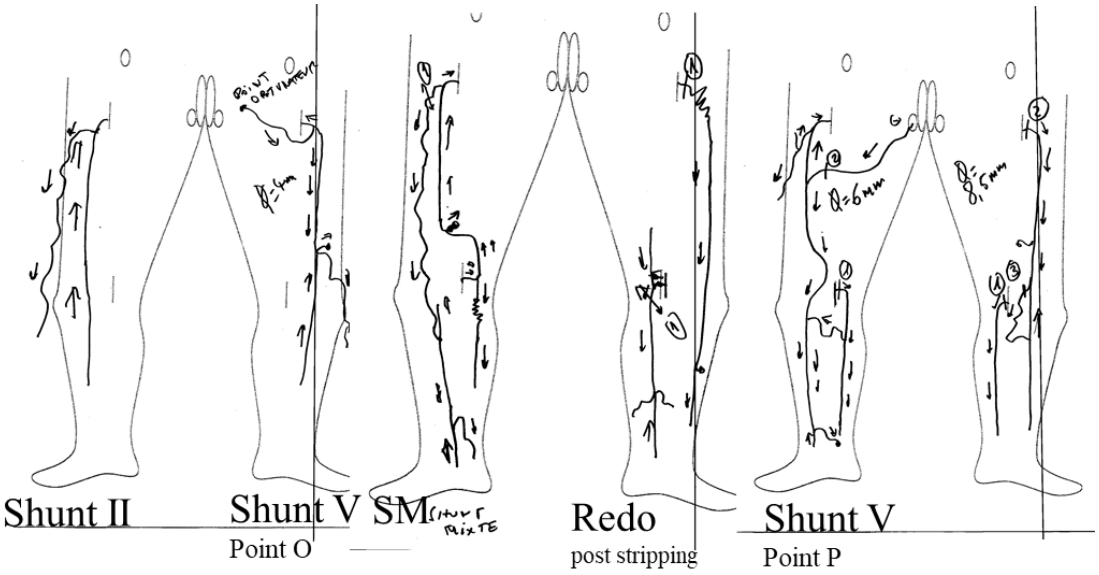

V+
CHIVA
V+
V+
CHIVA
Shunt III
Shunt I+II
V+
Devalvulation
Shunt V
V+
Shunt VI
V+
Disconnection

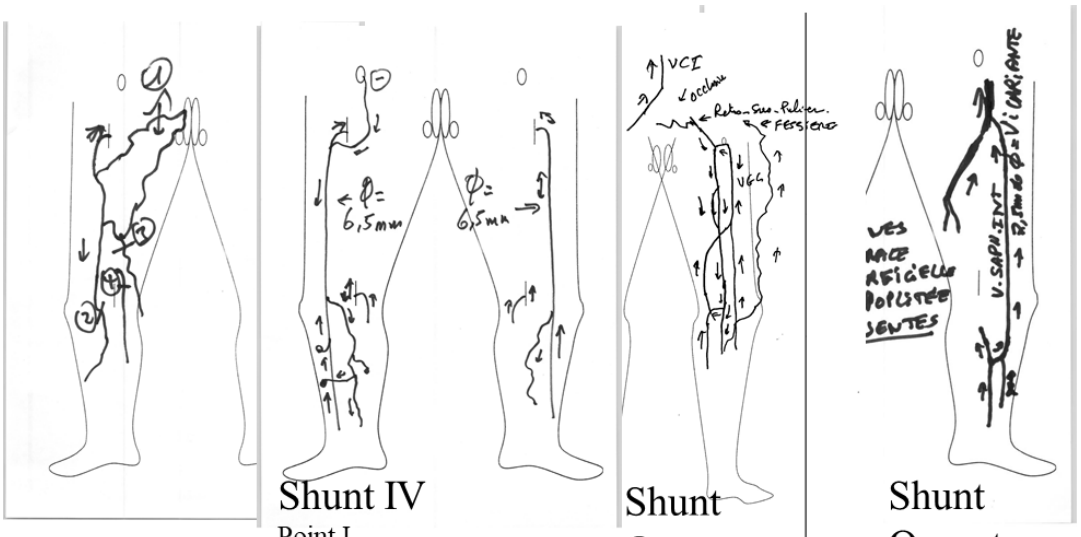

V+
V+
V+
V+
CHIVA
CHIVA
Shunt I + II
Shunt IV
Shunt III: Thigh EP
V+
V+
V+
V+
Disconnection



Some personal daily cases: Mapping + CHIVA strategy


Shunt VI
Shunt I+II
Shunt I+II
Shunt I+II
Shunt VI

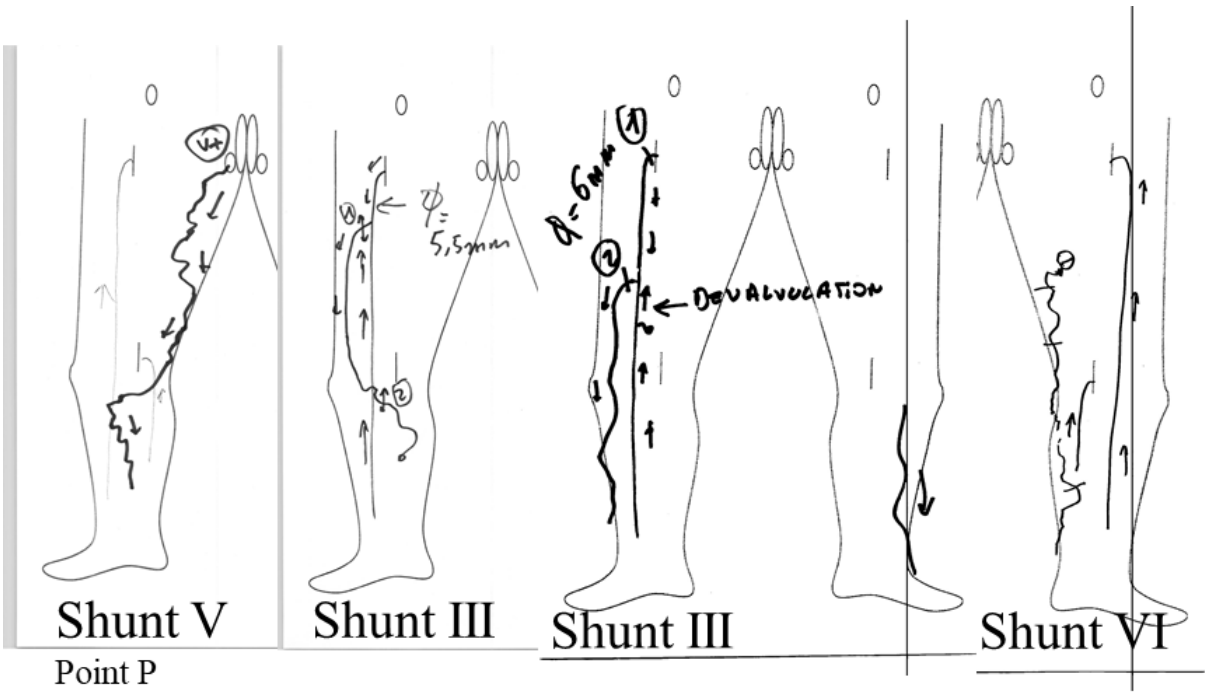

Shunt II
Shunt V
Point O
Redo
post stripping
Shunt V
Point P



Shunt IV
Point I

Shunt
Ouvert
Vicariant

Shunt
Ouvert
Vicariant

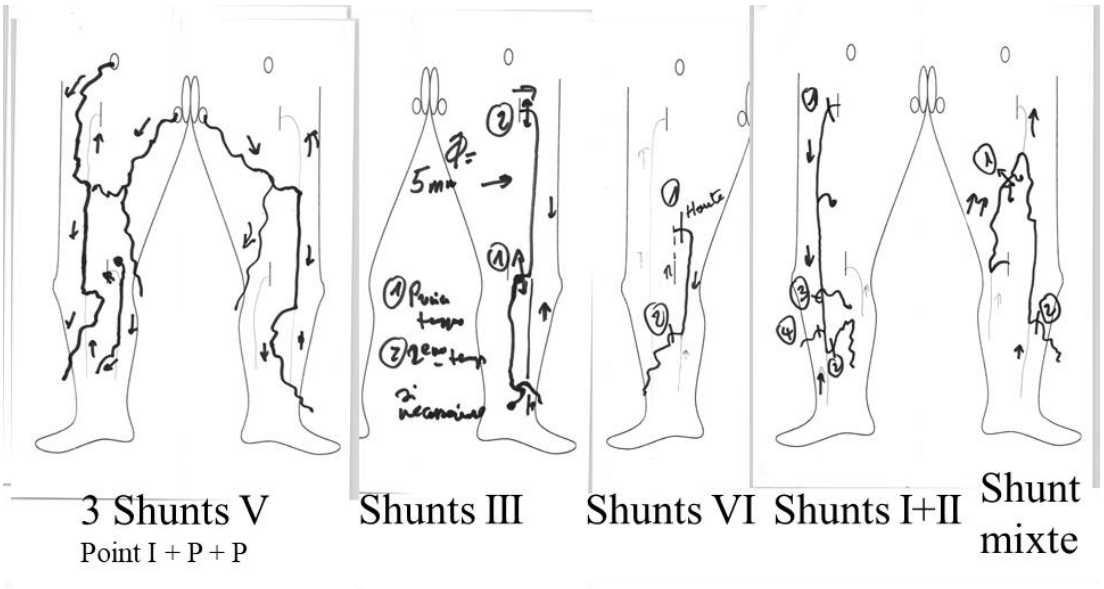


Shunt V
Point P

Shunt III

Shunt III

Shunt VI



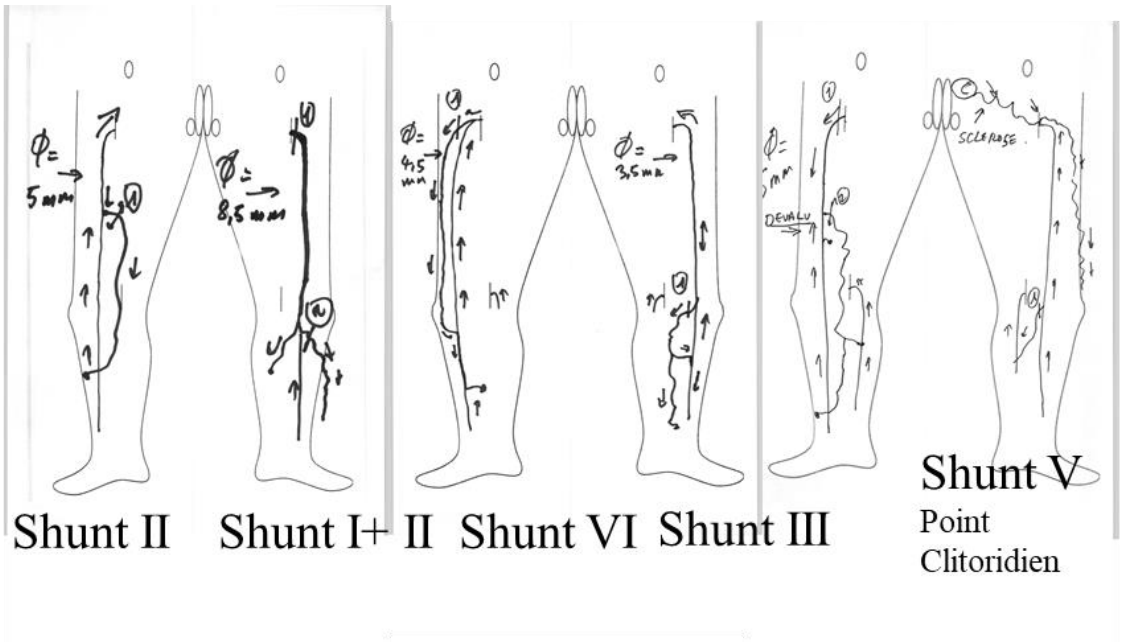
3 Shunts V
Point I + P + P

Shunts III

Shunts VI

Shunts I+II

Shunt mixte



Shunt II

Shunt I+ II

Shunt VI

Shunt III

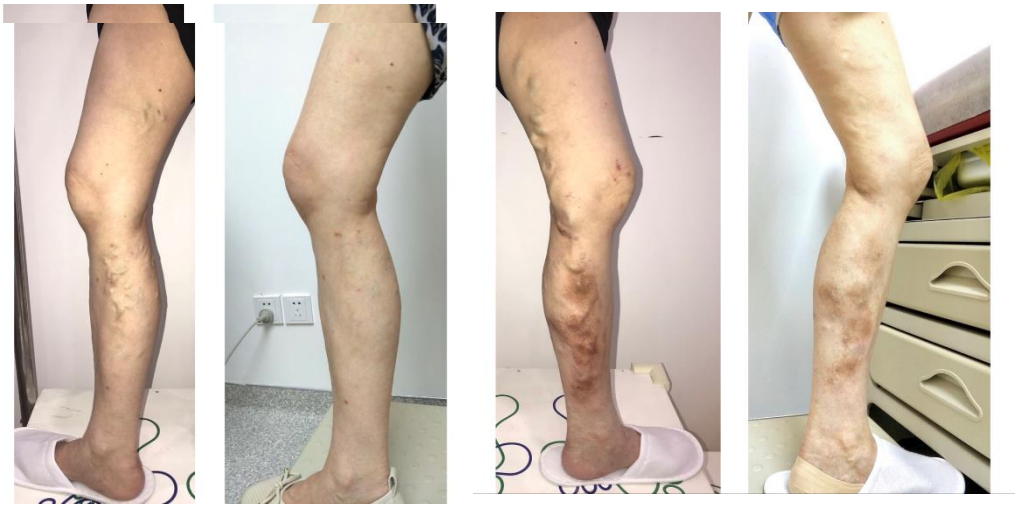
Shunt V
Point Clitoridien

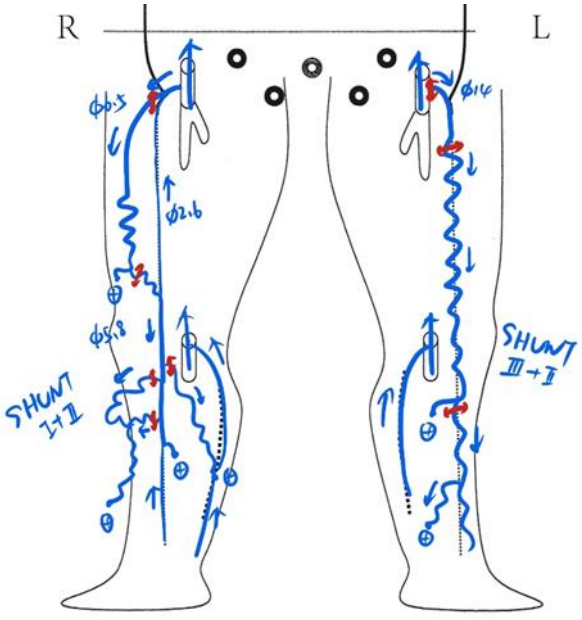
9 CHIVA CASES

Dr Sophie ZUH Dr Smile Group Shanghai

CASE 1

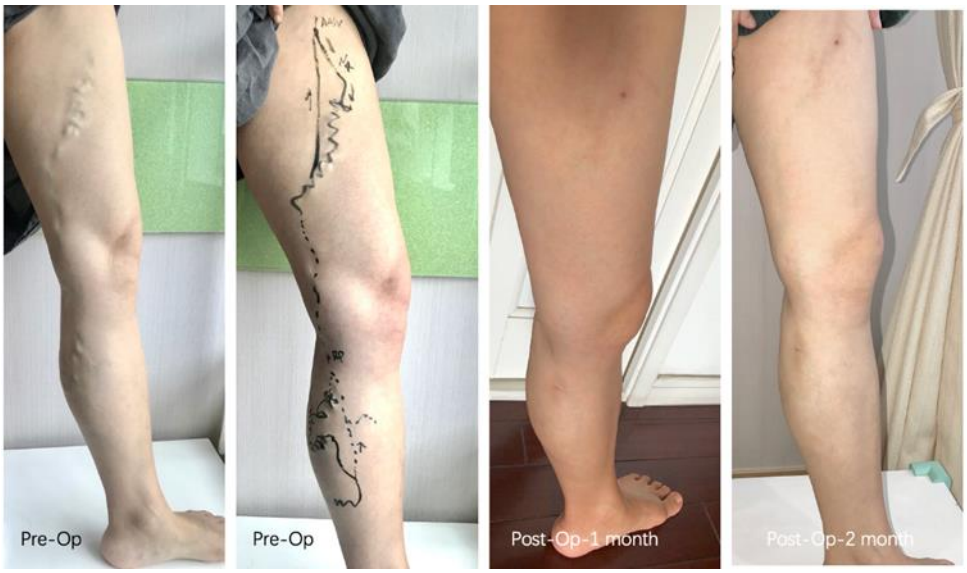
A 70-year-old female Presented with varicose veins in both legs for over 10 years. She had chronic eczema and skin pigmentation of the left calf. No DVT history. Perthes test showed varicose veins totally collapsed. *Hemodynamic ultrasound showed SHUNT 3 in left and SHUNT I+II in right. CHIVA was performed at both legs in one surgery. 10 months follow-up showed varicose veins collapsed and skin improved.*

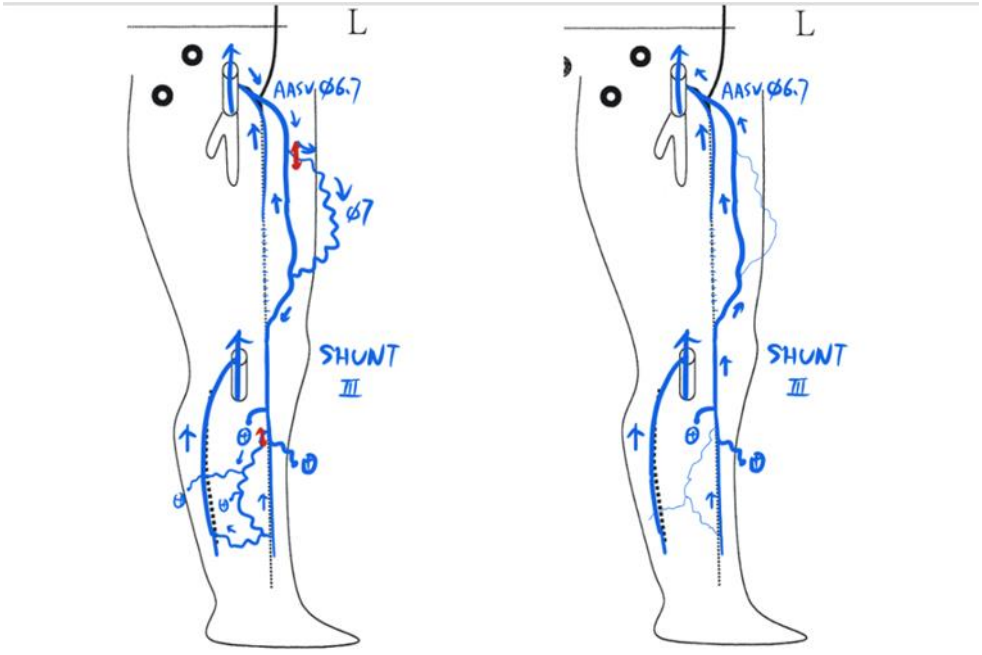




CASE 2:

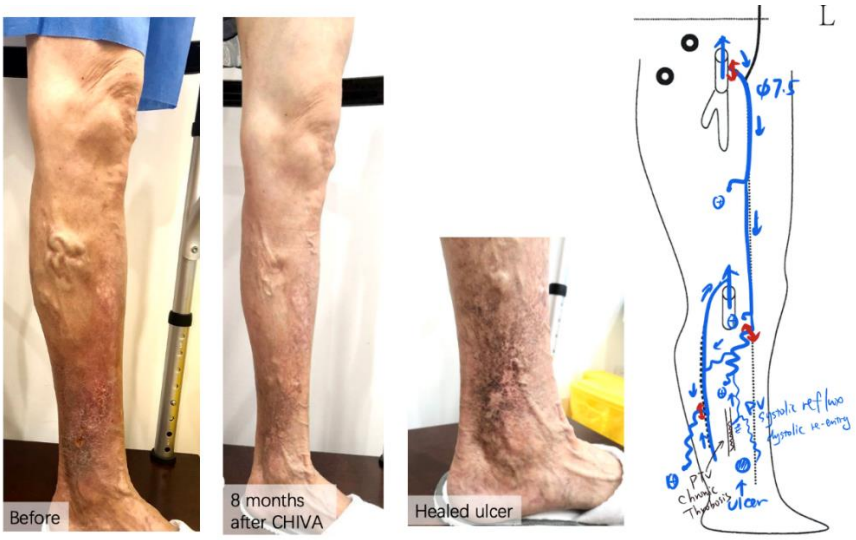
A 48-year-old female presented with varicose veins and leg oedema in left leg for over 5 years. Hemodynamic ultrasound showed SHUNT III in left leg. SFJ, anterior accessory saphenous vein (AASV) and its tributary were incompetent. CHIVA2-step1 was performed. The patient was checked by duplex ultrasound in 2 months after operation and the SFJ and AASV repented competent. After 2 years follow up, it showed no need to perform step 2





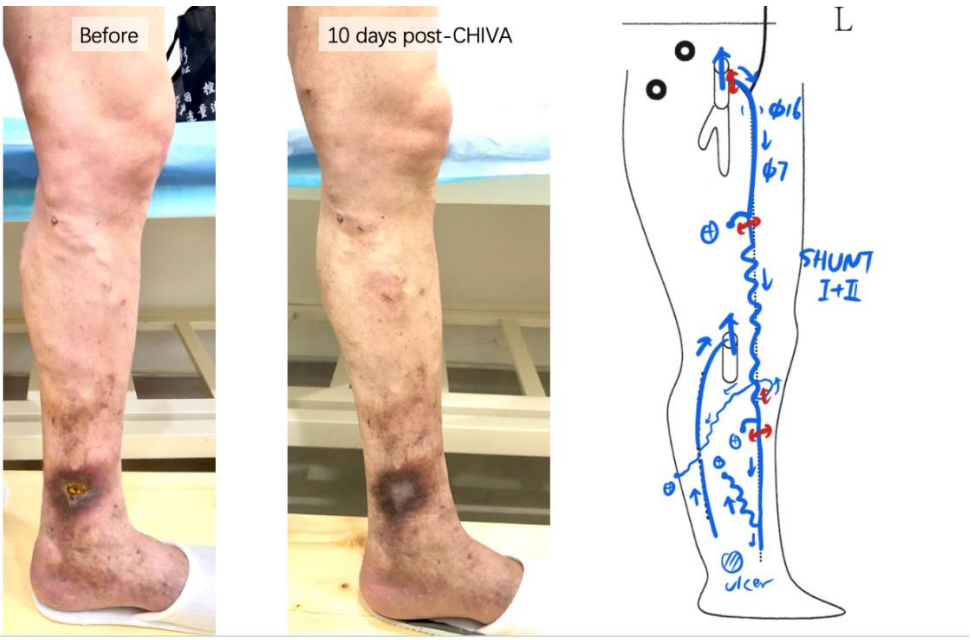
CASE 3:

A 62-year-old female presented with varicose veins for 16 years and recurrent venous ulcer for 1 year. Hemodynamic ultrasound showed SHUNT I+II in left leg. SFJ, GSV and its tributaries were incompetent. Obstacle was found in post tibial vein and systolic reflux tested in a perforator nearby. CHIVA was performed without disturbing GSV and the reflux perforator. The ulcer was healed in 2 weeks post operation.



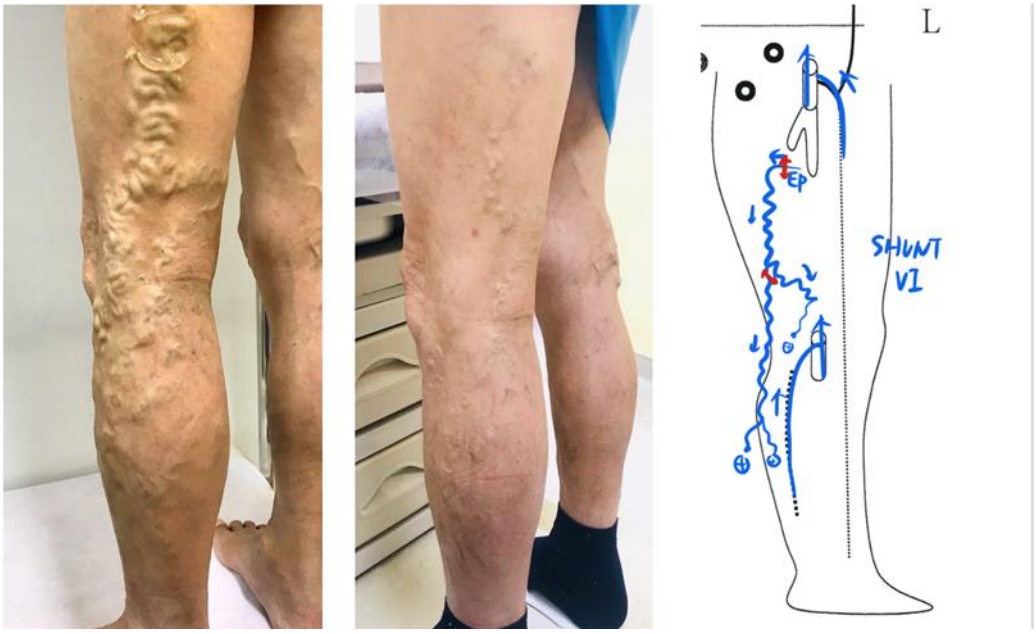
CASE 4:

A 70-year-old male repented with unhealed venous ulcer for 2 years. Hemodynamic ultrasound showed SHUNT I+II in left leg. CHIVA was performed with pressure column fragmentated. The ulcer was healed in 10 days after CHIVA procedure.



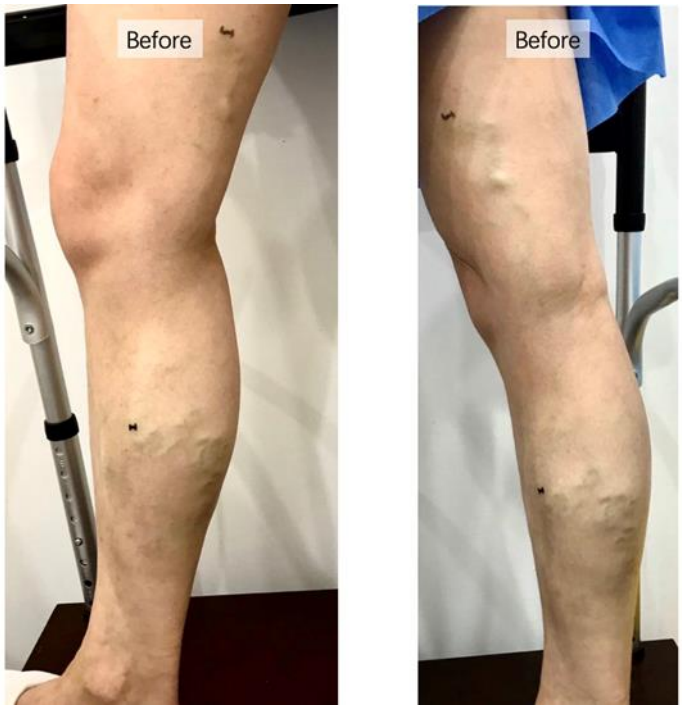
CASE 5:

A 69-year-old male presented with severe varicose veins in the lateral side of left leg. Hemodynamic ultrasound showed a diastolic reflux perforator dilated on lateral thigh and repented SHUNT VI. CHIVA was performed with closure of escape point and fragmentation of pressure column. Bulged varicose veins were collapsed in 6 months.



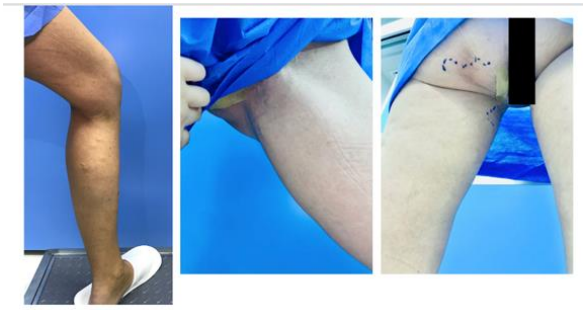
CASE 6:

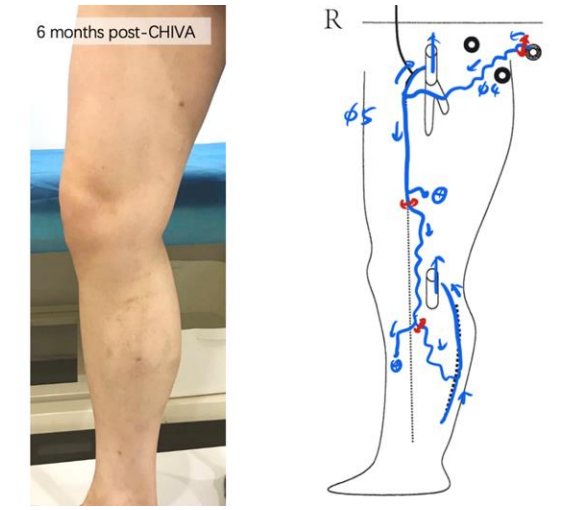
A 37-year-old female presented with varicose veins for 7 years after delivery. She felt swelling and Pain of varicose veins during the period. Hemodynamic ultrasound showed pelvic escape in CP and presented as SHUNT VI. CHIVA was performed with closure of escape points. Symptoms improved after procedure.

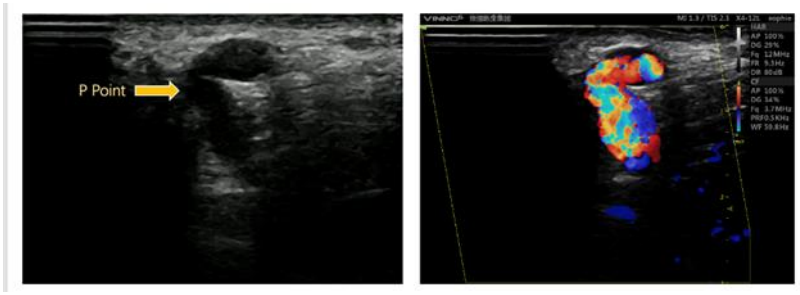


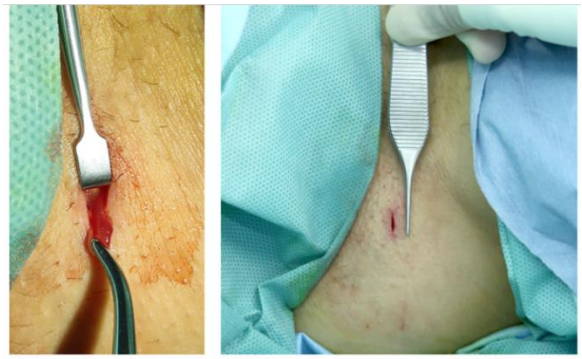
CASE 7:

A 45-year-old female presented with perineal and calf varicose veins for 1 years with Pain of varicosities in standing position. Hemodynamic ultrasound showed pelvic escape in P point and presented as SHUNT VI. CHIVA was performed with closure of escape points. Pain disappeared after the procedure.



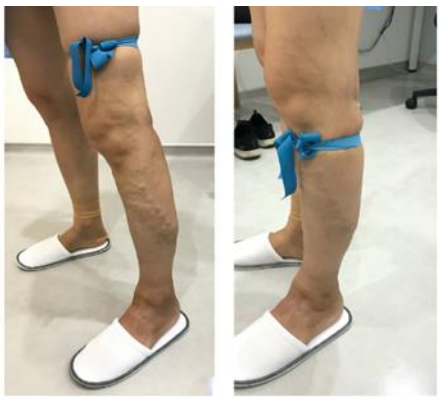




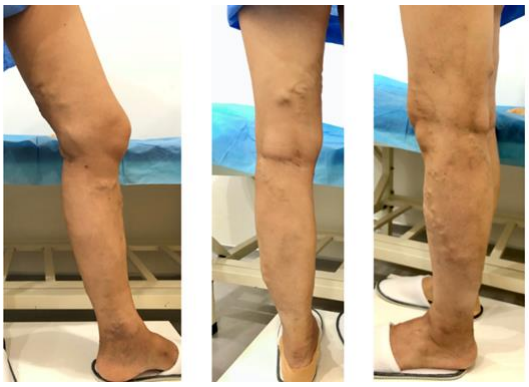


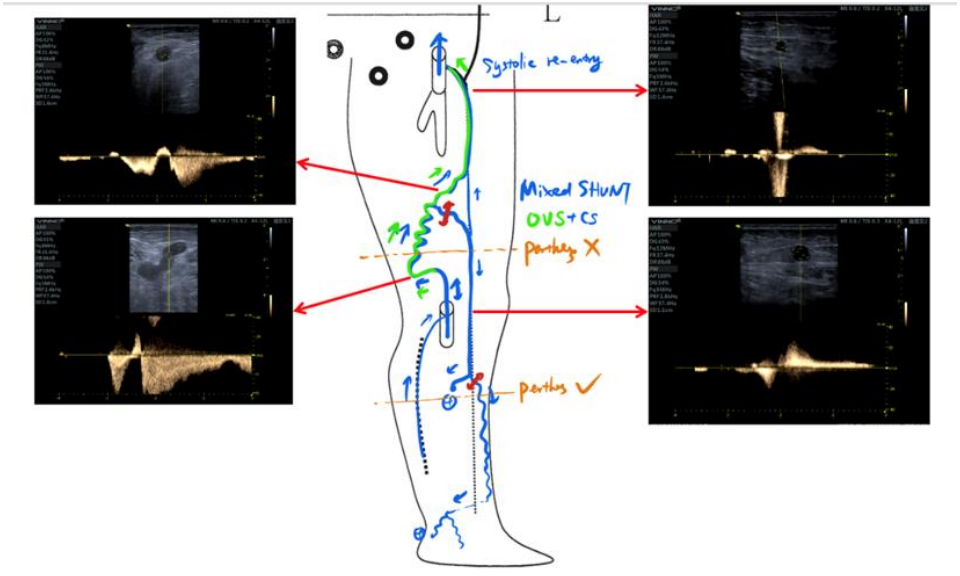
CASE 8:

A 64-year-old female presented with varicose veins in left calf for over 30 years. She felt heaviness in calf after long time standing. She had a history of leg swelling and Pain which wasn't diagnosed and treated after a procedure for ectopic pregnancy 15 years ago. Several years later varicose veins were found at posterior thigh. Hemodynamic ultrasound showed a combination of open vicarious shunt and closed shunt. Femoral vein was Patient and mild incompetent. CHIVA was performed with open vicarious shunt preserved and closed shunt disconnected.



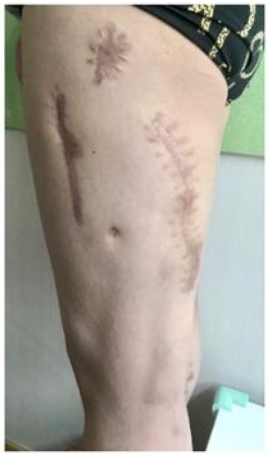
A. Elastic band was performed above knee where open vicarious shunt was pressed. After the patient walking for 30 steps, the calf varicose veins became more bulged. B. Elastic band was performed below knee where only closed shunt was pressed. The varicose veins disappeared after walking.



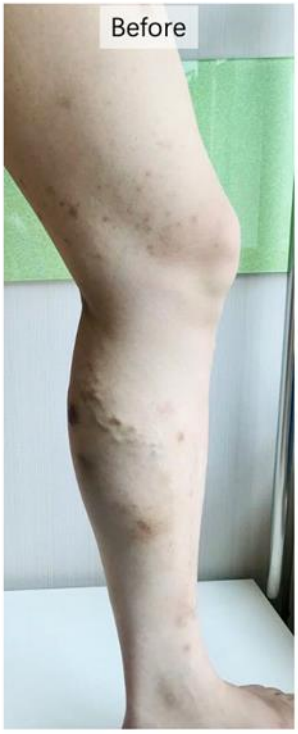


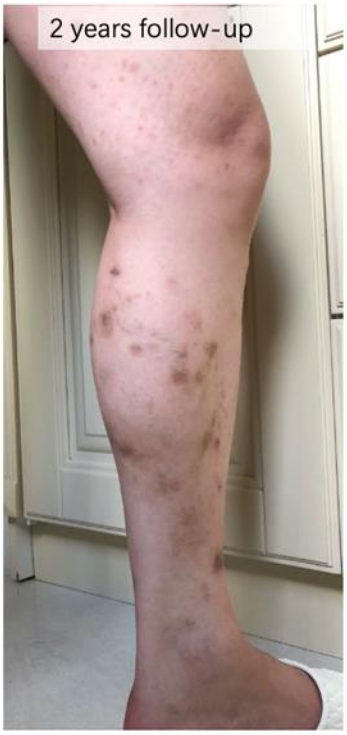
CASE 9:

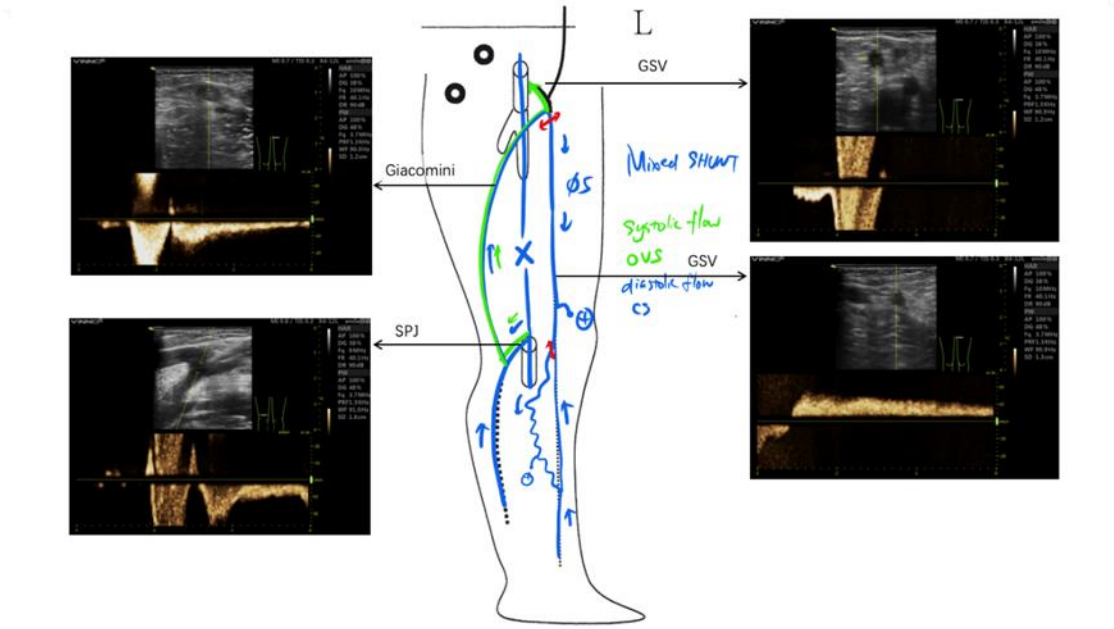
A 35-year-old male presented with varicose veins. He had a history of stab wound 13 years ago. Hemodynamic ultrasound showed MIXED SHUNT and femoral vein obstacle. CHIVA was performed with open vicarious shunt preserved and closed shunt disconnected.

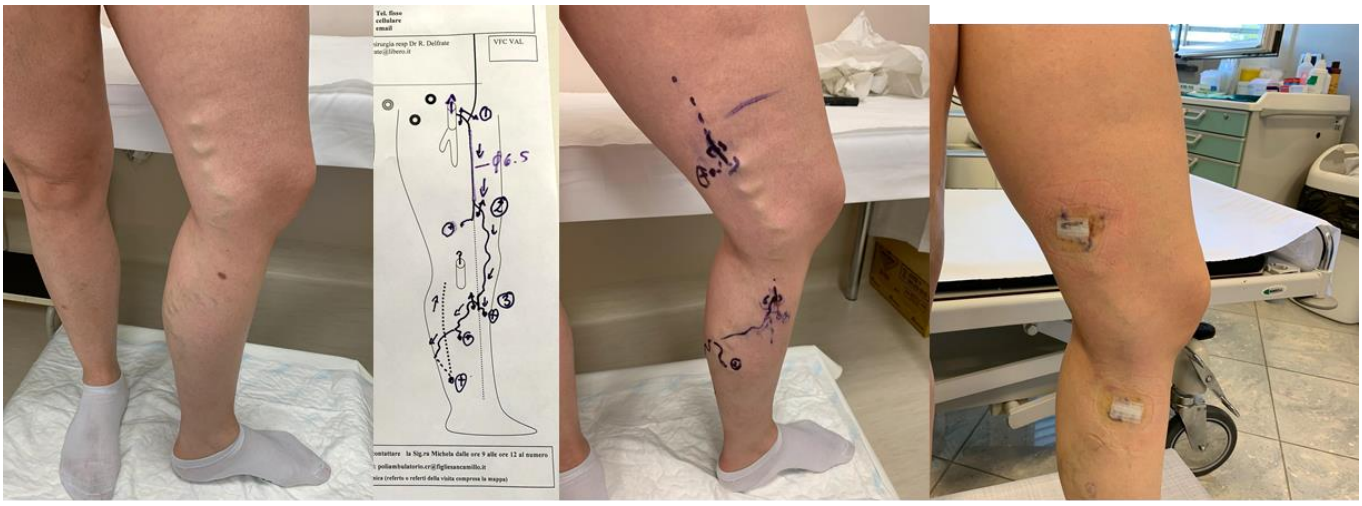


Patient had an history of stab wound 13 years ago

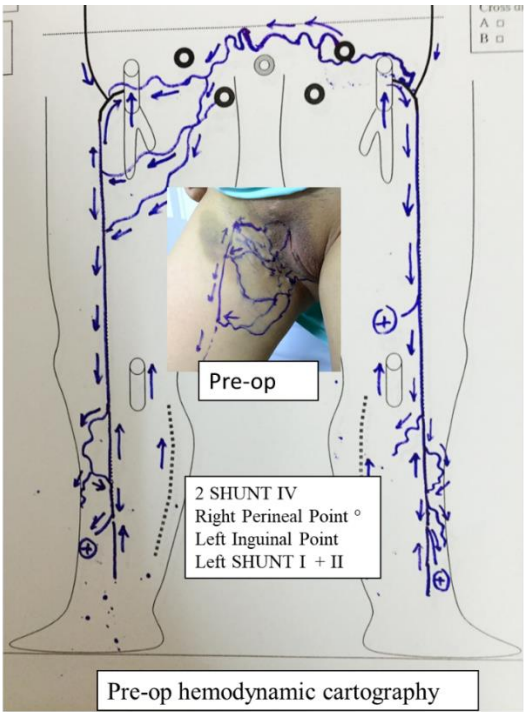


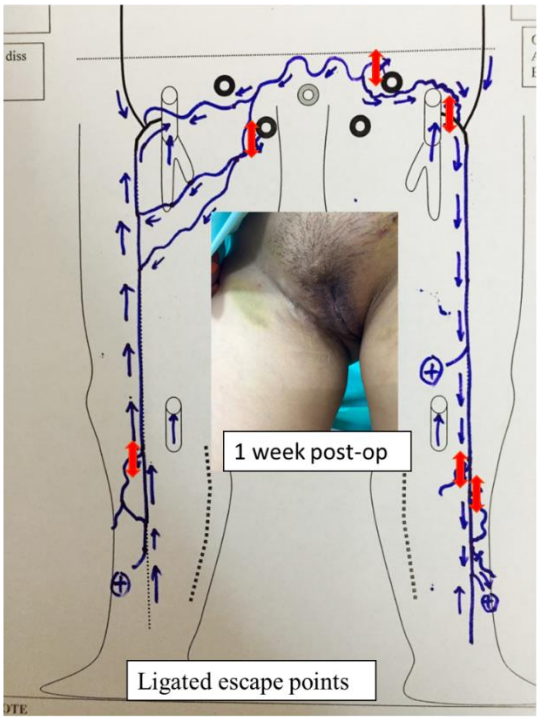






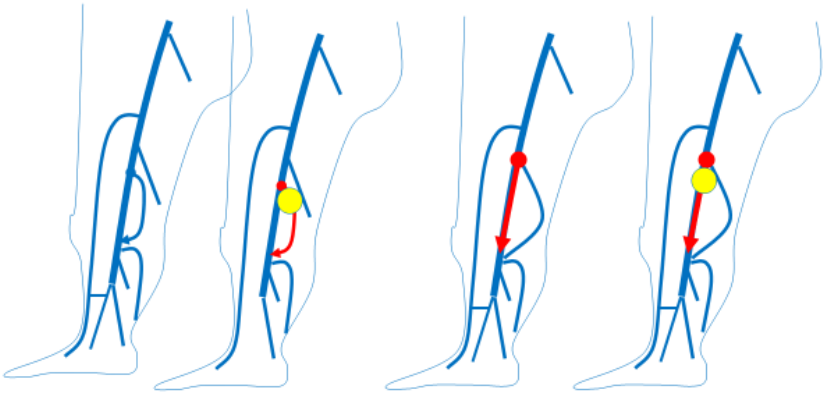
Shunt I + II Dr Roberto Delfrate Cremona Italy





Dr Le Thanh Phong Hô Chi Minh-Ville Vietnam

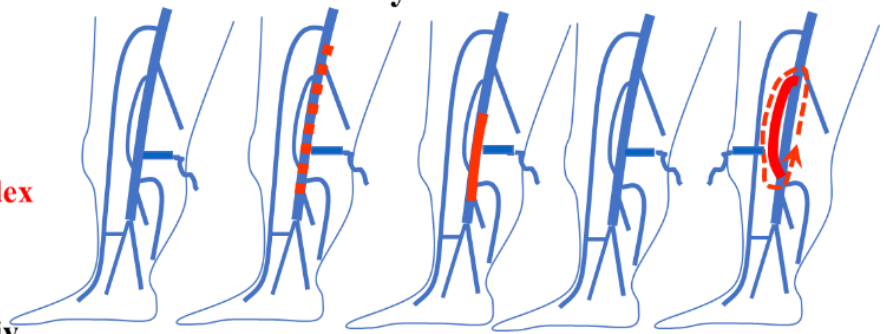
737942- *Deep mapping*



Double superficial femoral vein with an incontinent collateral is a closed shunt corrected by CHIVA

Incontinent superficial femoral vein and continuous deep femoral vein connected to the popliteal vein is a closed shunt corrected by CHIVA

Doppler Reflux Grade Classification. Dynamic reflux Index DRI



Dynamic Reflux Index

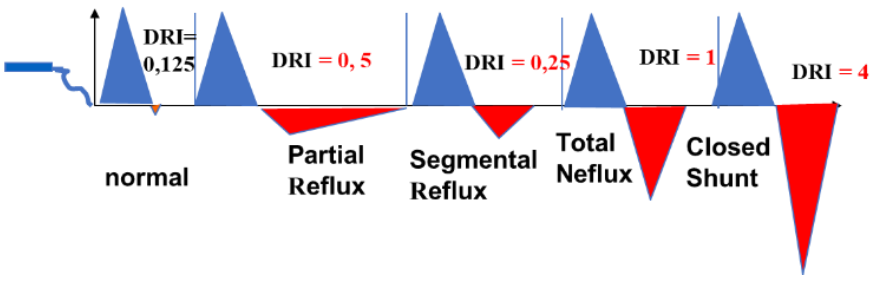
$$DRI = \frac{VmN^2 \times tN}{VmS^2 \times tS}$$

VmR =Reflux Velociy mean

tR = Reflux time

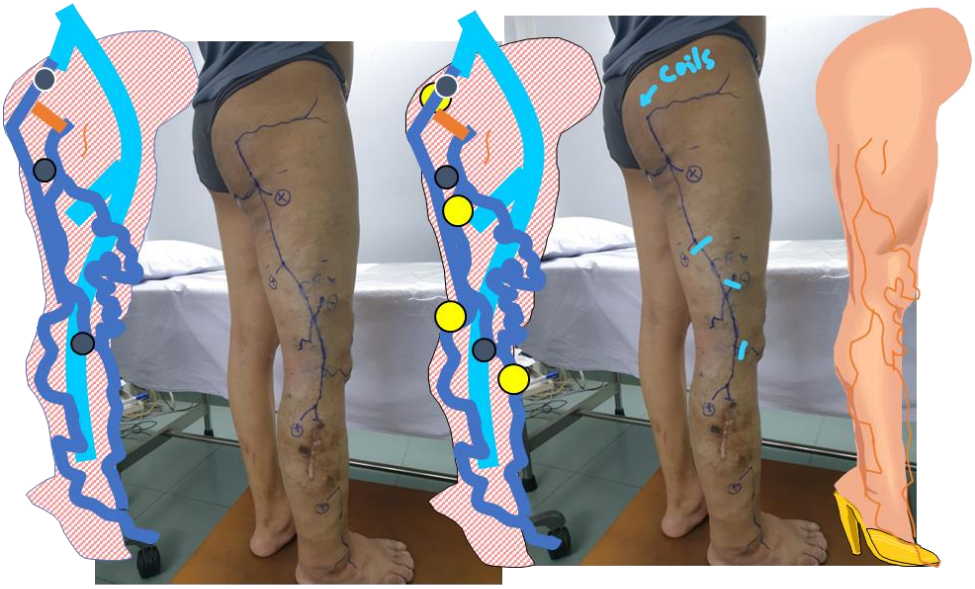
VmS = Systole Velociy Mean

tS= Systole time



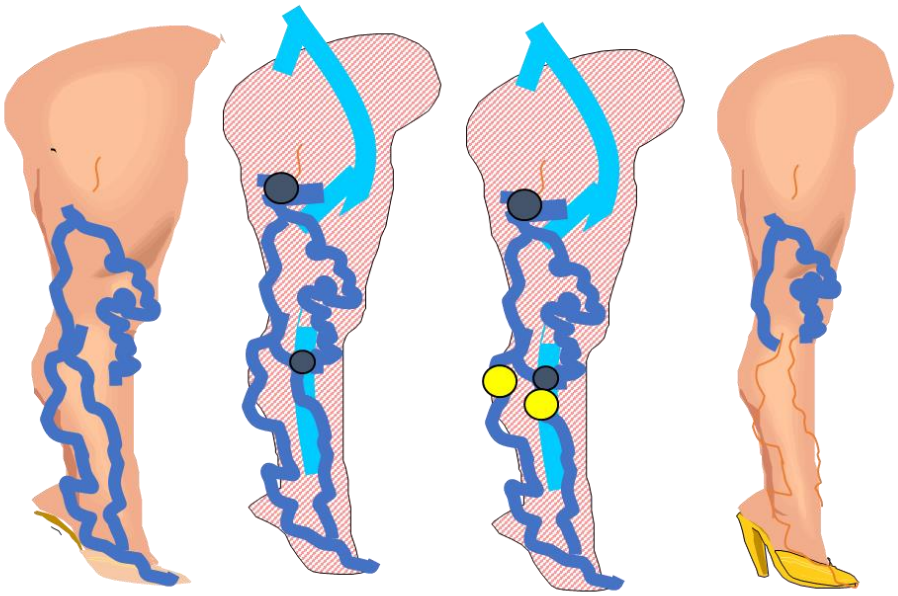
737943- *Venous malformation mapping*

Same clinical presentation, but different hemodynamic conditions identified by echodoppler and different treatments

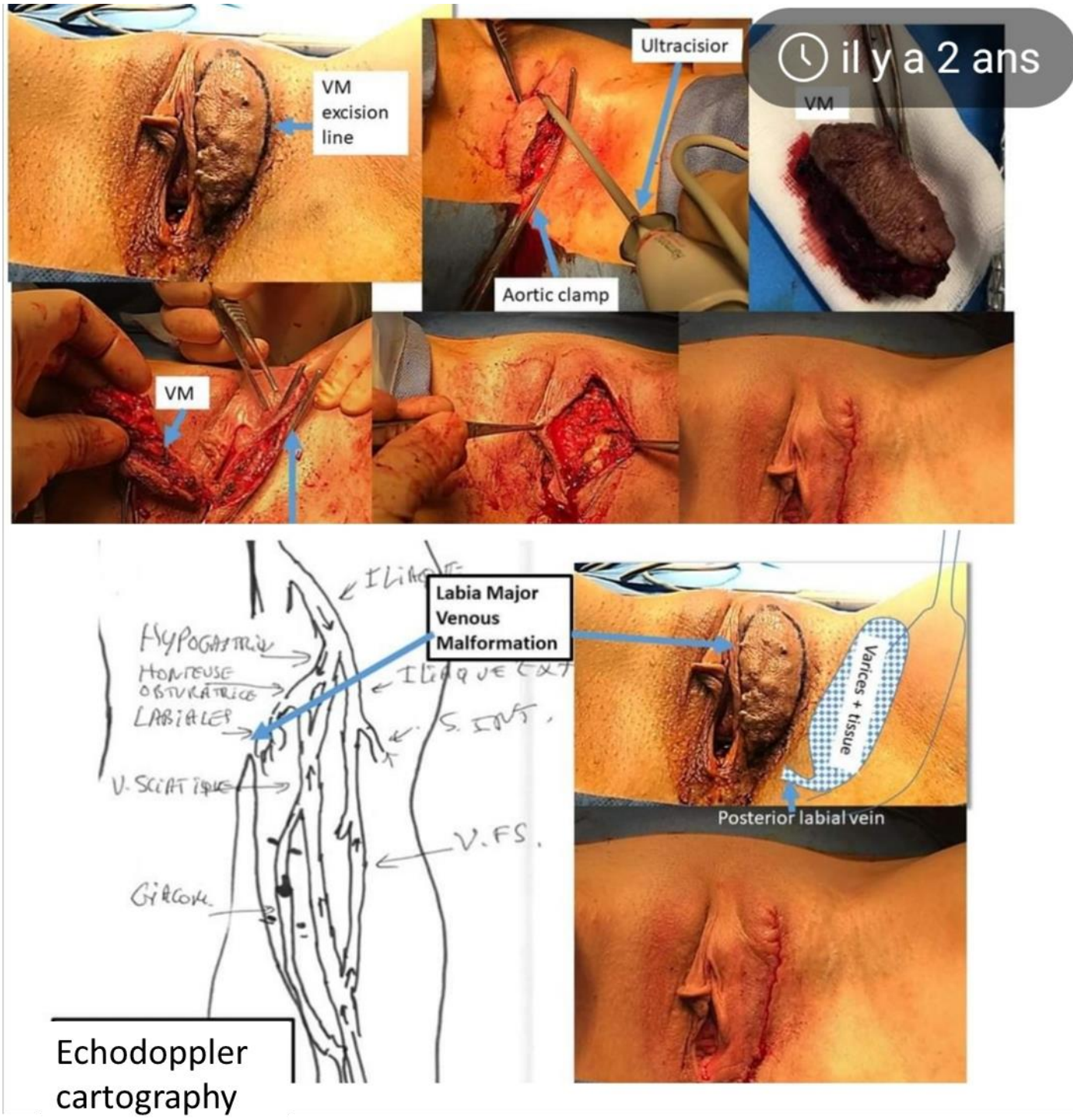


Dr Le Thanh Phong Hô Chi Minh-Ville Vietnam

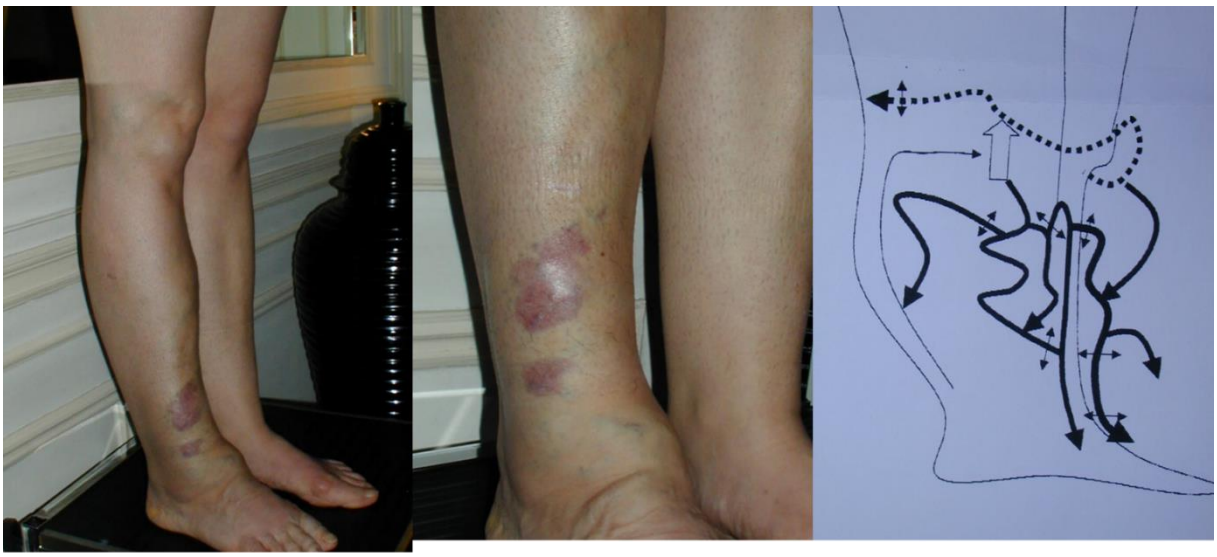
Klippel Trenaunay Weber syndrome sans varices vicariantes ●
Shunt VI étagés à déconnecter



Klippel Trenaunay Weber syndromemême aspect Clinique mais configuration hémodynamique différente. Varices de la veine marginale vicariant SOV l'hypoplasie fémorale à ne pas déconnecter + SHUNT VI à deconnecter . Restent les varices vicariantes car indisepnesbles au drainage de la jambe. ●



Labia Major varicose malformation fed by incompetent pudendal vein via posterior labial vein, associated with Giacomini shunt II. Hemostasis with aortic clamp . Easy exeresis.



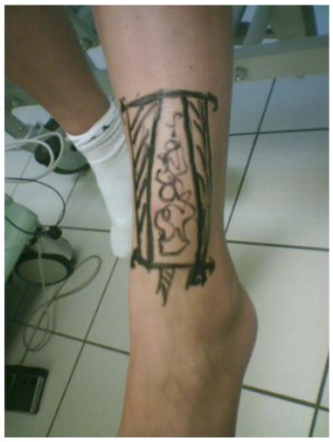
Venous malformation fed by varicose veins closed shunts VI+ shunts II to be disconnected without phlebectomy.

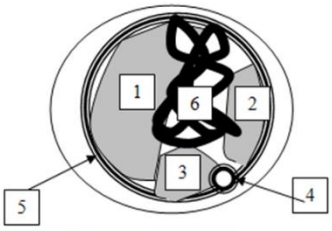
Les TRONCS VEINEUX PROFONDS des deux membres inférieurs sont normaux, sans incontinence, ni thrombose,ni altération pariétale et sans séquelles post-phlébitiques.

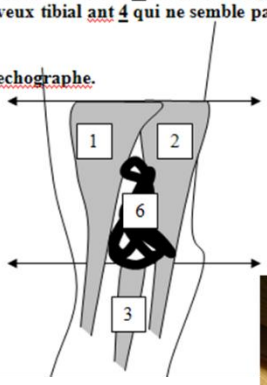
Les TRONCS VEINEUX SUPERFICIELS des deux membres inférieurs sont normaux, sans incontinence, ni thrombose,ni altération pariétale et sans séquelles post-phlébitiques.

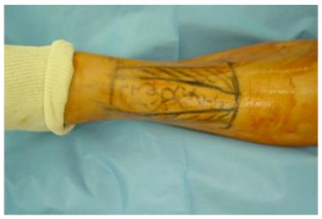
Les TRONCS VEINEUX PROFONDS des deux membres inférieurs sont normaux, sans incontinence, ni thrombose,ni altération pariétale et sans séquelles post-phlébitiques. **SAUF: ANGIOME VEINEUX 6 capillaire intrant partiellement caverneux (partiellement compressible et liquide) , sous-aponevrotique 5 , large de 17 mm et profond de 16 mm au 1/4 inf de la loge ant de jambe gauche (78 mm de haut) limité en dedans par le muscle et tendon jambier ant.1 , en dehors par le muscle et tendon extenseur commun 2 et en arrière par l'extenseur propre du GO 3 et le paquet vasculo nerveux tibial ant 4 qui ne semble pas être infiltré.**

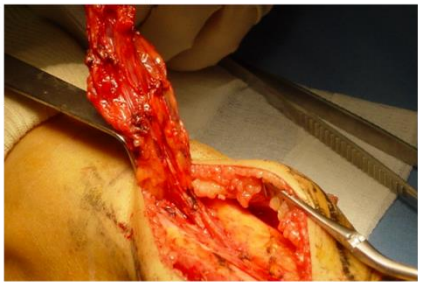
Dessin fait sur la peau: et photos sauvegardées dans echographe.



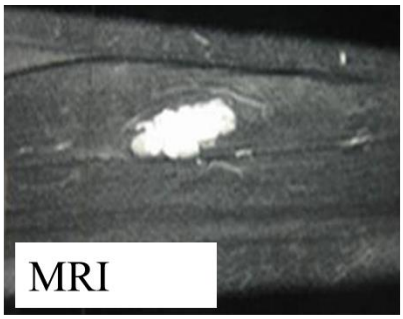




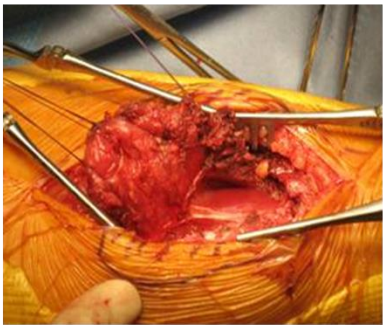


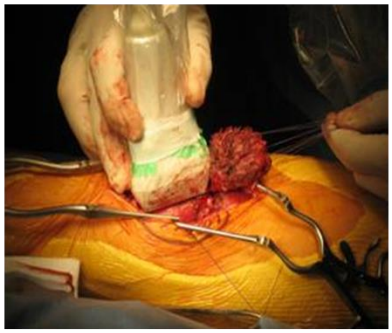


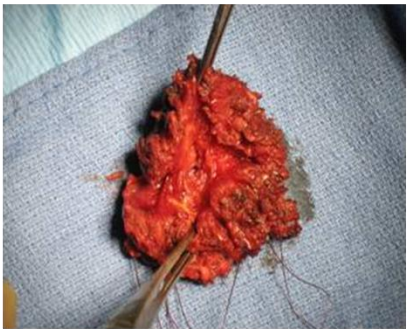
Acurate Echodopper superficial and deep cartography and marking in order to ease the exeresis of a capillar veous malformation

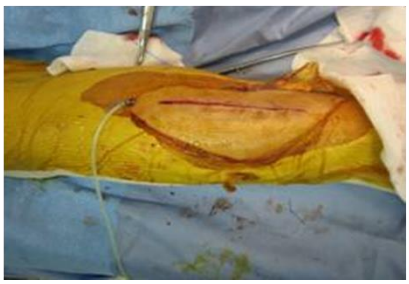


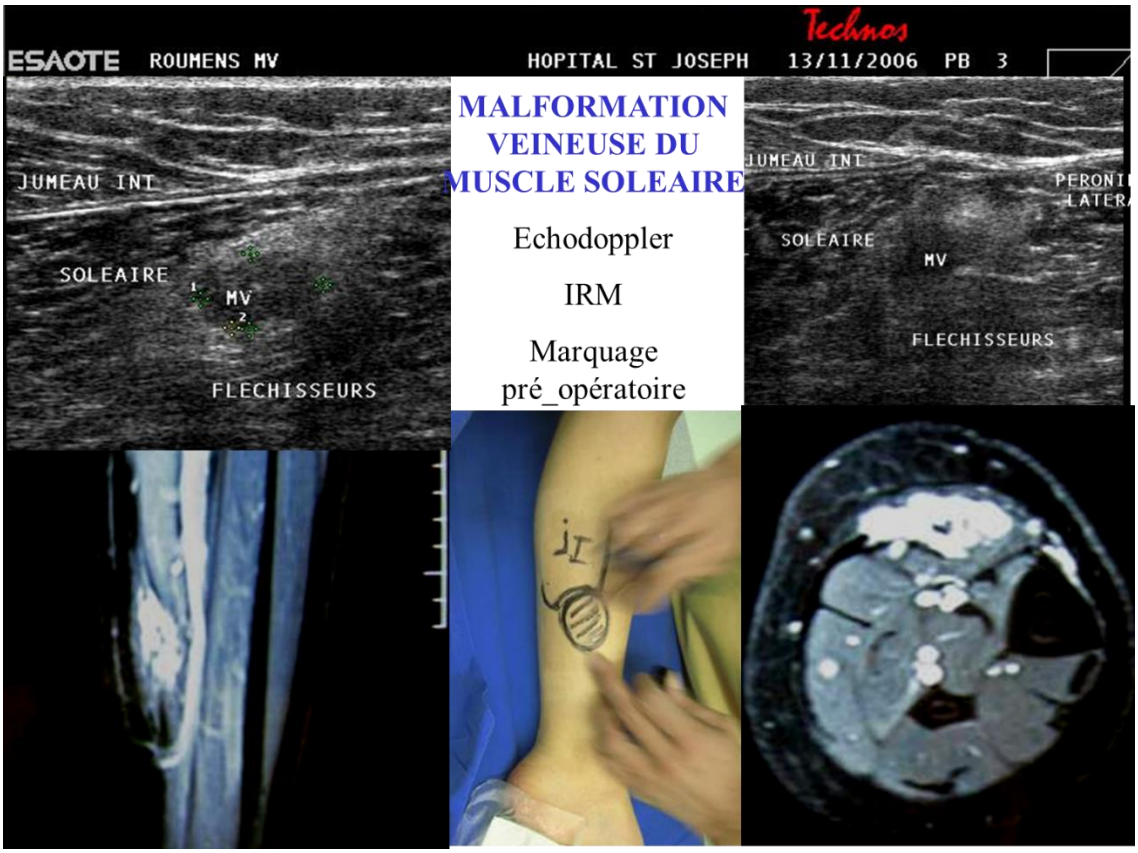
Duplex marking









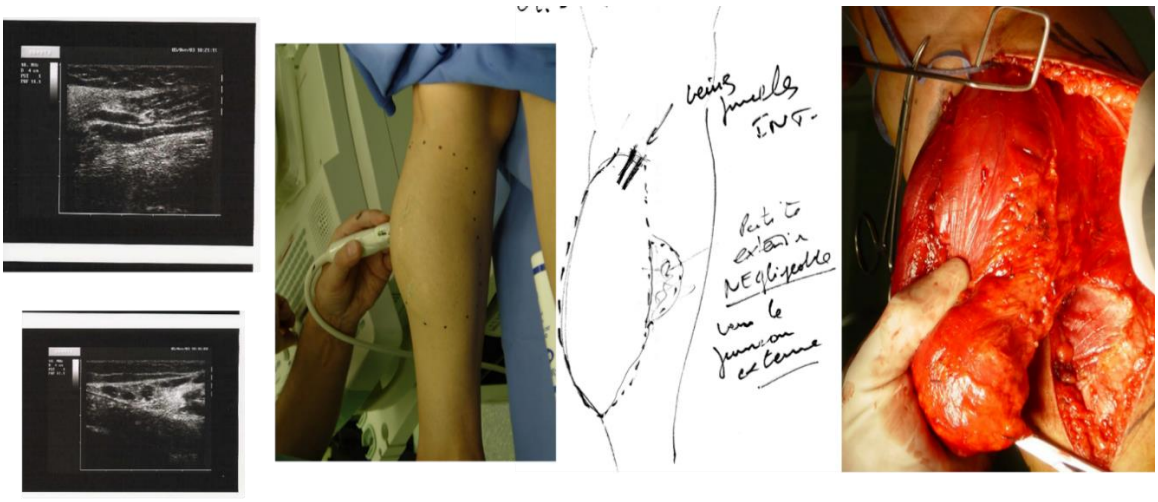


MALFORMATION VEINEUSE DU MUSCLE SOLEAIRE

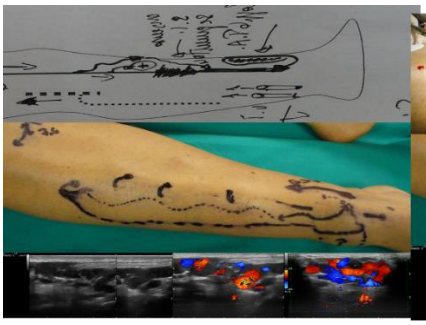
Echodoppler

IRM

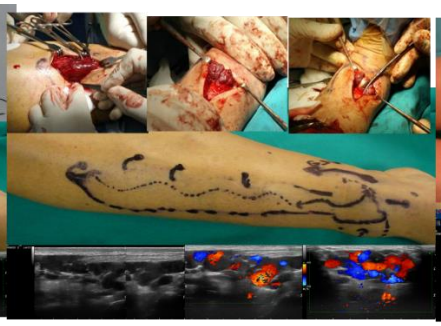
Marquage pré_opératoire



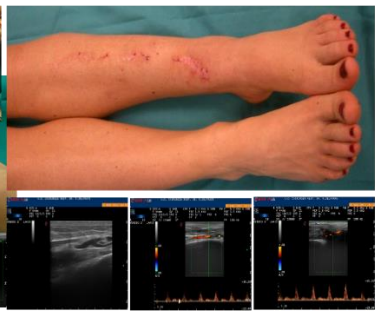
Venous malformation of all the veins of the medial gastrocnemous muscle no more functionnal (no voluntary nor reflex contraction). Echodoppler Muscle exeresis.



VM sub aponeurotic Ant. Tibial compartment.Previous foam treatment.Pain increased after foam (1 year) .VM unchanged

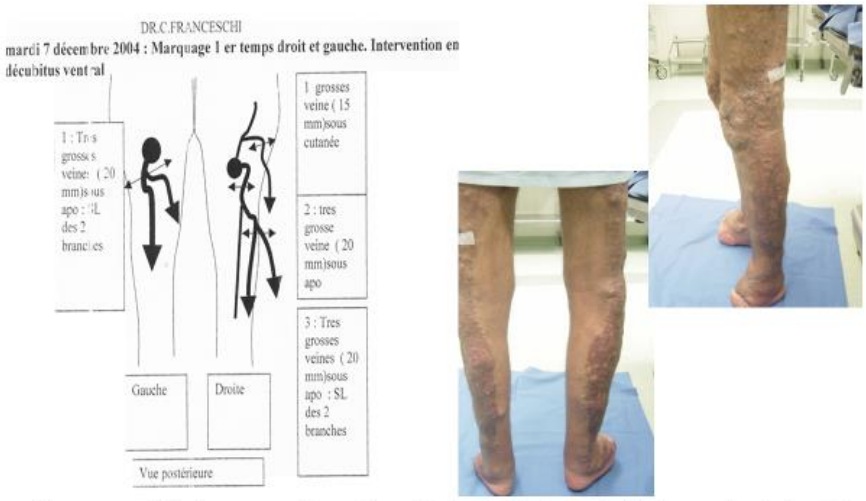


Echo guided surgical exhaustive ablation



30 days post Op. Swelling dramatically reduced. No pain.

Dysplasie veineuse unilatérale avec troncs veineux profonds présents sauf abouchet de veine fémorale superficielle droit mais compensée par la veine femorale profonde. Malfom1ations de:, veines marginales droite et gauche. CHIV A possible En plusieurs temps.



Venous superficial venous malformation with shunts VI of marginal veins, and aplasia of the right femoral vein well compensated by the deep femoral vein OVS. Possible successive disconnections.

Chapter 8

Each chapter includes some of the elements of the previous chapters and anticipates those of the following chapters.

8-Treatment of venous insufficiency

81- Medical treatments

811- Oral or local biochemical treatment

812- Hemodynamic medical treatment

8121-Reducing intravenous lateral pressure IVLP.

8122-Thermal reduction of residual pressure RP by cold means.

82- Increase of extravenous pressure EVP by support and compression of the extremities.

821-Homogeneous compression:

8211-Immersion in a liquid

8212-Air inflated cuff

822- Heterogeneous compression.

8221- Non-elastic band compressions.

8222- Compressions with elastic bands and stockings.

8223- Bandages, socks, stockings, tights, tights

83- CHIVA treatment

831- CHIVA treatment Definition

832-Indications

8321-Informed consent

8322- Erroneous indications

8323-Pelvic escape points can be directly disconnected

8324-Aesthetics

833- CHIVA method

8331-Strategy

83311- Fractionation of the incompetent column

83312-Disconnection of closed CS and ODS open deviated shunts

83313- Preservation of open vicarious shunts OVS

83314-Elimination of non-draining varicose veins

83315- Preservation of great saphenous vein GVS

83316- Keeping the number of disconnections as low as possible

83317- Apply class 2 support

83319-Mapping strategy and CHIVA

83318-Post-operative follow-up and monitoring

8332- Tactics

83321-The haemostator is a fast, efficient and haemostatic tool

83322-Non-absorbable suture threads and ligatures

83323- Do not leave behind stumps

83324- Closure of the fascia with non-absorbable thread

8333- Specific procedures according to escape points and types of shunts

83331- Saphenofemoral junction.

83332- Saphenopopliteal junction.

833321- Localization of the sciatic nerve

833322- Disconnection of the small saphenous vein

833323- In the absence of Giacomini

833324- The position of the saphenopopliteal junction

833325- The aponeurosis is always closed with nonabsorbable suture.

833326- Popliteal cavernomas

83333- The popliteal perforator

83334-- Incompetent femoral saphenous thigh perforators

83335--Pelvic leak points.

833351--Perineal escape point. P point

833352-. Inguinal escape point: i Point

833353- Obturator escape point: O Point.

833354- SHUNT III CHIVA

8333541-1. Disconnection only of saphenofemoral escape N1>N2.

8333542-2. CHIVA 2, i.e. CHIVA in 2 steps.

8333543-. CHIVA in 1 step by devalvulation.

83336-Deep escape points.

833361- Deep closed femoral shunt.

8333611- surfacefemoral-deep femoral closed SHUNT.

8333612- Superficial femoral-femoral SHUNT

84- Results of CHIVA treatment

85- CHIVA cure by sclerotherapy:

86- Methods of reconstruction and valvular prostheses.

87- Deep revascularization

871- Therapeutic excesses

872- Stent length and size can also be evaluated by Poiseuille's law.

88- Venous malformations

89- Venous ulcers

80A- Haemorrhoids

8-*Treatments for venous insufficiency*

*The **number of proposed treatments reflects** the diversity of indications, techniques and **confused physiopathological conceptions of venous insufficiency.** **The "haemorrhoids" must also be reported among the physiopathological confusions.***

Surprisingly, very few articles and lectures regarding the varicose veins treatments, consider the loss of arterial by-pass chance despite evidences

Ref: *1-No touch technique of saphenous vein harvesting: Is great graft patency rate provided? Papakonstantinou NA J Thorac Cardiovasc Surg. 2015 Oct;150(4):880-8. doi: 10.1016/j.jtcvs.2015.07.027. Epub 2015 Jul 15. 2-The no-touch saphenous vein for coronary artery by-pass grafting maintains a patency, after 16 years, comparable to the left internal thoracic artery: A randomized trial. Samano N1,:ClinicalTrials.gov NCT01686100.Copyright © 2015 The American Association for Thoracic Surgery. 3-Meta-analysis of infrapopliteal angioplasty for chronic critical limb ischemi.Marcello Romiti, (J Vasc Surg 2008;47:975-81.). 4-Meta-analysis of popliteal-to-distal vein by-pass grafts for critical ischemia. Maximiano Albersand (J Vasc Surg 2006;43:498-503.). 5*-DELFRATE R.: Thanks to the CHIVA strategy may the histoarchitecture of great saphenous vein-sparing, make it suitable as graft for bypasses? Veins and Lymphatics 2019; volume 8:8227 2

Before detailing the treatments, it is necessary to underline the general aspects.

a-Cosmetic issues:

The only aesthetic indications regarding varicosities and telangiectasias *aim at immediate and "perfect" results****, even if they must be repeated throughout life.***

This relates to the *irremediable evolution due to ageing but also to non-hemodynamic strategies responsible for recurrences*

b-Essential" varicose veins are the most frequent manifestations *of venous insufficiency*

They are generally benign and not very progressive.

They are treated *for:*

- *aesthetic and/or*

-functional reasons

according to their symptomatology

-aesthetic discomfort, pain, heaviness, intolerance to heat) and the

- signs related to drainage defects of varying severity (oedema, hypodermitis, ulcers, varicose thrombosis).

c*-The proposed treatments are* ***mainly destructive,***

-in particular that of the great saphenous vein,

-without considering the loss of chance for a future vital by-pass of the arteries, *including in the* ***lightest forms,***

-to *respond to* ***aesthetic*** *demands* ***or to fear of an improbable serious evolution.***

d- Destructive *treatments are* ***encouraged*** *by*

their ***economic returns*** *and*

-supported by ***sponsorship from the destructive products industry,*** *without which the majority of congresses would be unfeasible* ***in this regard***

Bioprotec Lyon saint Priest https://bioprotec.fr recovers stripped saphenous veins from varicose vein patients and sells them as grafts for arterial bypass surgery

.

The result is a choice of "readymade" treatments of good economic return, attractive because of their "innovative" aspect, although they are based on outdated scientific concepts.

e- However, non-destructive treatments based on hemodynamic are available, from simple restraint and postures to the CHIVA cure which will be explained below.

Brief History

Non Hemdynamic Destructive

1906 Stripping Mayo Babcok

1947 Sclerosis

2000RF, Laser,Mousse, Glue……

Hemodynamic Conservative

1788-1899: Saphenous vein ligation Trendelenburg

HEMODYNAMICS IGNORED for 90 ans !!!

1988: CHIVA

Varico Centric Therapy

Destruction of incompetent veins

-Phlebectomy

-Sclerosis

-Laser

-Glue

-Radio Frequence

No respect ofdrainage

Tissue damages

Vicarrious recurrence

Venious capital destruction

Hemodynamico Centric Therapy

REDUCE Trans Murale TMP

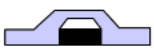
Increase Extravenous P

-Compression

Lower Intravenous P

-Posture

-Suppress obstacles



CHIVA

-Graviatationnal Hydrostatic P fractioning

-Shunts disconnection

-Respect of drainage

So, clear, and complete Informed consent should ethically and medico-legally also propose conservative and hemodynamic treatments, from simple compression to the CHIVA validated by RCTs and Cochrane reviews.

Patients most often consult for 3 types of reasons.

for unsightly manifestations (varicose veins, varicosities to prevent serious

for complications of simple varicose veins (haemorrhage, phlebitis, pulmonary embolism),

or for severe signs and symptoms of trophic disorders (hypodermitis, ulcers).

The answer must be clear.

This is where "informed consent" takes on its full importance, both ethically and medico-legally.

In the first case, reassure the patient by confirming that his pathology is benign and its complications rare and easy to avoid by simply wearing support socks.

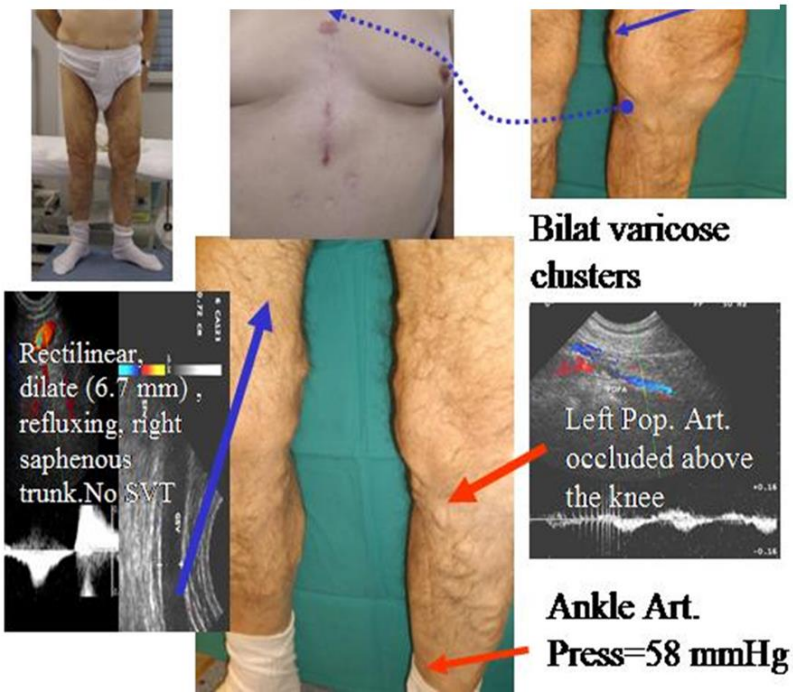
In the second and third cases, explain to the patient the possibilities and real limits of the various treatments according to the results of the Doppler hemodynamic mapping (cartography).

In all cases, inform the patient that the great saphenous vein, whether competent or not, is a precious material in the event of the need for a vital arterial by-pass. Add that it can be preserved by medical (compressive stockings, lifestyle) and/or surgical (CHIVA) treatment methods, which are conservative and hemodynamic for venous insufficiency.

The Saphenous vein can save life, even in varicose people. Why destroy it without warning the patient? Especially since it can be treated effectively without destroying the saphenous vein by hemodynamic methods

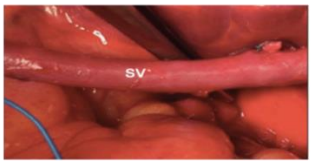
Male 78 y

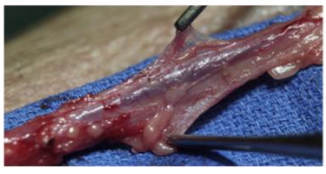
To-day:
-left leg limp
-Bilat varicose clusters
10 years ago
-5 coronary by-passes (3 left GSV) +
-Right GSV crossectomy for SVT



Bioprotec (Lyon France) *collects, freezes and sells stripped great saphenous veins as allografts*

The great saphenous vein, continuous or incompetent
-Aorto-coronary bypasses
- Peripheral bypasses
Patch, vascular access
Bypass surgeries in septic environments, especially infected prosthesis





- No touch" harvesting
- Less spasm
- No dilation (less endothelial trauma)
- Conservation of vasa vasorum,
- less parietal ischemia
- Conservation of NO synthesis (less intimal hyperplasia

Problème éthique de la destruction du capital veineux (G.DE WAILLY)

Principe de non malfaisance

- Probabilité de la nécessité d'un pontage artériel après chirurgie veineuse : 3% (1)
  - chirurgies veineuses : 200 000 / an + 6000 000 sclérothérapies

⇒ **6000 pontages / an**

- Pontages veineux aorto-coronariens
  - 70% des malades ayant des varices avaient des segments veineux compatible avec réalisation du PAC (2)

(1) Lofgren EP. In Bergan JJ, Yao JST (eds). Surgery of the veins 1985 285-299
(2) Cohn et al, Ann Thor Surg 2006 81(4) 1269-4

f-The informed consent I give to patients is this:

Varicose vein treatment: The saphenous vein is vital for future peripheral and coronary venous bypasses, and should not be destroyed without the informed consent of the patients, especially as this benign disease can be treated simply by compression socks or minimally invasive surgical methods (CHIVA cure), which are less expensive, ALWAYS conservative and validated (CHIVA), better than destructive surgical (stripping) or endovenous techniques (sclerosis, foam, laser, Radiofrequency C).

The Great Saphenous Vein (GSV) in healthy subjects but also in varicose veins (the saphenous veins removed from a patient to treat his varicose veins are frozen by a Lyon-based company (Bioprotec) and then resold to carry out bypasses on another patient) can be removed for a coronary or limb by-pass.

Scientific studies:

A- Coronary venous by-pass equivalent to mammary artery by-pass

1-No touch technique of saphenous vein harvesting: Is great graft patency rate provided?

Papakonstantinou NA J Thorac Cardiovasc Surg. 2015 Oct;150(4):880-8. doi: 10.1016/j.jtcvs.2015.07.027. Epub 2015 Jul 15.

2-The no-touch saphenous vein for coronary artery by-pass grafting maintains a patency, after 16 years, comparable to the left internal thoracic artery: A randomized trial.Samano N1, :ClinicalTrials.gov NCT01686100.Copyright © 2015 The American Association for Thoracic Surgery. Published by Elsevier Inc. All rights reserved.

B-Venous by-pass of the lower limb arteries is still the most effective. -

1-Meta-analysis of infrapopliteal angioplasty for chronic critical limb ischemia

Marcello Romiti, (J Vasc Surg 2008;47:975-81.)

2-Meta-analysis of popliteal-to-distal vein by-pass grafts for critical ischemia

Maximiano Albersand (J Vasc Surg 2006;43:498-503.)

D- The CHIVA method gives less recurrence than destructive methods (Stripping, Laser, radiofrequency, sclerosis, foam).

C- CHIVA cure

1-CHIVA method for the treatment of chronic venous insufficiency. Bellmunt-Montoya S1, Cochrane Database Syst Rev. 2015 Jun 29;(6):CD009648. doi: 10.1002/14651858.CD009648.pub3

2-Hemodynamic classification and CHIVA treatment of varicose veins in lower extremities (VVLE)Hua Wang1, et al, China. Int J Clin Exp Med 2016;9(2):2465-2471 www.ijcem.com /ISSN:1940-5901/IJCEM0016552"".

3- Carandina S, Mari C, De Palma M, Marcellino MG,Cisno C, Legnaro A, et al.Varicose Vein Stripping v sHaemodynamic Correction (CHIVA): a long term randomised trial. European Journal of Vascular and Endovascular Surgery 2008;35(2):230-7

4- Parés JO, Juan J, Tellez R, Mata A, Moreno C, Quer FX,et l.Varicose vein surgery: stripping versus the CHIVA Method: a randomized controlled trial. Annals of Surgery 2010;251(4):624-31

5- Iborra-Ortega E, Barjau-Urrea E, Vila-Coll R, Ballon-Carazas H, Cairols-Castellote MA. ComPArative study oftwo surgical techniques in the treatment of varicose veins of the lower extremities: results after five years of followup. Estudio comPArativo de dos técnicas quirúrgicas en el tratamiento de las varices de las extremidades inferiores: resultados tras cinco años de seguimiento]. Angiología 2006; 58(6):459-68.

6-]P.Zamboni and all: Minimally Invasive Surgical management of primary venous Ulcer vs. compression Eur J vasc Endovasc Surg 00,1 6 (2003)

7- Chan, C.-Y.a , Chen, T.-C.b , Hsieh, Y.-K.a , Huang, J.-H.c Retrospective comparison of clinical outcomes between endovenous laser and saphenous vein-sPAring surgery for treatment of varicose veins (2011) World Journal of Surgery, 35 (7), pp. 1679-1686

8- The CHIVA method gives less recurrence than destructive methods (Stripping, Laser, radiofrequency, sclerosis, foam). Guo et al. Long-term efficacy of different procedures for treatment of varicose veins A network meta-analysis. Medicine (2019) 98:7

g-Acute Deep Venous Insufficiency:

I do not insert with Acute Deep Venous Insufficiency *in this book because their strategy is essentially antithrombotic, intended to avoid pulmonary embolism in the immediate future and post-phlebitis disease in the future.*

Chronic deep venous insufficiency *of the lower extremities, dominated by post-phlebitis disease, is the subject of* ***miraculous endovenous deobstructive and endoprosthesis treatments*** *when the indication is hemodynamically justified.*

h-Overtreatment is the result of confusion between the signs of valve incompetence and venous occlusion or overdiagnosis of incompetence and/or obstruction.

-The May Thurner and Nutcracker syndromes are representative of the effects of trendy therapeutic and over-treatment due to lack of knowledge of hemodynamic parameters.

Both really exist *but some of them are victims of postural artefacts* ***linked to the conditions of diagnosis in horizontal decubitus, easily recognizable by the echodoppler in a semi-sitting position.***

-Hemodynamic pelvic venous insufficiency *due to the presence of varicoceles and incompetence of the pelvic veins, particularly in single or multiparous women****, should be only treated if it is symptomatic, i.e. responsible for a "pelvic congestion syndrome".*** *However, they are not necessary if the congestion syndrome is not confirmed* ***by eliminating other causes.*** *They are also* ***useless for the treatment of pelvic escape points where direct treatment is sufficient. Ref:*** R.Delfrate, Massimo Bricchi, Claude Franceschi ***Minimally-invasive procedure for pelvic leak points in women Veins and Lymphatics 8 May 2019 https://www.researchgate.net/publication/341487042_Minimally-invasive_procedure_for_pelvic_leak_points_in_women***

-The treatment of venous ulcers is the subject of an endless race to the ever more sophisticated but not necessarily more effective ointment, *if the primary hemodynamic cause, excess Transmural pressure" is not correctly treated****. Simple and inexpensive local antiseptics are then largely sufficient. Ref:*** Claude Franceschi, Massimo Bricchi, Roberto Delfrate. Anti-infective effects of sugar-vaseline mixture on leg ulcers. Veins and Lymphatics 2017; volume 6:6652.

-Venous malformations are "poorly known" *because of their rarity, so they are managed in specialized care centers****. I will show the basic hemodynamic assessment and treatment of the the most frequent cases.*** *You will note that the* ***hemodynamic data and mapping are not specific of the aetiology, whatever its aetiology, malformative or not,*** *but vary according to the specific hemodynamic impairment of each patient.*

-Valvular incompetence *is the subject of* ***prosthetic research and valve reconstruction. The CHIVA deep CHIVP cure treats incompetence*** *by disconnecting deep closed shunts instead of superficial shunts.*

-<u>Haemorrhoids</u> have been 'integrated' by some into hemorrhoidal vein embolization programs, although the cause of this condition is not, despite its name, a disease of the veins but of the anal canal. Ref: C.Franceschi. Hémorroïdes : maladie des veines ou d'un quatrième facteur. Essai d'analyse physiopathologique. Conséquences thérapeutiques. Actualités Médicales Internationales. Angiologie (8), n° 145, décembre 1991
VIDEO https://youtu.be/1FoYynLlb98.

81- Medical treatments

811- Biochemical treatments by oral or local route intended to improve capillary permeability is not developed here. *However, we can* ***point out the specific treatments that re-establish the oncotic pressure*** *responsible for reducing the drainage of interstitial liquids when it is reduced by renal, hepatic, or other insufficiency.*

812- Hemodynamic medical treatments are always desirable because they

correct excesses of Transmural pressure very significantly TMP. They are non-invasive, painless, little or no cost. They are applicable alone or as a complement to surgical or endovenous interventions.

TMP = Lateral Intravenous pressure LVP - Extravenous pressure EVP.

8121-Reduce Lateral Intravenous pressure IVLP.

IVLP = Hydrostatic Gravitational pressure GHSP + Residual pressure RP (see Bernoulli equation)

Reduction of Hydrostatic Gravitational pressure GHSP by posture

Distal GHSP is reduced by the supine position, and more so the higher the legs are elevated. Remember, before Trendelenburg, *the only effective treatment was lying down for weeks****. The miracle of St. Pregrin*** *is that God healed his ulcer when he lay down a single night after 30 years of standing to keep his head closer to God****. It would be not a miracle, but "normal" for science if it took a few weeks instead of a single night as the legend reports. Ref****: C Franceschi - Venous hemodynamics, knowledge and miracles. Journal of Theoretical and Applied Vascular Research (page 39) - JTAVR 2019;4(2)

8122-Thermal reduction of the Residual pressure RP by cool means.

Residual pressure RP increases with the decrease of microcirculatory resistance. ***The thermoregulatory response to heat reduces microcirculatory resistance, thereby increasing RP and superficial venous flow to release calories outside the body. Cold treatment absorbs***

more calories with the feeling of cold. *It is recommended, especially for individuals with venous insufficiency.*

8123- *Increase in Extra Venous pressure EVP by support and compression of the extremities.*

Support and compression techniques are much more efficient when their rational is in mind of the practitioner.

Transmural pressure TMP is too high in two conditions.

When the extra-venous pressure EVP is too low and when the lateral intravenous pressure IVLP is too high.

*When the **extra-venous pressure EVP is too low** in the first case the **normal EVP must be restored.** This is the case at altitude or in an airplane because the atmospheric pressure is too low.*

*When the **lateral intravenous pressure IVLP is too high**, the **EVP must be higher than normal to compensate for the excess of IVLP.***

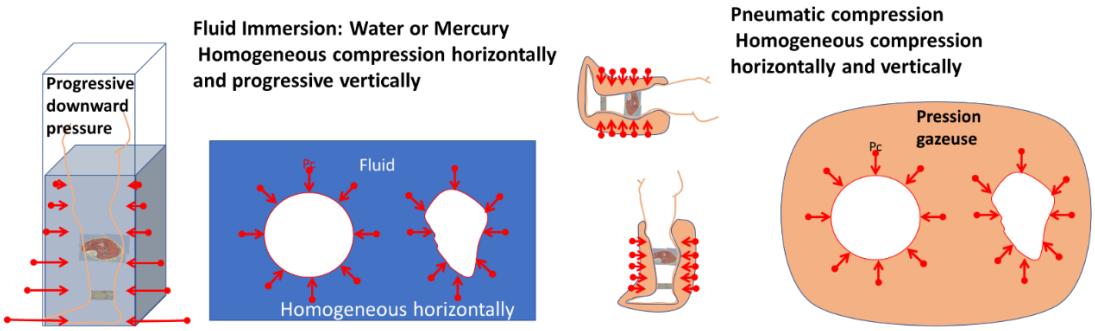
***Compression** must **consider** the shape, volume, and compressibility of the limb tissues.*

*The **distribution and homogeneity of compression** varies according to the **external compression medium** used and the compression/elasticity modulus of the leg.*

81231-Homogeneous compression:

812311-Immersion in a heavy liquid (water, mercury) creates a homogeneous circumferential compression that progresses vertically from top to bottom like the GHSP. *As we have seen, the **sensation of feeling lighter in water does not mean that the body is lighter, but that, in accordance with Archimedes' law, the weight of the water is distributed over the entire surface of our body instead of bearing entirely on our feet when we are out of the water**. In the same way, the **superficial veins decrease in size in water not because the column of Hydrostatic Gravitational pressure GHSP is lighter than in the open air,** which would be magic, but because the hydrostatic pressure of the water in the pool compresses them in proportion to their depth.*

812312- A sleeve inflated with air exerts a circular and homogeneous compression independent of the position and therefore of gravity**. It can be uneven if the sleeves are staggered and inflated differently. When it is **inflated and then deflated intermittently,** it produces **a pumping effect like the valvulo-muscular pumps.



81232- *Heterogeneous compression.*

<u>The transmission of pressure to the veins and capillaries of the leg with elastic or inelastic bandages is uneven due to the uneven distribution of the modulus of compressibility/elasticity of its tissue</u>.

Compressive bandages transmit a pressure force P which they transmit in accordance with <u>Laplace's Law which states the "counterintuitive" fact that, for the same tension force T, it is stronger when the radius r of the limb is smaller</u>*. Laplace's Law: T=RP. Otherwise, bandaging with the same tension force the ankle and the thigh, transmits more pressure in the first than in the second.*

The pressure transmitted is equal everywhere if the bandaged segment is perfectly circular. *If the segment has an irregular Superficial, the pressure varies with the tension as the radius of curvature of each bumpy irregularity decreases (Laplace)****<u>. Again, the irregularity of the limb can be corrected by pads that fill the hollows to make it more homogeneous and circular.</u>***

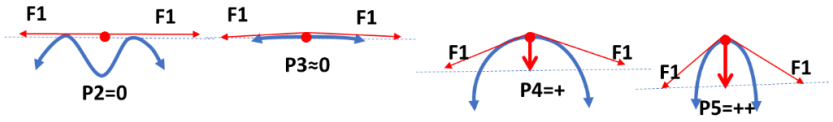
One can ***avoid compressing the arteries****, especially the pedal artery****, by affixing pads on each side*** *that prevent them from being in contact with the bandage.*

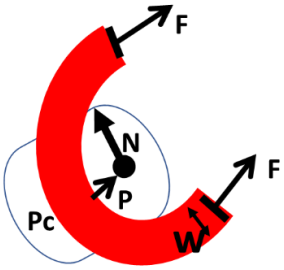
<u>In all cases, the risk of ischemia must be feared and prevented by ensuring that the Doppler or plethysmographic flow of the forefoot is equal to that recorded before compression.</u>

LAPLACE's law
Pressure P against the skin
F: tension force F
w: width of the band
N: Nadius of the cylinder Pc
Transmitted pressure = F/wN = F/N when w=1cm
P: hecto Pascal, F: Newton 1mmHg = 1,333 hector Pa = 1,359 cm H^2O = 0,00131 atm

INHOMOGENEOUS COMPNESSION

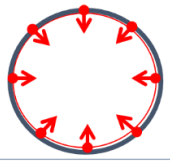
Bandage





P5>P4>P3>P2: Pressures transmitted by the tension F according to N.

depends on the arc angle where the compression is applied



Circular:
Homogeneous Transmission

Not Circular :
Inhomogeneous Transmission

:

Bandage compression: LAPLACE'S LAW

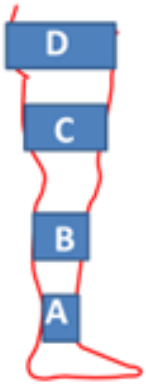
Pressure = F/wr = F/r when w=1cm

For the same stretch force F, the sub-bandage pressure P decreases when the average radius of the leg increases

For the **same stretch force** , the resulting **sub-bandage decreases** from the ankle up to the buttock

A>B>C>D

SO:Compression above the knee is inefficient and useless



812321- *Non-elastic band compressions:*

Non-elastic band compressions prevent volume increase beyond that achieved by tightening.

They exert a ***passive resistive back pressure equivalent to the internal pressures*** *that tend to increase the volume of the limb.*

Any further increase in internal pressure that cannot be absorbed by the change in volume (pressure/volume ratio), increases the tension/pressure of the bandage accordingly.

This explains why calf compression increases sharply during walking, *when the body of the muscles grows as it shortens.* ***Therefore it is called "working" compression.***

On the other hand, ***when the volume decreases, the compression force decreases (pressure/volume ratio).***

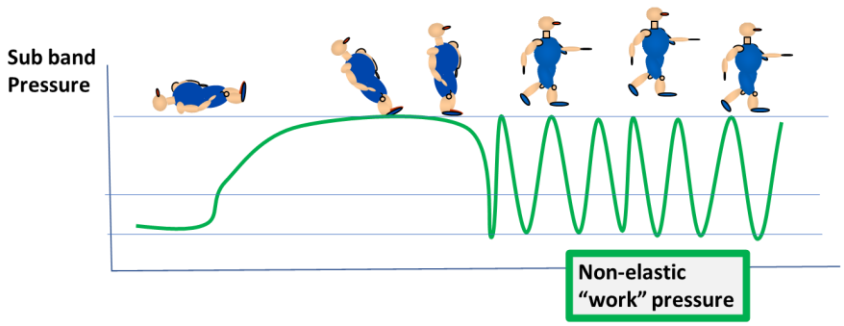
This is the case in the laying position (resting position and not working position), because the intravenous pressure is greatly reduced by the considerable drop in GHSP, which in turn reduces the volume, and consequently the bandage pressure that can lower down to 0. Moreover, compression *exerts little back pressure in the supine position, which reduces the risk of decubitus ischemia*

Therefore, ***non-elastic compression*** *is* ***more effective*** *and* ***less dangerous*** *than* ***elastic compression.***

Non-elastic compression

Non-elastic bandage is a passive support because it does not exert any active pressure. It is a resistive force to the pressure produced by limb when its volume tends to overwhelm the volume of the bandage.

Thus, the non-elastic and non-stretchable bandage resists the volume/pressure of the limb and returns it. This happens especially during walking.



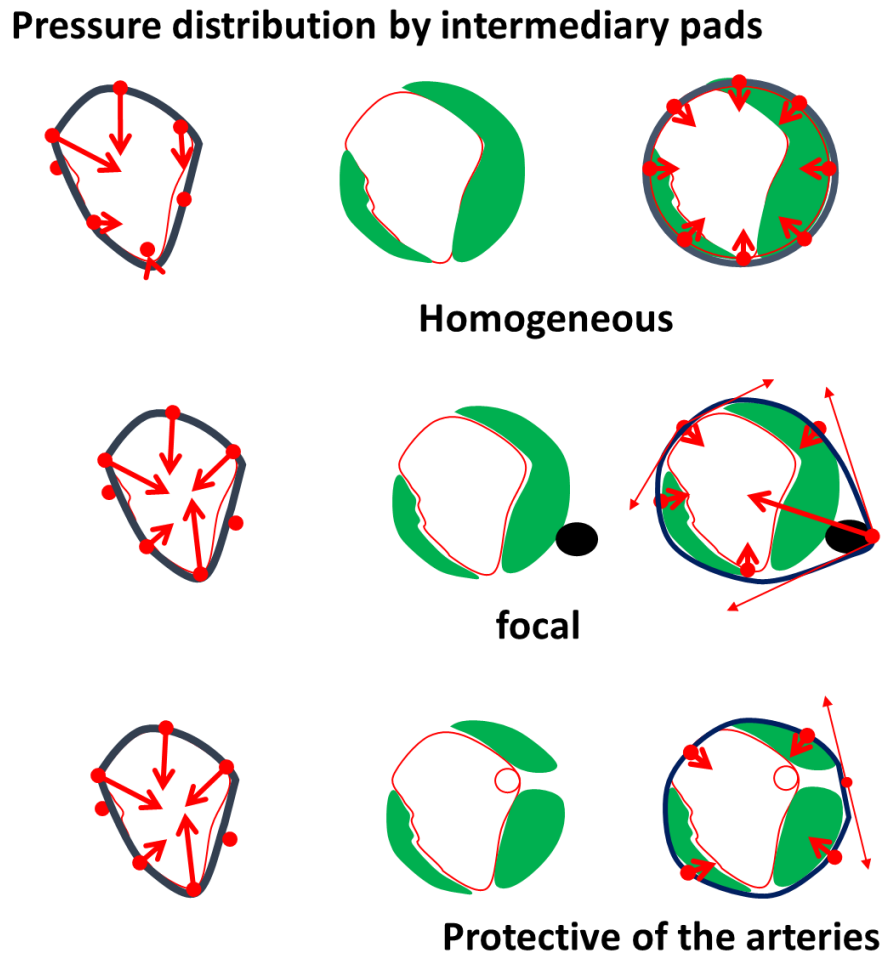
It is criticized for losing its effectiveness during the day**. The reason is not the stretch** *of the bandage, but the reduction of the oedema favoured by this bandaging that increased the drainage.*

Therefore, ***I recommend an initial reduction of the oedema*** ***by laying down for 2 hours,*** *with the* ***leg elevated by 50 cm*** *and* ***bandaged*** *with a* ***slightly tight elastic bandage. So, edema is dramatically reduced and the bandage can be kept for at least 1 week without pressure loss.***

The disadvantage of non-elastic compression is that it is less adaptable to limb irregularities.

This can be corrected by ***pre-filling with pads the*** *hollows to make the limb more homogeneous and circular.*

Alternatively, a compromise can be sought in the form of a semi-elastic bandage.



812322- *Elastic band and stocking compressions:*

Elastic compressions are not passive but active *in the sense that, when stretched, they* ***exert a shortening force until their resting length is restored.*** *This elastic force is equal to the force that had to be applied to stretch it (Young's modulus).*

This compression does not have the same therapeutic virtue as non-elastic compression *because,* ***for a compression in the supine position equal to that of non-elastic compression****, it will be* ***less compressive when walking*** *than the latter.*

Indeed, its elasticity *allows the volume to increase, which* ***reduces the "working" effect of the non-elastic support.***

Moreover, ***its permanent elastic activity continues to act in decubitus when the arterial pressure is much lower than in standing position (decreased arterial GHSP) with the risk of ischemia especially in case of associated arteriopathy.***

The advantages *of elastic support are its* ***better conformability*** *to the irregularities of the limb surface and its* ***use as a stocking.***

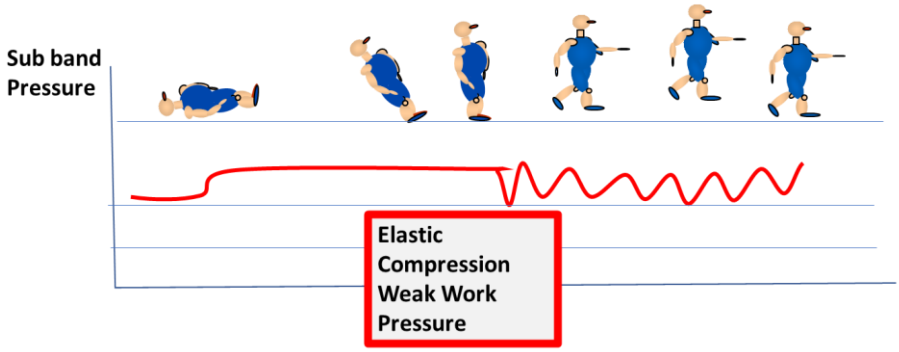
Therefore, it is preferable for all cases where the TMP is not too high, i.e. most often.

Here too, the irregularity of the limb can be corrected by filling pads in the hollow parts to make it more homogeneous and circular.

*Here again****, compression of*** *the arteries, especially the* ***pedal artery,*** *should be* ***avoided by applying pads on each side*** *to prevent them from to be pressed by the bandage****.***

Elastic band and stocking compression

Elastic compression is not passive but active due to its potential shortening force (Hysteresis) and stores instead of resisting part of the pressure volume variations of the limb.

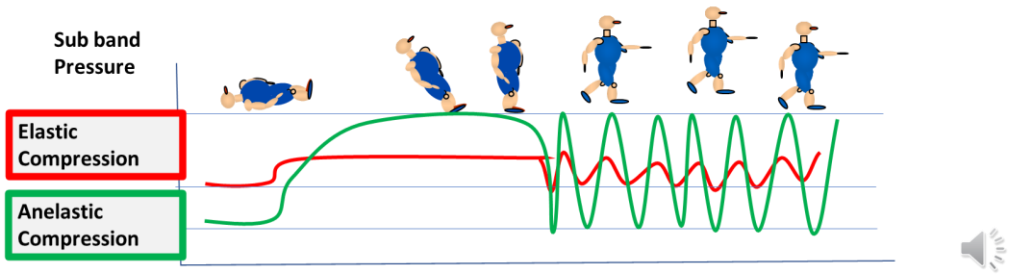


Anelastic and elastic compression does not have the same therapeutic virtue.

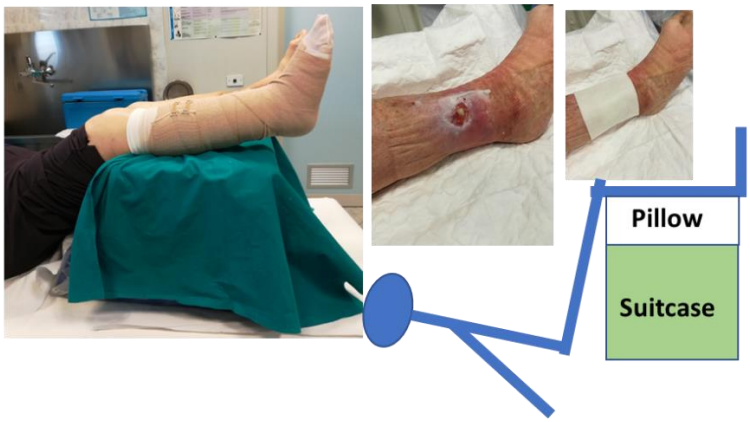
For a lesser sub-bandage pressure in lying position, anelastic bandage reduces more Transmural pressure than elastic bandage during standing and walking.

It reduces the risk of ischemia in lying position, which is essential in case of associated arteriopathy.

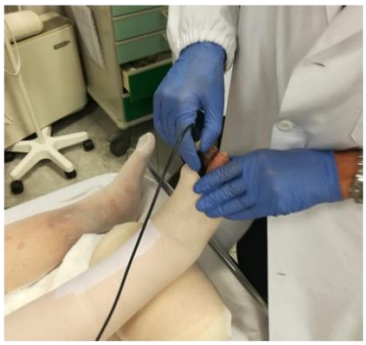
Nevertheless, due to its better conformability , elastic compression remains preferable for all the cases where the TMP is not too high, which is fortunately the most frequent case.



Non-elastic compression after 2 hours of elevation + light elastic compression



Distal arterial Doppler control



CHIVA is French acronyme of cure"conservatrice et hémodynamique de l'insuffisance veineuse en ambulatoire » i.e Conservative and Hemodynamic Treatment of Venous Insufficiency in the ambulatory setting

Why the CHIVA Cure and no other methods?

I do not deal here with destructive methods based on classical physiopathological concepts which are disproved by rational hemodynamic analysis.

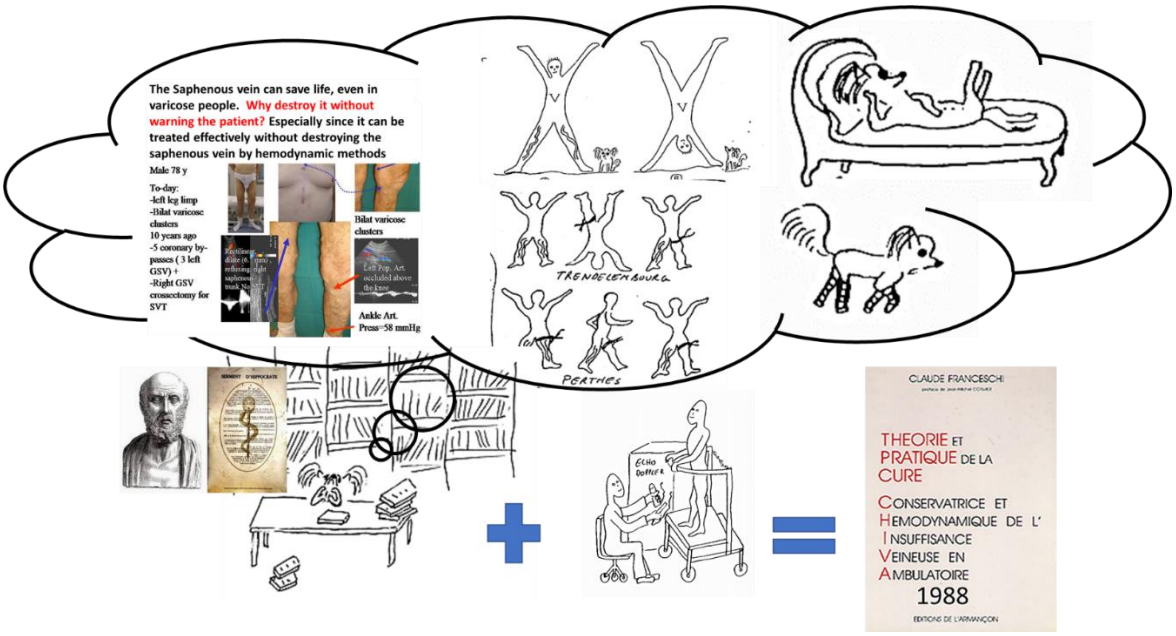
According to these "classical" concepts, varicose veins and refluxing veins must be destroyed and/or occluded by open surgery (stripping and phlebectomies) or by endovenous route (sclerotherapy, laser, radiofrequency, glue, ultrasound). The complications (telangiectasias, matting) and recurrences are either the recanalisation of the obstructed veins or the occurrence of other compensatory anarchic varicose veins These destructive methods are condemnable for ethical and medico-legal reasons.

My research on venous physiopathology was motivated by my desire to avoid destroying, to "treat" a benign pathology, the great saphenous vein GSV which, although incompetent, can save a life or a limb in case of need for venous by-pass.

The value of venous by-pass surgery remains major despite the progress of prostheses and arterial deobstruction and stenting procedures, both in terms of medium- and long-term efficacy (no-touch harvesting technique for aorto-coronary bypasses equivalent to Aorto Coronary bypasses Ref: 1-No touch technique of saphenous vein harvesting: Is great graft patency rate provided? Papakonstantinou NA J Thorac Cardiovasc Surg. 2015 Oct;150(4):880-8. doi: 10.1016/j.jtcvs.2015.07.027. Epub 2015 Jul 15. 2-The no-touch saphenous vein for coronary artery by-pass grafting maintains a patency, after 16 years, comparable to the left internal thoracic artery: A randomized trial.Samano N1, :ClinicalTrials.gov NCT01686100.Copyright © 2015 The American Association for Thoracic Surgery) ***and by-passes below the knee superior to endo-vascular procedures, stents and prostheses .*** ***Ref:***1-Meta-analysis of infrapopliteal angioplasty for chronic critical limb ischemia.Marcello Romiti, (J Vasc Surg 2008;47:975-81.) 2-Meta-analysis of popliteal-to-distal vein by-pass grafts for critical ischemia Maximiano Albersand (J Vasc Surg 2006;43:498-503.)

I therefore proposed a non-destructive hemodynamic method that I called CHIVA treatment Conservative and Hemodynamic Treatment of Venous Insufficiency in the ambulatory setting in 1988, CHIVA is not a technique but a strategy in accordance with a physiopathological model based on venous hemodynamics, which has not been sufficiently taken into account until now.

Since then, it has been the subject of more than 120 articles and a few books and has been validated by controlled RCTs and Cochrane reviews as superior to destructive methods. ***(see chapter 9)***



The informed consent that I give to patients is as follows

The saphenous vein is vital for future peripheral and coronary venous bypasses and should not be destroyed without the informed consent of the patients, especially as this benign disease can be treated simply by compression socks or minimally invasive surgical methods (CHIVA cure), which are less expensive, ALWAYS conservative and validated (CHIVA), better than destructive surgical (stripping) or endovenous techniques (sclerosis, foam, laser, Radiofrequency C).

The Great Saphenous Vein (GSV) in healthy subjects but also in varicose veins (the saphenous veins removed from a patient to treat his varicose veins are frozen by a Lyon-based company (BioProtec) and then sold to carry out bypasses on another patient) can be removed for a coronary or limb by-pass.

Scientific studies:

A- Coronary venous by-pass equivalent to mammary artery by-pass

1-No touch technique of saphenous vein harvesting: Is great graft patency rate provided?

Papakonstantinou NA J Thorac Cardiovasc Surg. 2015 Oct;150(4):880-8. doi: 10.1016/j.jtcvs.2015.07.027. Epub 2015 Jul 15.

2-The no-touch saphenous vein for coronary artery by-pass grafting maintains a patency, after 16 years, comparable to the left internal thoracic artery: A randomized trial.Samano N1, :ClinicalTrials.gov NCT01686100.Copyright © 2015 The American Association for Thoracic Surgery. Published by Elsevier Inc. All rights reserved.

B-Venous by-pass of the lower limb arteries is still the most effective. -

1-Meta-analysis of infrapopliteal angioplasty for chronic critical limb ischemia

Marcello Romiti, (J Vasc Surg 2008;47:975-81.)

2-Meta-analysis of popliteal-to-distal vein by-pass grafts for critical ischemia

Maximiano Albersand (J Vasc Surg 2006;43:498-503.)

D- The CHIVA method gives less recurrence than destructive methods (Stripping, Laser, radiofrequency, sclerosis, foam).

C- CHIVA cure

1-CHIVA method for the treatment of chronic venous insufficiency.Bellmunt-Montoya S1, Cochrane Database Syst Rev. 2015 Jun 29;(6):CD009648. doi: 10.1002/14651858.CD009648.pub3

2-Hemodynamic classification and CHIVA treatment of varicose veins in lower extremities (VVLE)Hua Wang1, et al, China. Int J Clin Exp Med 2016;9(2):2465-2471 www.ijcem.com /ISSN:1940-5901/IJCEM0016552"".

3- Carandina S, Mari C, De PAlma M, Marcellino MG,Cisno C, Legnaro A, et al.Varicose Vein Stripping v sHaemodynamic Correction (CHIVA): a long term randomised trial. European Journal of Vascular and Endovascular Surgery 2008;35(2):230-7

4- PArés JO, Juan J, Tellez R, Mata A, Moreno C, Quer FX,et l.Varicose vein surgery: stripping versus the CHIVA Method: a randomized controlled trial. Annals of Surgery 2010;251(4):624-31

5- Iborra-Ortega E, Barjau-Urrea E, Vila-Coll R, Ballon-Carazas H, Cairols-Castellote MA. ComPArative study oftwo surgical techniques in the treatment of varicose veins of the lower extremities: results after five years of followup. Estudio comPArativo de dos técnicas quirúrgicas en el tratamiento de las varices de las extremidades inferiores: resultados tras cinco años de seguimiento]. Angiología 2006; 58(6):459-68.

6-] P. Zamboni and all: Minimally Invasive Surgical management of primary venous Ulcer vs. compression Eur J vasc Endovasc Surg 00,1 6 (2003)

7- Chan, C.-Y.a , Chen, T.-C.b , Hsieh, Y.-K.a , Huang, J.-H.c Retrospective comPArison of clinical outcomes between endovenous laser and saphenous vein-sPAring surgery for treatment of varicose veins (2011) World Journal of Surgery, 35 (7), pp. 1679-1686

8- The CHIVA method gives less recurrence than destructive methods (Stripping, Laser, radiofrequency, sclerosis, foam). Guo et al. Long-term efficacy of different procedures for treatment of varicose veins A network meta-analysis. Medicine (2019) 98:7

831- ***CHIVA treatment Definition: . Ref :*** C. Franceschi. *La cure Conservatrice et Hémodynamique de l'Insuffisance Veineuse en Ambulatoire CHIVA* Editions de L'Armançon. 1988 Précy sous Thil France 2-

French acronyme for - Conservatrice et Hémodynamique de l'Insuffisance Veineuse en -Ambulatoire

-Conservative and

-Hemodynamic treatment of

-Venous insufficiency in

-Outpatient setting,

Cure CHIVA is

It aims to normalize the Transmural pressure TMP by

1-Reducing the Lateral Intravenous pressure IVLP,

2-Restoring the dynamic fractionation of the gravitational hydrostatic pressure DFGHSP and

3-Suppressing the flow and pressure overload by the

a-closed shunts CS and

b-open deviated shunts, when walking and

c-respecting the compensatory vins formed by the Open Vicarious shunts OVS.

4-Preserving the Great saphenous as eventual vital grafting.

It meets the requirements of conservation and its hemodynamic corollary *because conservation is necessary for hemodynamic correction and hemodynamic correction is necessary for conservation.*

It is ambulatory because it can almost always be performed by minimally invasive surgery under local anaesthesia. Sclerosis may be used in specific hemodynamic conditions, especially when the surgery is not technically feasible. This aspect will be explained at Chapter 10 by Massimo Cappelli.

Ist pathophysiological model regards also deep venous insufficiency called CHIVP.video. Sperfifcial CHIVA: https://www.youtube.com/watch?v=SDVnHCmJBf0&t=5s
Deep CHIVA: https://www.youtube.com/watch?v=t6vGDEwx9XI&t=230s

832-Indications:

8321-Informed consent.

*First, it is a question of obtaining the informed consent of the patient who has been previously informed according to the model I presented at the beginning of this chapter**, in particular the conservation of the great saphenous vein in order not only to reduce recurrences but above all to save a graft for possible arterial by-pass.***

8322-Mistaken indications.

The CHIVA treatment *must only respond to signs and symptoms directly linked to venous insufficiency****, i.e., after having eliminated all other non-venous causes such as arterial ulcers, necrotizing microangiopathies, cytosteatonecrosis, inflammatory oedema, osteo-muscular and neuropathic pain.***

8323- Pelvic escape points can be disconnected directly without prior embolization of the gonadal or deep pelvic veins in the absence of a clinical syndrome of pelvic congestion.

Ref: Delfrate R, Bricchi M, Franceschi C. Minimally invasive procedure for pelvic escape points in women. Veins and Lymphatics. 2019; 8:7789, 10-16.

Pelvic embolization may be considered first in the case of true pelvic congestion syndrome and secondarily in the case of recurrent pelvic escape points.

Note that pelvic embolization reduces reflux, which even when it has become Valsalva negative, *continues to overload the superficial veins of the lower extremities*

8324-Aesthetics.

The treatment must also respond to the cosmetic demand by any surgical or medical means (sclerosis) ***but refuse procedures that destroy the great saphenous vein or are hemodynamically deleterious,*** *which are responsible for recurrence or even immediate or delayed aggravation.*

833- CHIVA method

The method is Strategic, which considers the rational to treat the venous function impairment essentially according to the type of the veno-venous shunts, and Tactical, which defines the specific technical actions intended to achieve the objectives of the strategy.

8331- Strategy. Video CHIVA: https://www.youtube.com/watch?v=SDVnHCmJBf0&t=5s

Deep CHIVA: https://www.youtube.com/watch?v=t6vGDEwx9XI&t=230s

83311-Fractionation of the incompetent column at the level of the escape points and under the leg re-entries above the re-entry point of the terminal re-entry perforator (Cappelli) to correct the defect of Dynamic Fractionation of the Gravitational Hydrostatic pressure DFGHSP.

83312-Disconnection of closed CS and open deviated shunts ODS at the escape point. N1>N2, N2>N3 and N1>N3

83313- Preservation of open vicarious shunts OVS, including those made by varicose recurrences after destructive treatments.

83314-Elimination of non-draining varicose veins.

83315-Conservation of the Great Saphenous Vein GVS.

Avoid fractionation of the great saphenous vein GSV at the level of the thigh to keep all its by-pass potential.

83316- Restrain the number of disconnection to the necessary (between 1 and 5) because an excessive disconnection cannot be corrected whereas a missing disconnection can be completed. In fact, the lower the number of disconnections, the better the diagnosis and the hemodynamic strategy. The Perthes maneuver is invaluable for realizing the efficiency with which sometimes enormous varicose veins can be collapsed, to the great surprise of the patient but also of the novice physicians..

833161: -Shunt I

Great Saphenous Vein: Disconnection flush the femoral vein

Small Saphenous Vein: Disconnection below the Giacomini connection to

Avoid unnecessary traumatic dissection

leaves behind a SSV arch flow (thrombosis and deep cavernoma prevention).

833162: SHUNT II

Tributary N3 Disconnection flush trunks GSV and SSV N2

833163: SHUNT III

2 strategies: 2 steps or 1 step.

2 steps:

First step: N2>N3 disconnection.

Second step: N1>N2 disconnection only when and if a N2 trunk re-entry has developed.

One step:

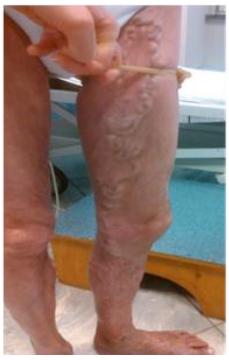
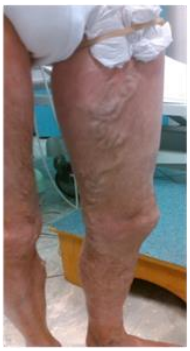
Simultaneous N1>N2 and N2>N3 disconnection

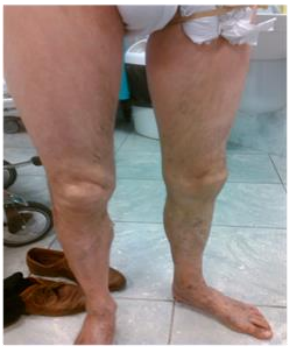
+ Devalvulation of the N2 below the N3>N2 disconnected junction to allow the N2 flow reach a re-entry below.

833164: SHUNT IV, V and V:

Shunt disconnection at the escape point and non-absorbable suture of the fascia.

Clinical maneuvers:Perthes Manoeuver checks the deep venous system status according to the varicose veins collapse during walking with a thigh tourniquet and estimates the clinical outcoms of CHIVA disconnection without any venous ablation



83317- Apply a class 2 support with thigh stockings for 2 weeks with immediate walking under anticoagulants in preventive doses.

83318-post-operative follow-up and monitoring

PATIENCE!

Wait for the progressive remodelling of the varicose veins to normal calibres at rest as shown during the preoperative Perthes manoeuvre.

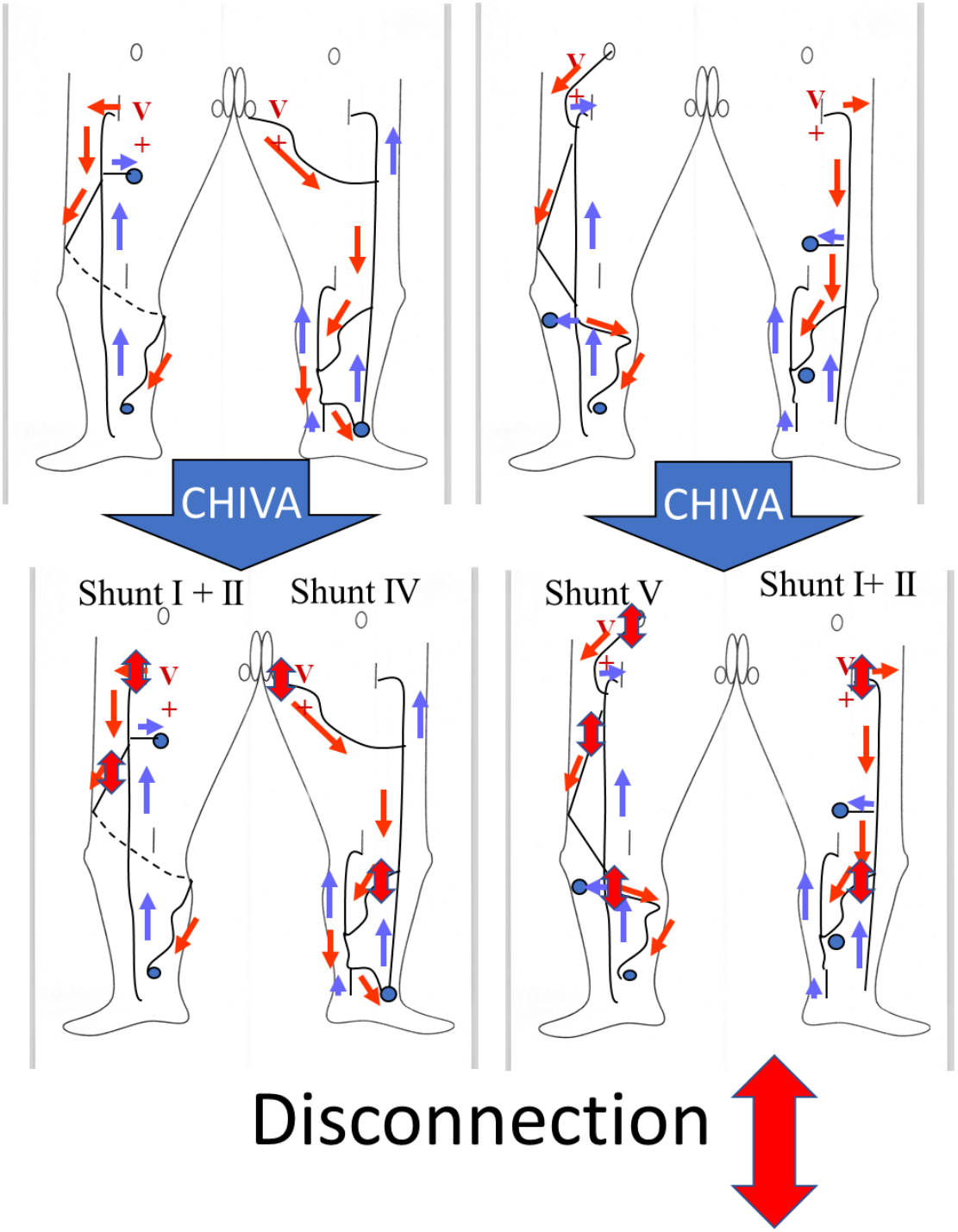
The patient should be informed that this delay may last a few weeks and that it is recommended not to do anything else unless an escape point was ignored or badly disconnected during the operation (Valsalva + if an escape point N1>N2 or N1N3 persists. I remind you that a retrograde flow (reflux) without an escape point is a shunt 0, a sign of a good result of the CHIVA treatment!)

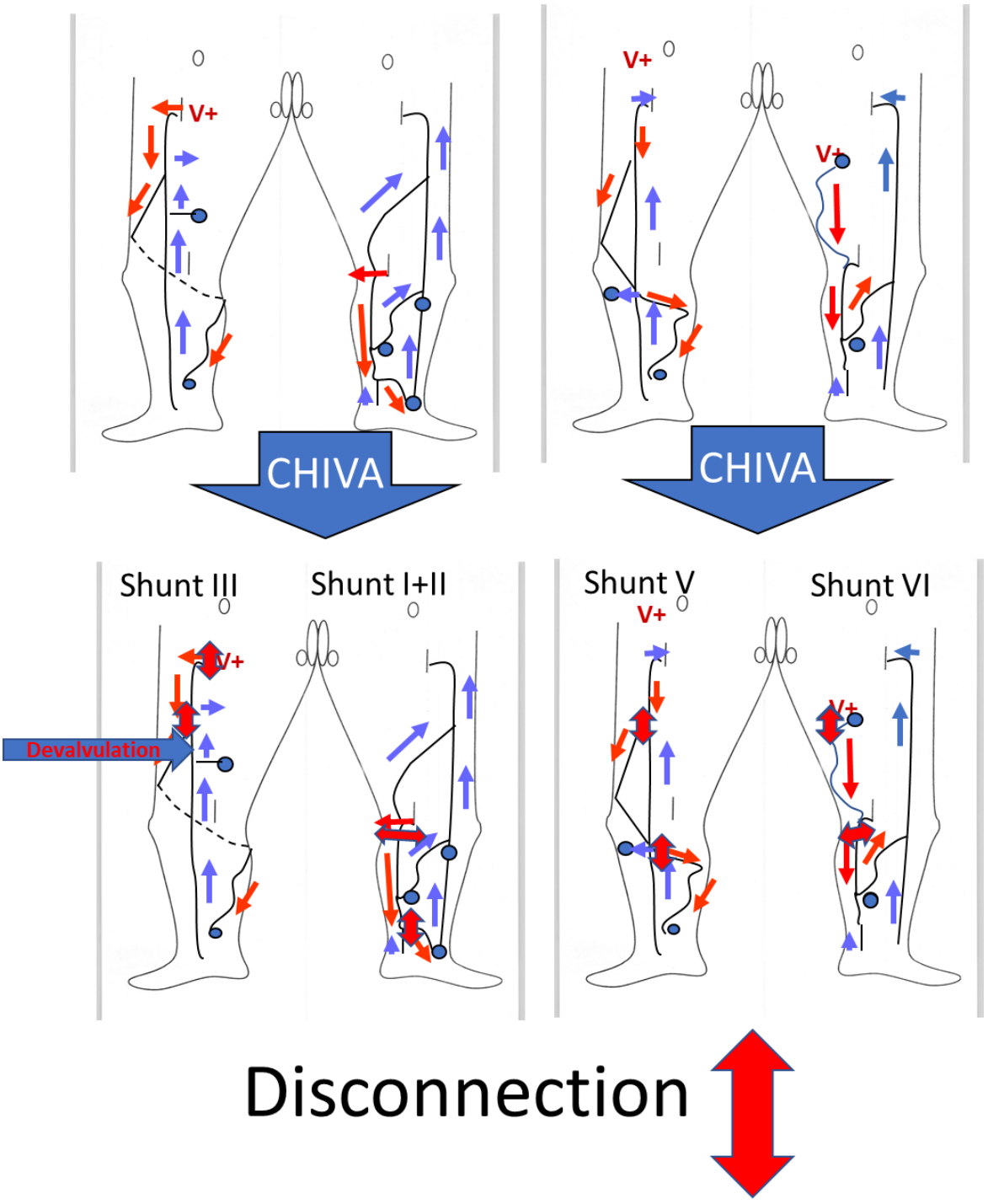
83319-Cartography (mapping) and CHIVA strategy

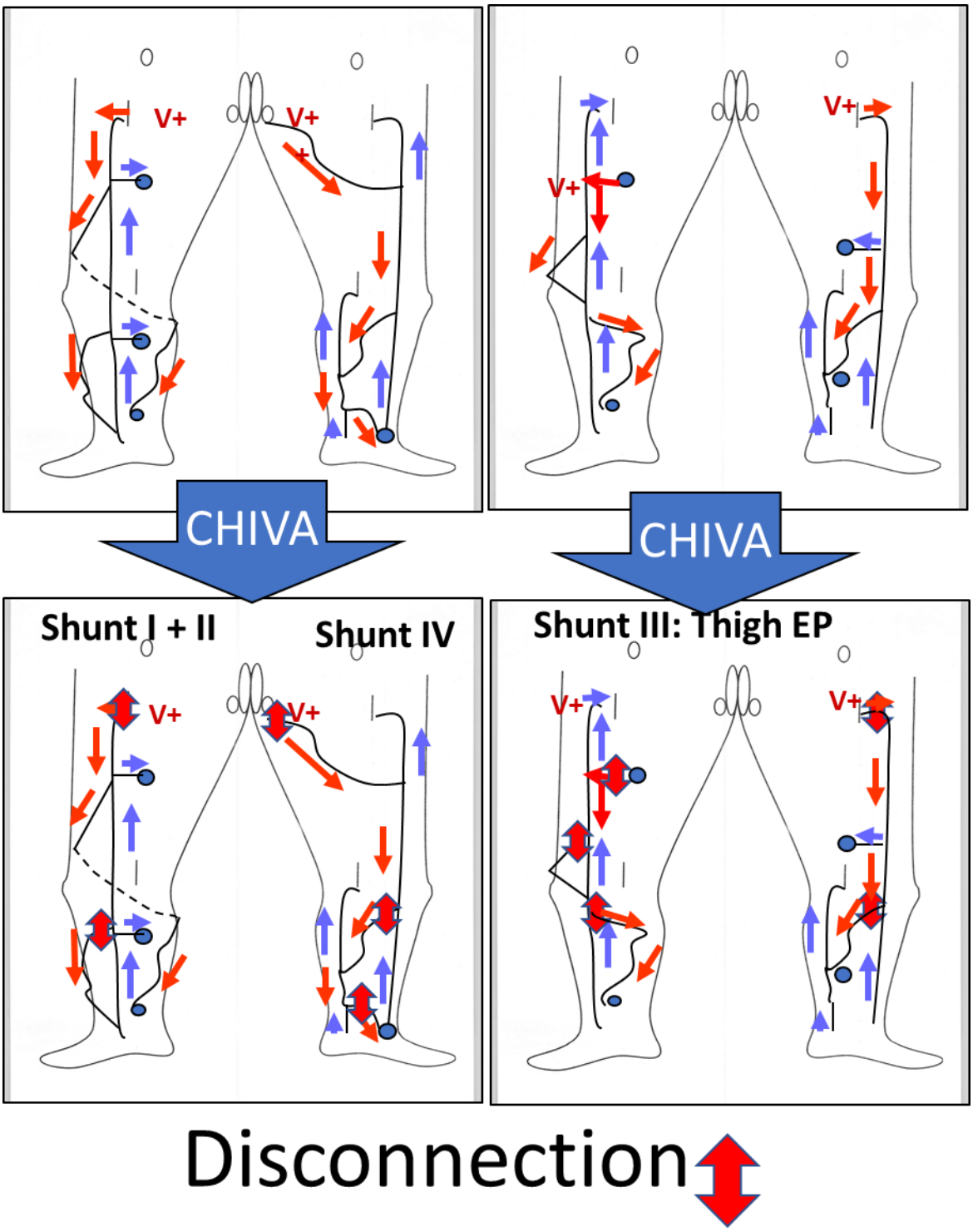
Again, Cartography is a topographic and hemodynamic mapping of the shunts with their pathways, escape points EP and re-entry points RP to establish the best strategy and propose the best tactics to achieve it.

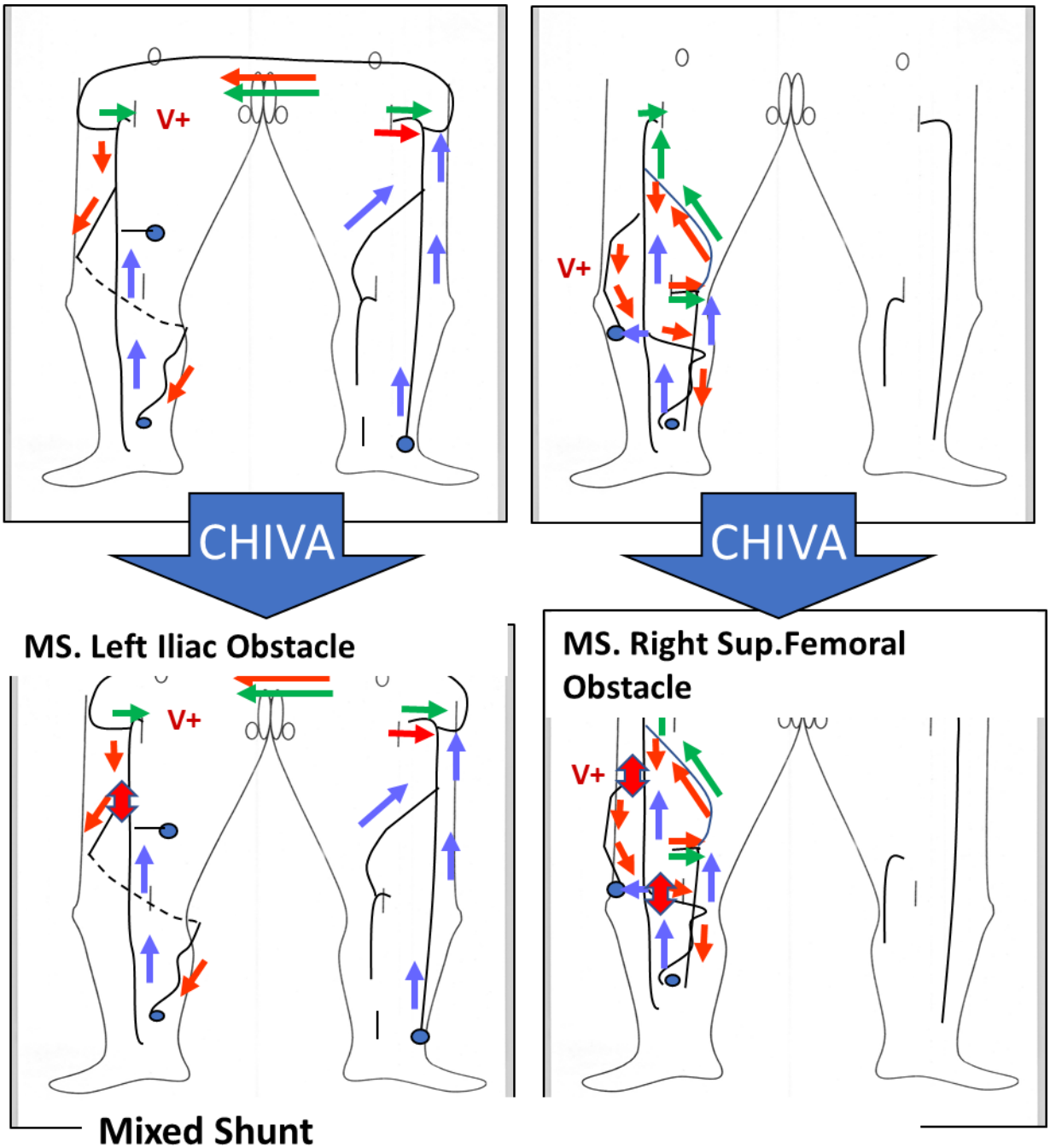
It is obvious, that CHIVA without such mapping CANNOT BE CHIVA. This core of CHIVA must be performed by who is expert in dedicated Echodoppler and related pathophysiology. Pretending to play chess with the rules of checkers is an illusion that leads to confusion *: Ref* Milone M, Salvatore G, Maietta P, Sosa Fernandez LM, Milone Recurrent varicose veins of the lower extremities after surgery. Role of surgical technique (stripping vs. CHIVA) and surgeon's experience.F. G Chir. 2011 Nov-Dec;32(11-12):460-3.

Here is a collection of mappings provided by CHIVA experts and their strategies shown by signs where the disconnection is elected.


CHIVA
CHIVA
Shunt I + II
Shunt IV
Shunt V
Shunt I+ II
Disconnection


V+
CHIVA
V+
V+
CHIVA
Shunt III
Shunt I+II
V+
Devalvulation
Shunt V
Shunt VI
V+
V+
Disconnection


V+
V+
V+
V+
CHIVA
CHIVA
Shunt I + II
Shunt IV
Shunt III: Thigh EP
V+
V+
V+
V+
Disconnection

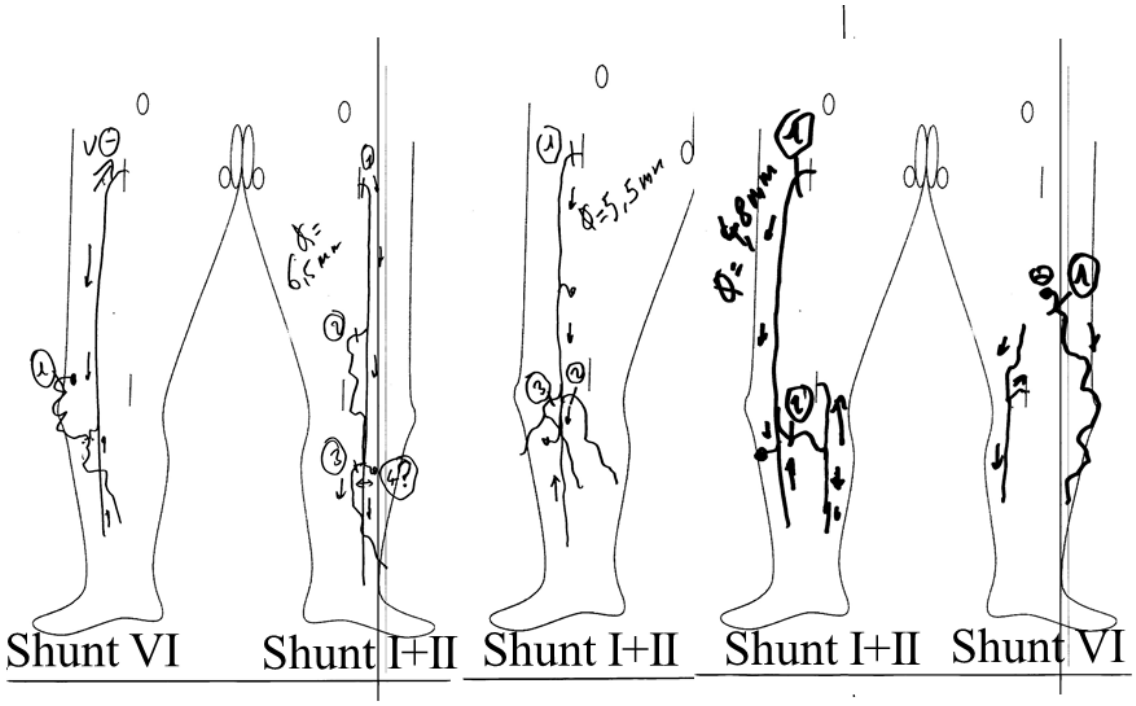


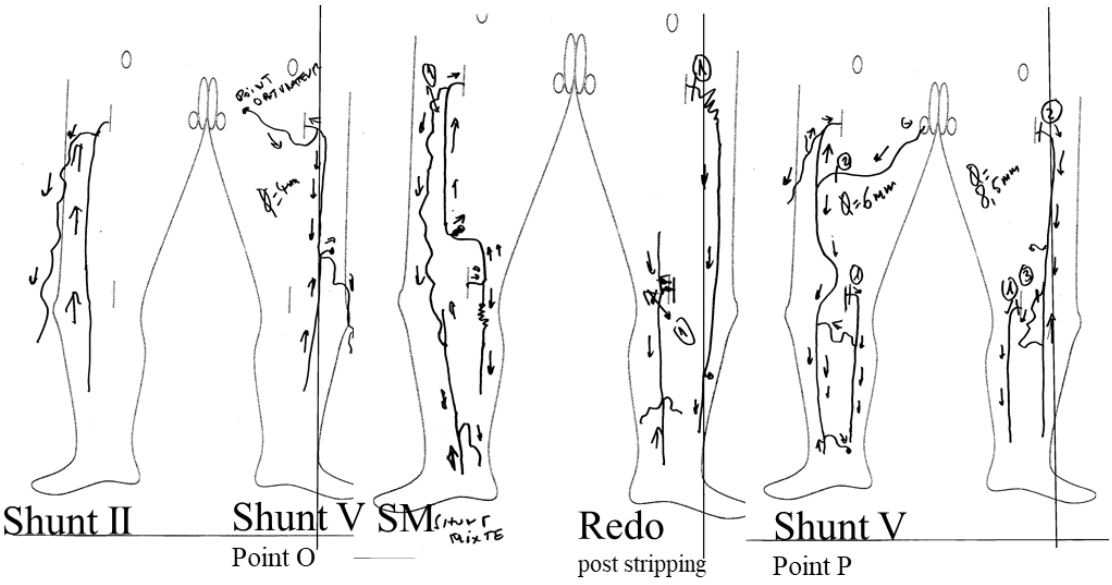
Mixed Shunt

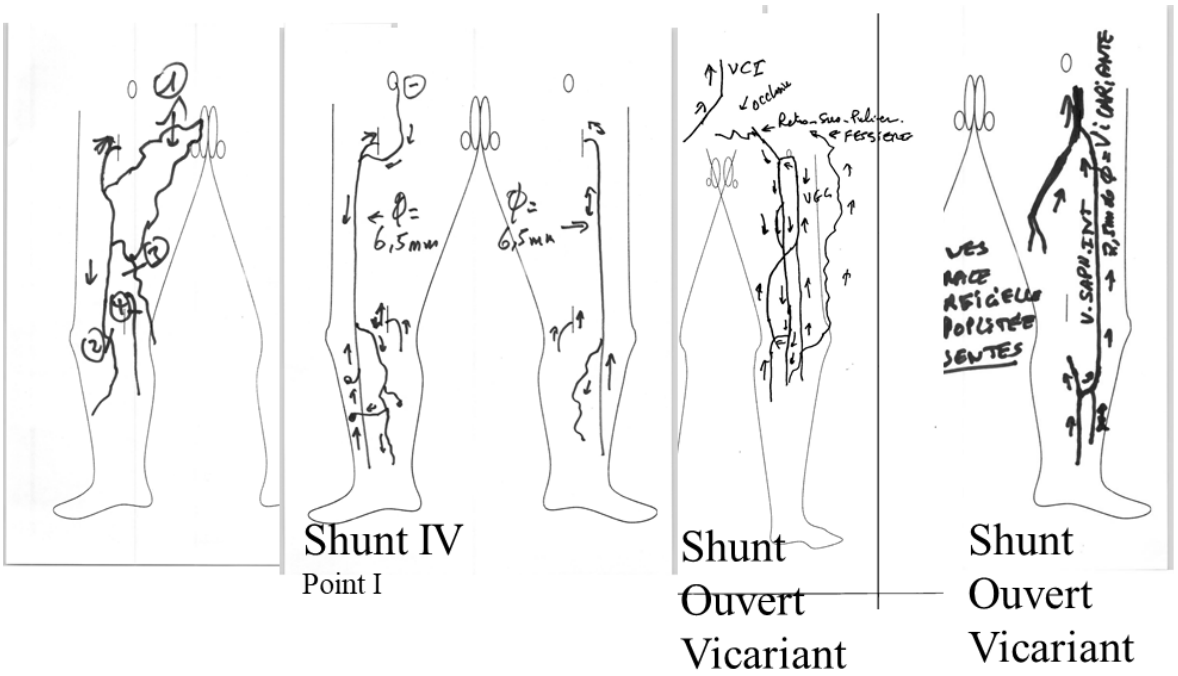
Red : Diastolic Closed shunt

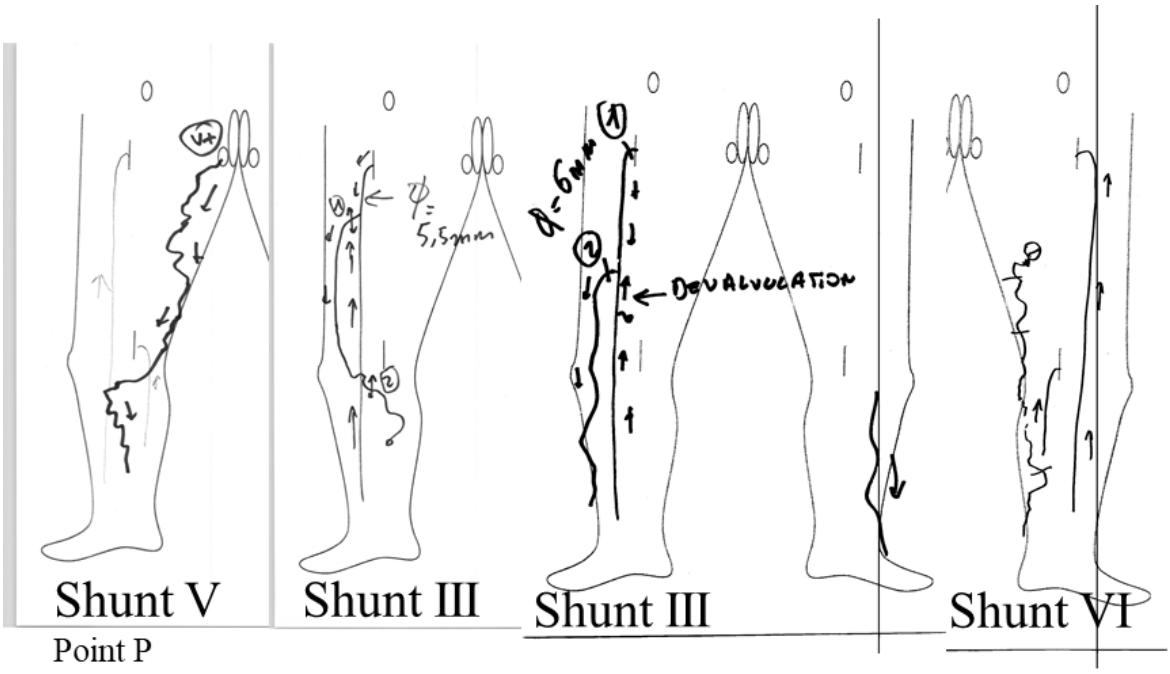
Green: systolic Open Vicarious Shunt

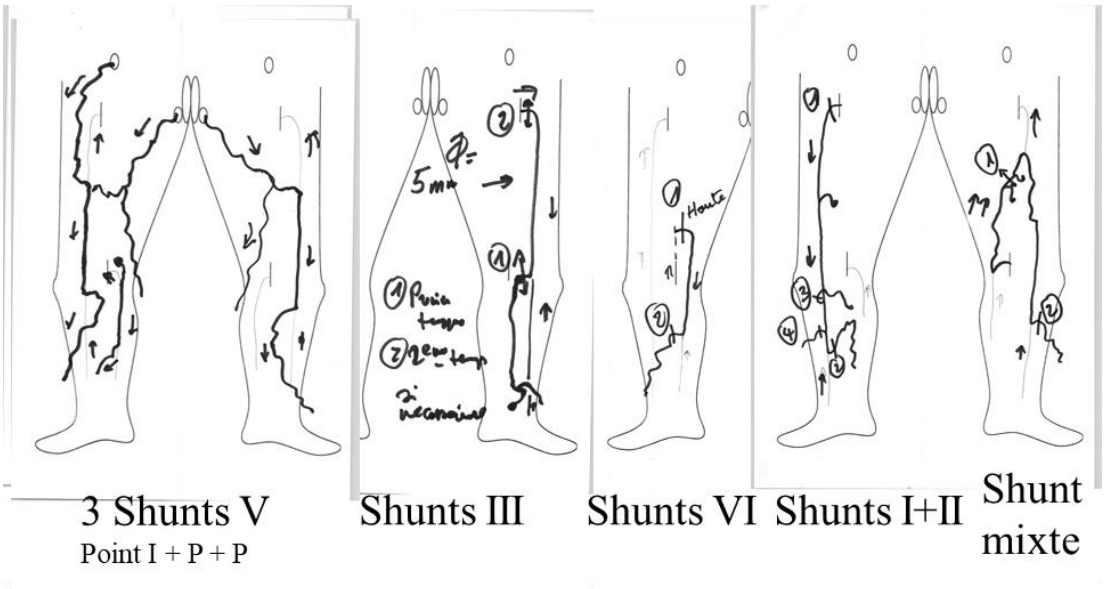
Disconnection

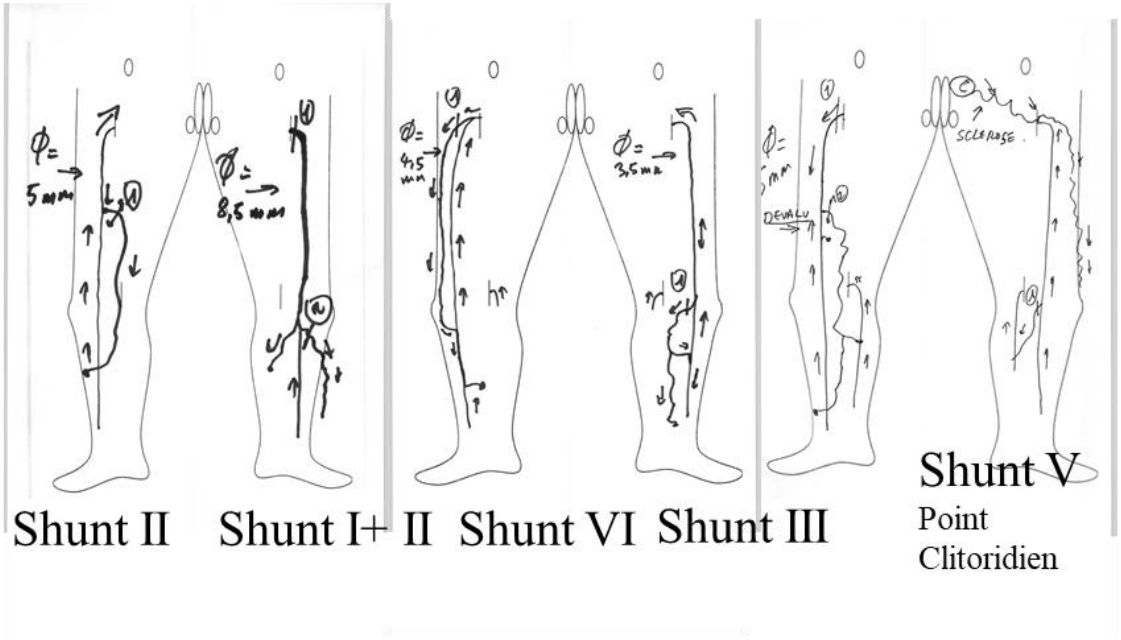

Shunt VI
Shunt I+II
Shunt I+II
Shunt I+II
Shunt VI


Shunt II
Shunt V
Point O
SM
Redo
post stripping
Shunt V
Point P


Shunt IV
Point I
Shunt
Ouvert
Vicariant
Shunt
Ouvert
Vicariant
VCI
Occlusion
Ø= 6,5mm
Ø= 6,5mm
VES NAGE AFICIELLE POPLITÉE JENTES
V. SAPH. INT

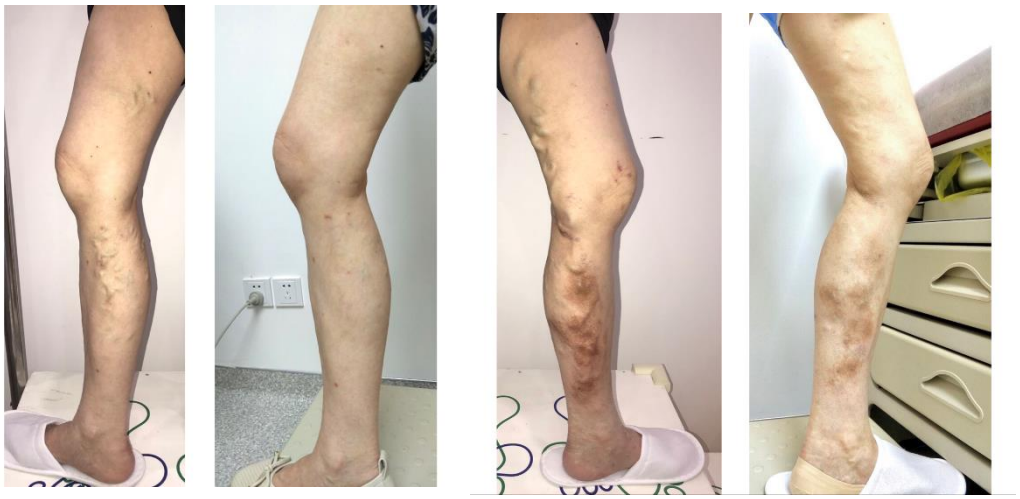

Shunt V
Point P
Shunt III
Shunt III
Shunt VI
Ø= 5,5mm
Ø= 6mm
DEVALVUCATION

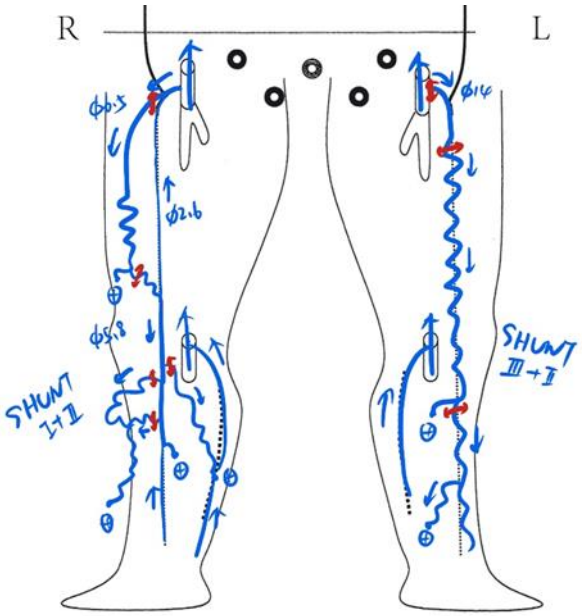

3 Shunts V
Point I + P + P
Shunts III
Shunts VI
Shunts I+II
Shunt mixte


Shunt II
Shunt I+ II
Shunt VI
Shunt III
Shunt V
Point Clitoridien

SE 1

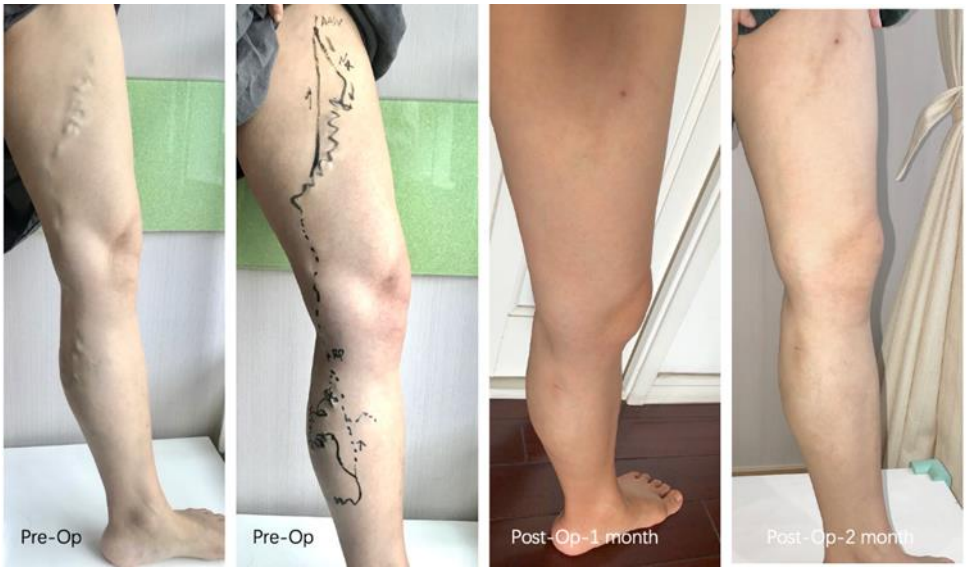
A 70-year-old female Presented with varicose veins in both legs for over 10 years. She had chronic eczema and skin pigmentation of the left calf. No DVT history. Perthes test showed varicose veins totally collapsed. ***Hemodynamic ultrasound showed SHUNT 3 in left and SHUNT I+II in right. CHIVA was performed at both legs in one surgery. 10 months follow-up showed varicose veins collapsed and skin improved.***

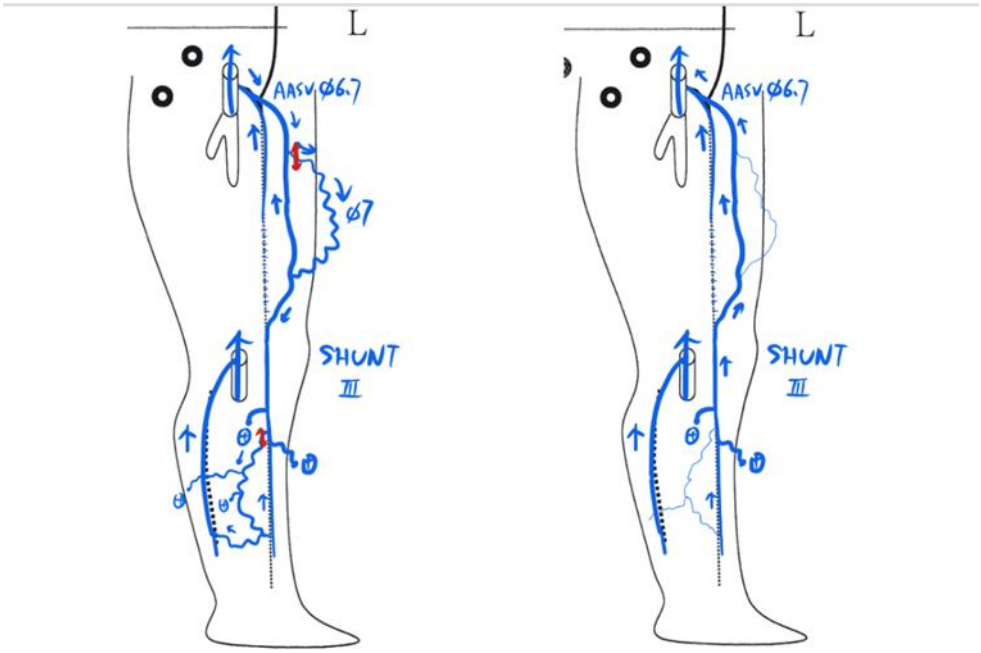




CASE 2:

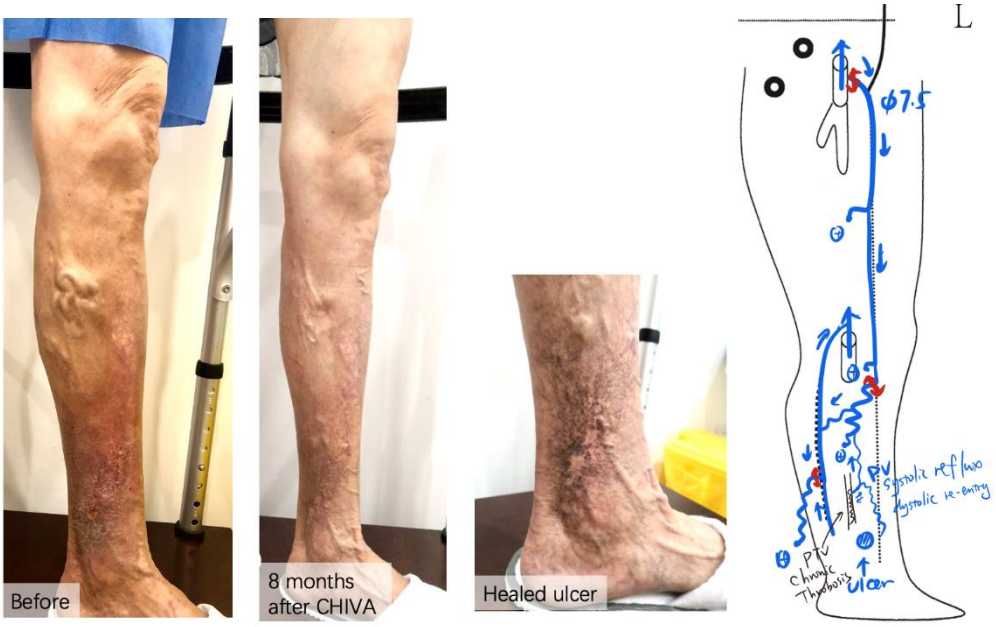
A 48-year-old female presented with varicose veins and leg oedema in left leg for over 5 years. Hemodynamic ultrasound showed SHUNT III in left leg. SFJ, anterior accessory saphenous vein (AASV) and its tributary were incompetent. CHIVA2-step1 was performed. The patient was checked by duplex ultrasound in 2 months after operation and the SFJ and AASV repented competent. After 2 years follow up, it showed no need to perform step 2.





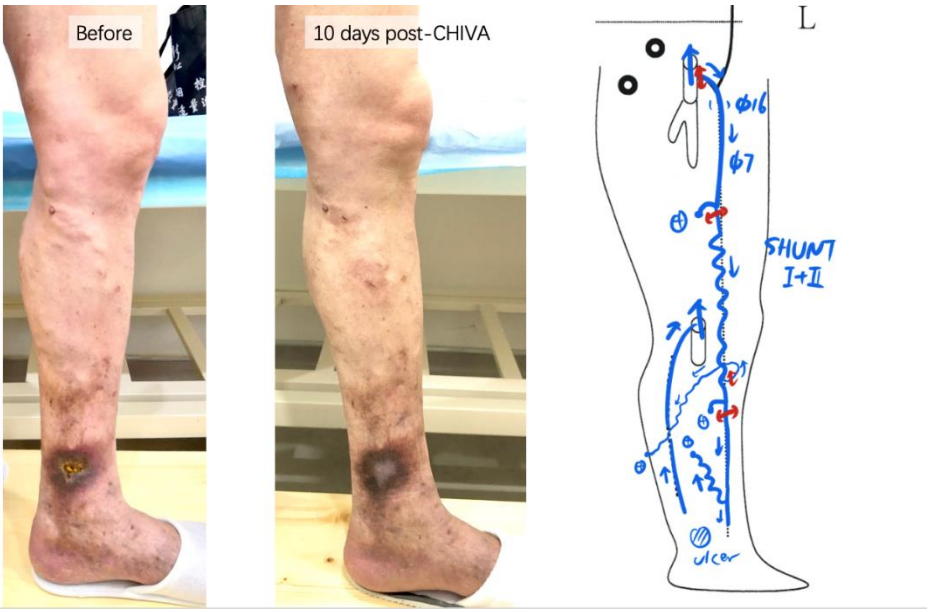
CASE 3:

A 62-year-old female presented with varicose veins for 16 years and recurrent venous ulcer for 1 year. Hemodynamic ultrasound showed SHUNT I+II in left leg. SFJ, GSV and its tributaries were incompetent. Obstacle was found in post tibial vein and systolic reflux tested in a perforator nearby. CHIVA was performed without disturbing GSV and the reflux perforator. The ulcer was healed in 2 weeks post operation.



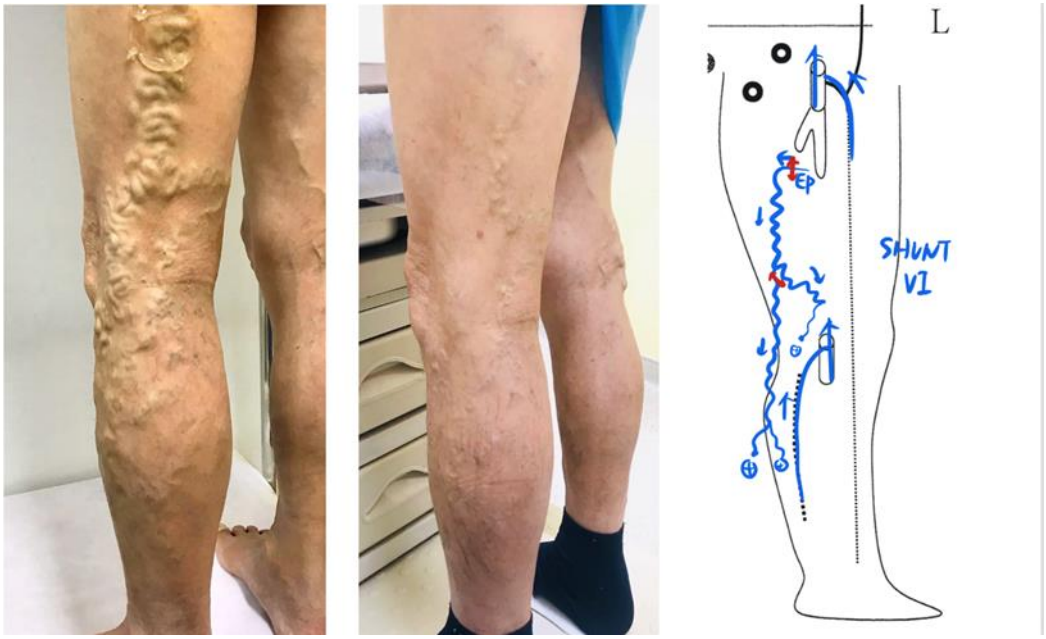
CASE 4:

A 70-year-old male repented with unhealed venous ulcer for 2 years. Hemodynamic ultrasound showed SHUNT I+II in left leg. CHIVA was performed with pressure column fragmentated. The ulcer was healed in 10 days after CHIVA procedure.



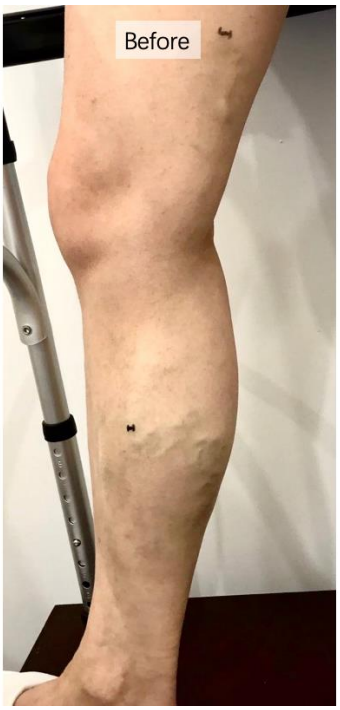
CASE 5:

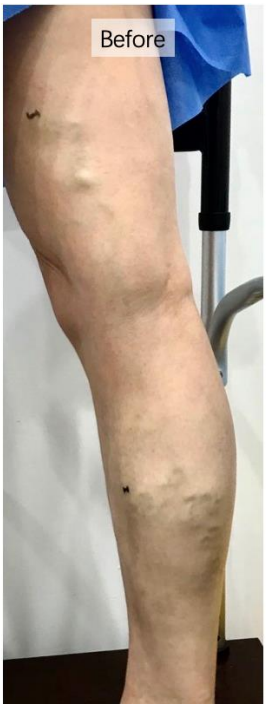
A 69-year-old male presented with severe varicose veins in the lateral side of left leg. Hemodynamic ultrasound showed a diastolic reflux perforator dilated on lateral thigh and repented SHUNT VI. CHIVA was performed with closure of escape point and fragmentation of pressure column. Bulged varicose veins were collapsed in 6 months.

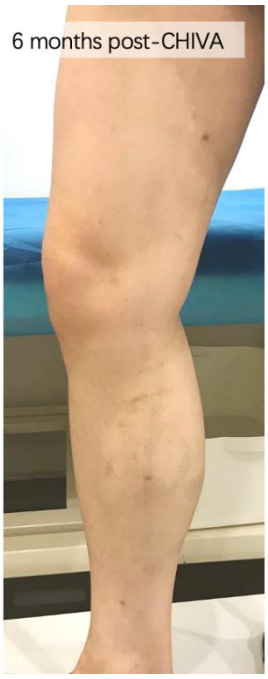


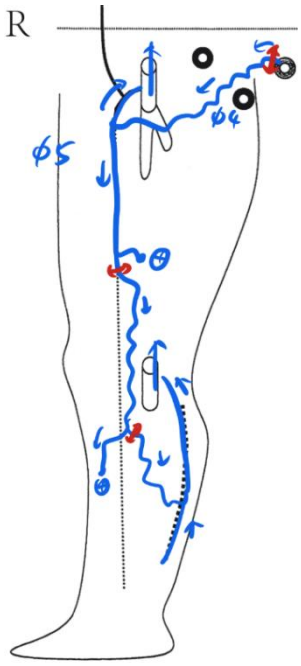
CASE 6:

A 37-year-old female presented with varicose veins for 7 years after delivery. She felt swelling and Pain of varicose veins during the period. Hemodynamic ultrasound showed pelvic escape in CP and presented as SHUNT VI. CHIVA was performed with closure of escape points. Symptoms improved after procedure.


Before

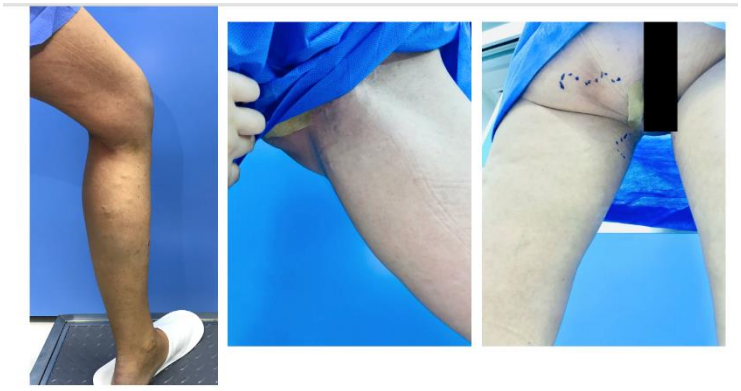

Before

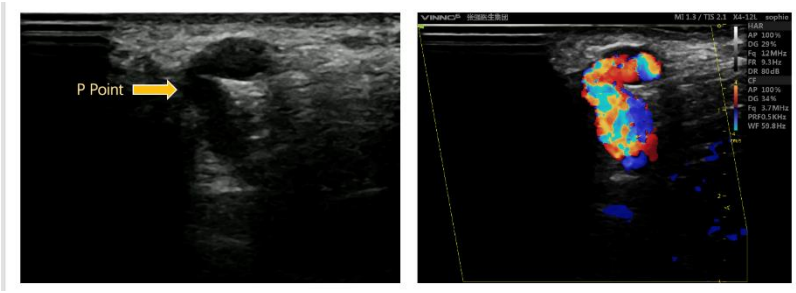

6 months post-CHIVA

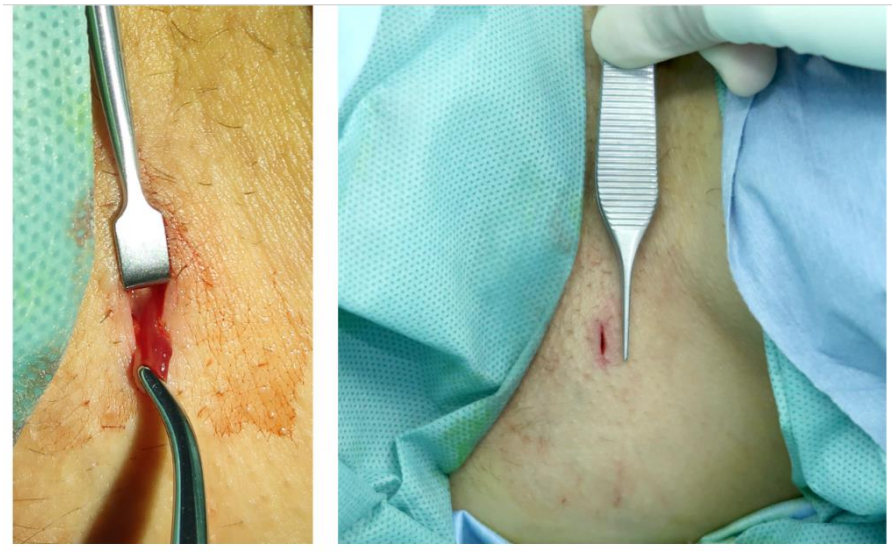

R

CASE 7:

A 45-year-old female presented with perineal and calf varicose veins for 1 years with Pain of varicosities in standing position. Hemodynamic ultrasound showed pelvic escape in P point and presented as SHUNT VI. CHIVA was performed with closure of escape points. Pain disappeared after the procedure.

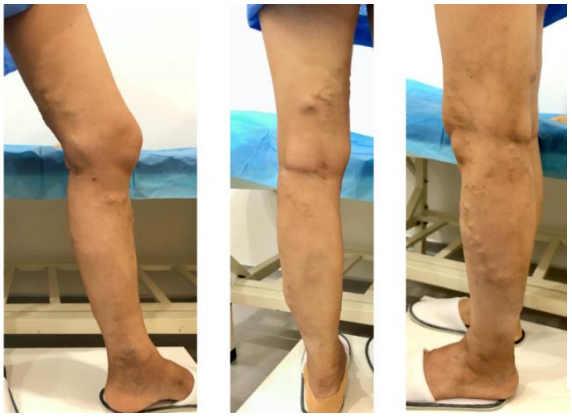


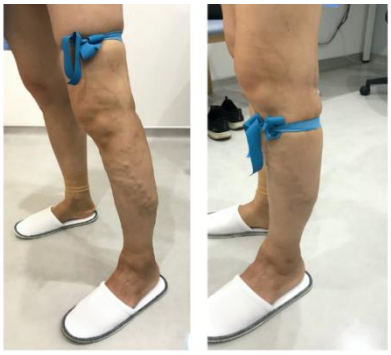




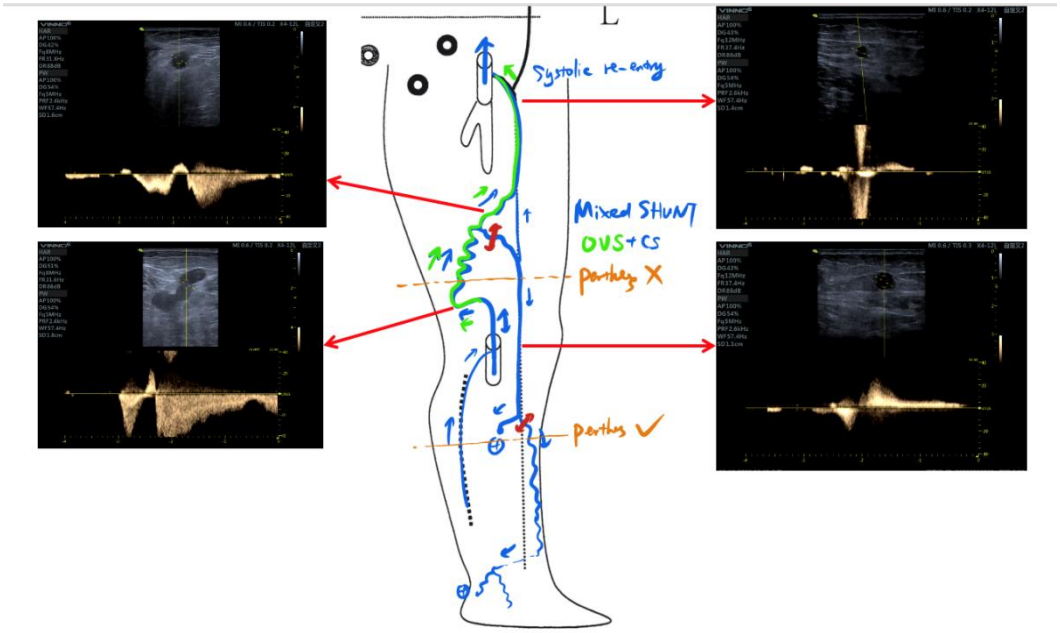
CASE 8:

A 64-year-old female presented with varicose veins in left calf for over 30 years. She felt heaviness in calf after long time standing. She had a history of leg swelling and Pain which wasn't diagnosed and treated after a procedure for ectopic pregnancy 15 years ago. Several years later varicose veins were found at posterior thigh. Hemodynamic ultrasound showed a combination of open vicarious shunt and closed shunt. Femoral vein was Patient and mild incompetent. CHIVA was performed with open vicarious shunt preserved and closed shunt disconnected.



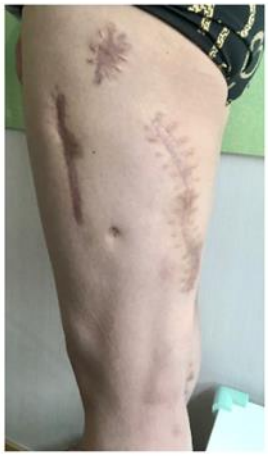


A. Elastic band was performed above knee where open vicarious shunt was pressed. After the patient walking for 30 steps, the calf varicose veins became more bulged. B. Elastic band was performed below knee where only closed shunt was pressed. The varicose veins disappeared after walking.

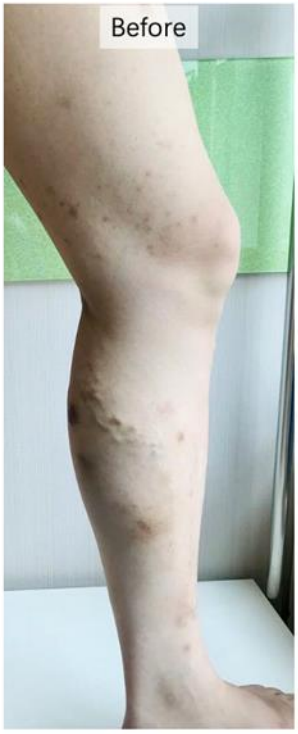


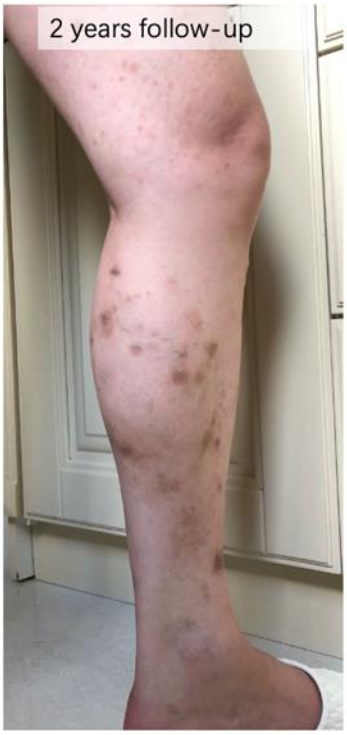
CASE 9:

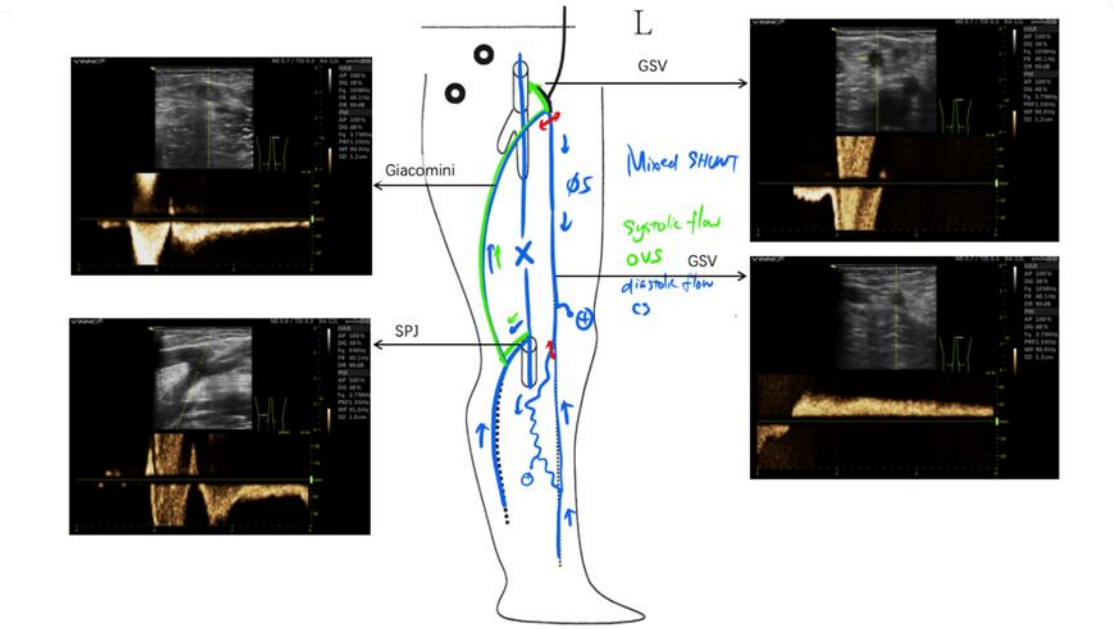
A 35-year-old male presented with varicose veins. He had a history of stab wound 13 years ago. Hemodynamic ultrasound showed MIXED SHUNT and femoral vein obstacle. CHIVA was performed with open vicarious shunt preserved and closed shunt disconnected.

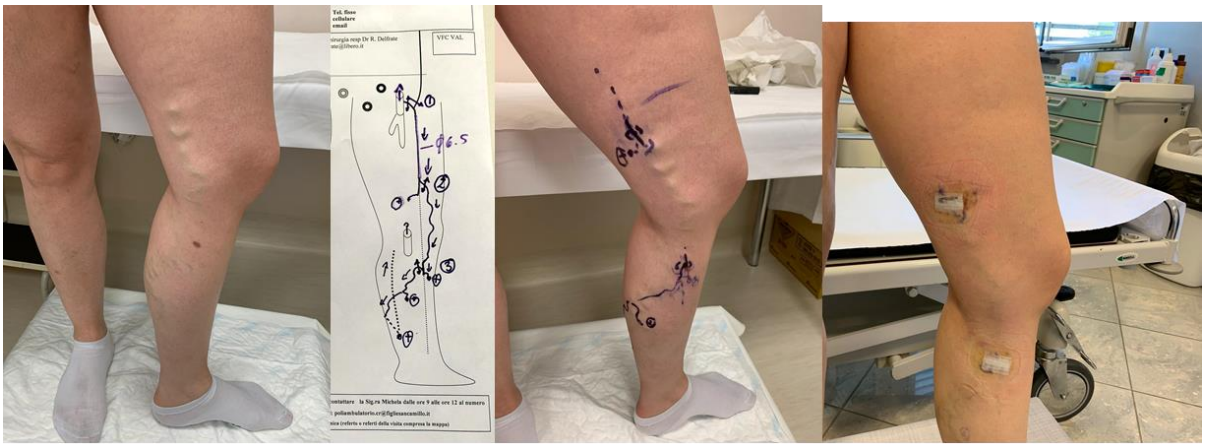


Patient had an history of stab wound 13 years ago

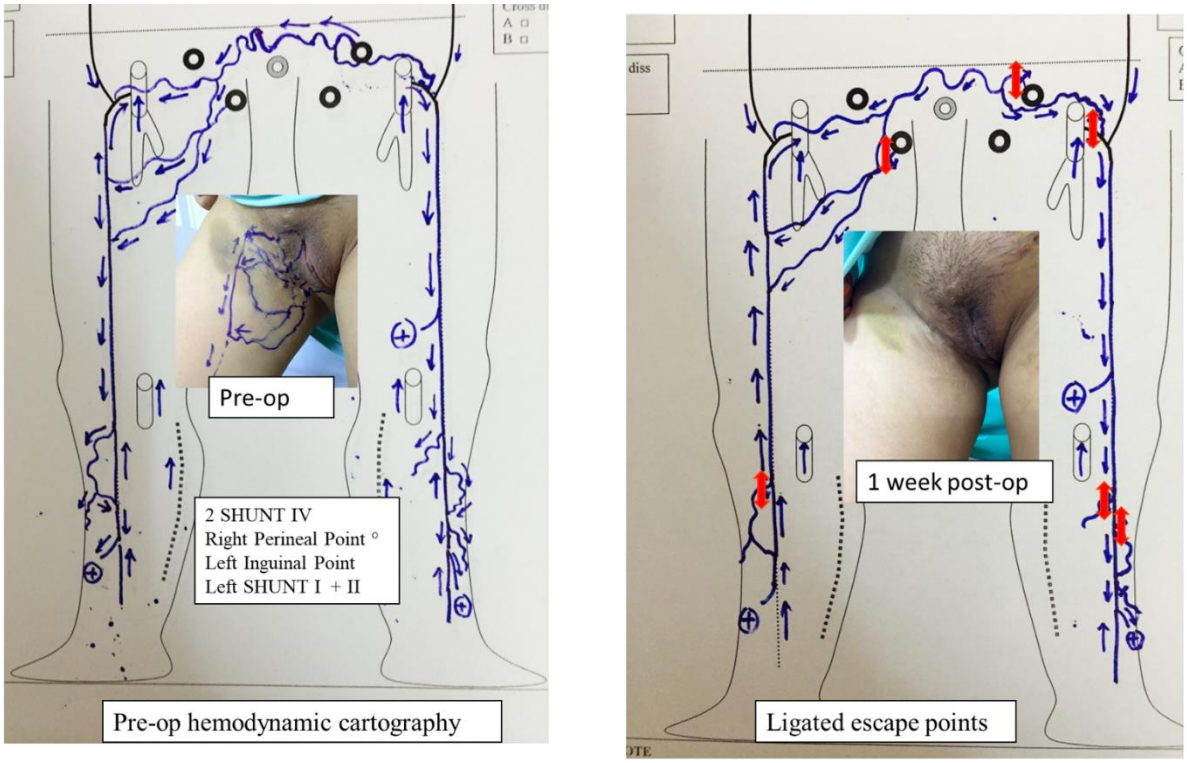








Shunt I + II Dr Roberto Delfrate Cremona Italy



Dr Le Thanh Phong Hô Chi Minh-Ville Vietnam

8332- ***Tactics***

Surgery is easier, less invasive, and faster when guided by preoperative precise marking and intraoperative ultrasound.

The shortest possible incisions are made at the marks under local anesthesia.

83321-The hemostator is a fast, efficient and haemostasis tool.

It is particularly useful in case of intraoperative injury of the small and large saphenous veins.

It is made of stainless-steel ring slightly open at the tip of a handle (see below).

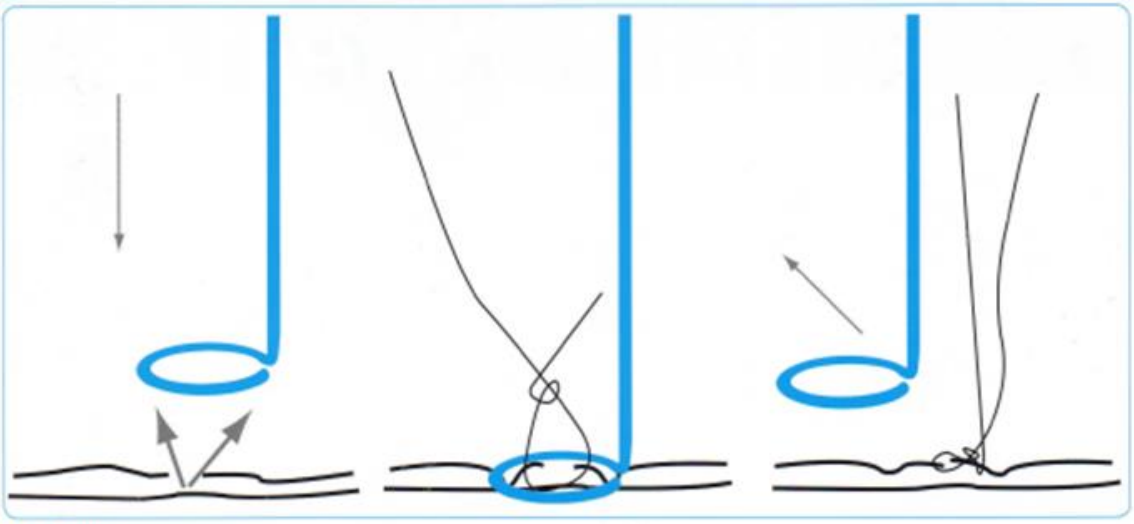
In case of bleeding difficult to control, it is sufficient to press the ring (variable diameter: 1,2,3,4cm) against the source of the bleeding*. The vessels surrounding the vascular wound are clamped by the ring which compresses them, thus stopping the bleeding. The vascular wound is dried in the centre of the ring where it can be easily sutured. A small opening in the ring allows hemostator to be removed without cutting the thread. This tool is also very useful in any vascular or non-vascular surgery,* ***to control bleeding that is difficult to access or that floods the operating field. For example,*** *it can be used to reduce bleeding in vascular malformations surgery. Another example is the control of lumbar artery bleeding in abdominal aortic aneurysm surgery.*

Note that I have released the patent and that the hemostator can therefore be easily copied*.*

Dr Franceschi's HEMOSTATOR®

Specifically designed for accidental per-operative arterial or veinous haemorrhages control, when vital prognosis or major risking complications are at stake.

Spécialement conçu pour contrôler une hémorragie peropératoire par plaie veineuse ou artérielle accidentelle, pouvant mettre en jeu le pronostic vital ou engendrer des complications majeures.



The facts:

> Emergency vascular suture is a very risky and stressful situation
> Veins are fragile, less visible, and more difficult to dissect and clamp than arteries
> Simply using the finger or dressing gauze that stop the bleeding hide the vessel
> Pressure required to compress an aorta : only 1,5 kg

The Specifications:

> 3 diameters for ideal fitting to the vessel size
> The ring is lightly opened to allow the clearing of suture thread
> The handle is oriented for a perfect control of the ring position
> The hemostator is in flexible steel, allowing esay torsion to obtain any desired angule

Les faits :

> Une plaie vasculaire accidentelle provoque une situation stressante à haut risque
> Les veines sont plus fragiles, moins visibles et plus difficiles à disséquer et clamper que les artères
> L'utilisation du doigt ou d'une compresse pour arrêter le saignement cache le vaisseau
> La pression nécessaire pour comprimer efficacement une aorte est seulement de 1,5 kg

Les specifications :

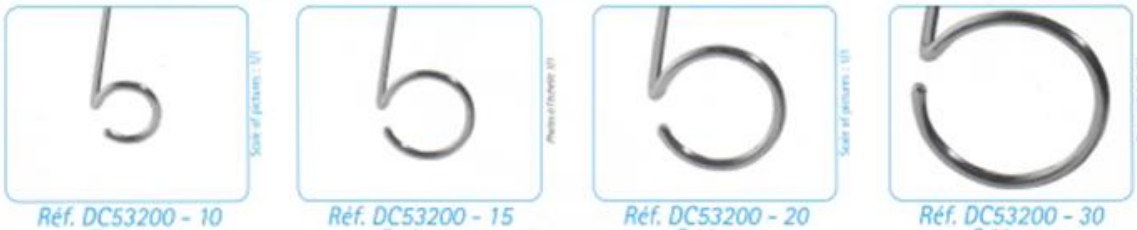
> 3 diamètres pour s'adapter à la taille du vaisseau
> Anneau discrètement ouvert pour permettre le dégagement du fil de suture
> Poignée orientée pour un contrôle parfait de la position de l'anneau
> La tige d'Hemostator est en acier flexible, permettant d'obtenir par simple torsion manuelle toute angulation souhaitée

Technical sheet:

> Stainless steel, medical plastic (POMC) sterilizable

Specifications techniques :

> Acier inoxydable, plastique médical (POMC) stérilisable



Réf. DC53200 - 10
Ø 10 mm

Réf. DC53200 - 15
Ø 15 mm

Réf. DC53200 - 20
Ø 20 mm

Réf. DC53200 - 30
Ø 30 mm

Brand of LANDANGER Group
9 Boulevard du Maréchal de Lattre de Tassigny / B.P. 2152 / 52906 CHAUMONT
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E-mail : clientele@delacroix-chevalier.fr / www.delacroix-chevalier.fr

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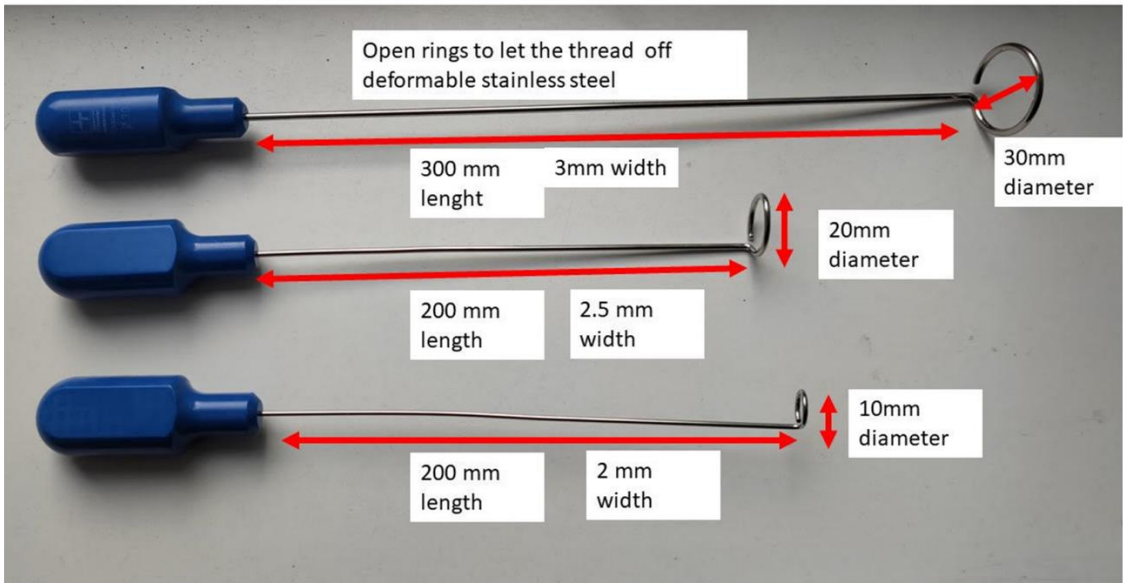
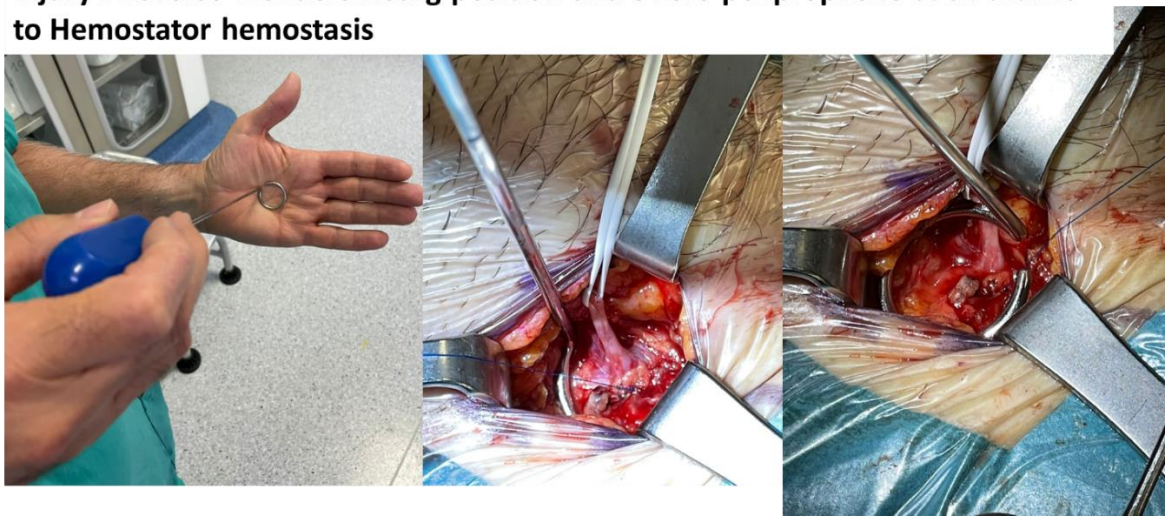


About hemostator:
Redoo surgery for groin recurrence with systolic valsalva refux through the terminal valve. Previous anterior saphena disconnection. Bleeding from stump injury . Reverse Trendelemburg position and 6 zero polipropilene stich thanks to Hemostator hemostasis

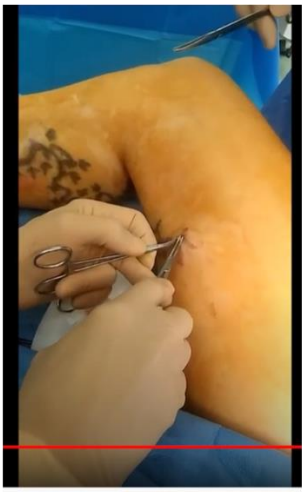


83322- ***Sutures and Ligations threads are <u>NON-absorbable</u> to avoid recanalisation and by-pass by inflammatory angiogenesis due to absorbable* threads**

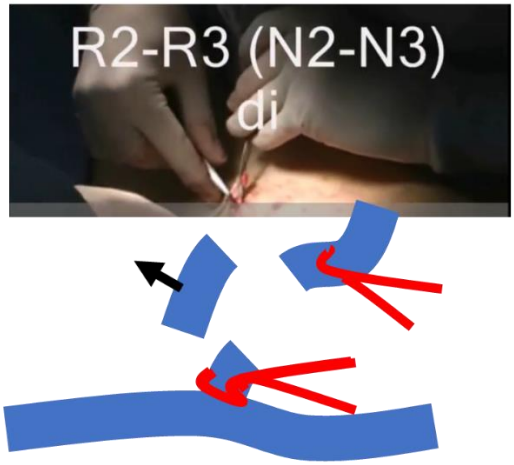
83323-Do not leave stumps behind.

N2>N3 junctions: Section of N3 ligation flush with N2 and resection of 2 to 3 cm to avoid leaving stumps which are sometimes a source of recurrence by bypassing.

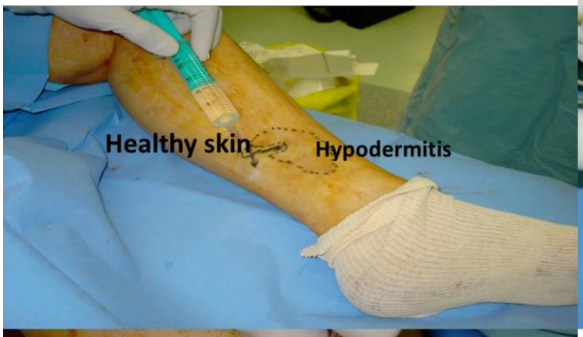
Non-aggressive lace hooks to avoid breaking the vein

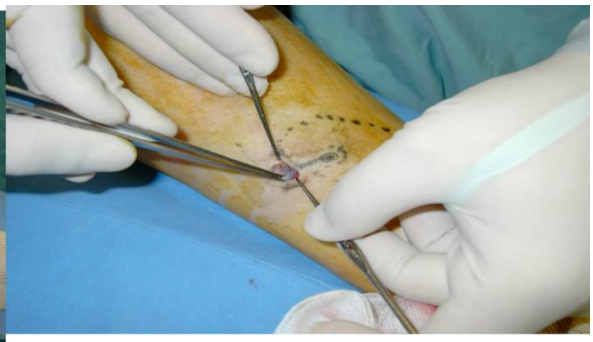


Incisions scalpel N°11



Section-ligature non absorbable flush the saphenous trunk + 2 cm excision





Incision in healthy skin then traction of the wound in order to ligate the tributary flush the saphenous trunk

83324- ***<u>Close the fascia with non-absorbable thread</u>,*** *especially after disconnection of the* ***Small saphenous veins, but also all the fascia crossed by disconnected reflux.***

8333- Specific procedures according to escape points and types of shunts

83331- Saphenofemoral junction.

Supine position with slight abduction-external rotation of the thigh. Local anaesthesia. Incision according to the marking.

The saphenous arch is pulled by loops around its tributaries, which facilitates its cleaning up to the saphenofemoral junction.

It is sometimes atypical: double or bypassing the femoral artery from behind.

2 methods of disconnection, crossotomy and triple ligation have shown the same long term no recurrence:

Crossotomy consists of great saphenous division close to the saphéno-femoral junction, non-absorbable ligation, clip flush the femoral vein, NO ligation nor division of the tributaries. So, it is not Crossectomy which consists of resection of the arch and ligation of the tributaries. According to Massimo Cappelli, Crossectomy provides more recurrence than crossotomy ***Ref:*** CAPPELLI M. et Al.: Ligation of the saphenofemoral junction tributaries as risk factor for groin recurrence. J Vasc Surg Venous Lymphat Disord. 2018 Mar;6(2):224-229. doi: 10.1016/j.jvsv.2017.09.005. Epub 2017 Dec 28.

Triple Sapheno-femoral Ligation (TSFL) which reduces the risk of bleeding, which is appreciable in ambulatory surgery. Three successive ligations are tightened between the sapheno-femoral junction and the first tributary of the great saphena. The wire of this triple ligation must be of very thick: N° 2 (and not 00!), to avoid incorporation of the wire in the wall with slow sectioning and recanalisation. Clip flush the femoral vein. **Ref:** R Delfrate, M Bricchi,C Franceschi, M Goldoni.Multiple ligation of th eproximal great saphenous vein in the CHIVA treatmentof primary varicose veins.Veins and Lymphatics 2014; volume 3:1919. **VIDEO** TSFL+ Crossotomy : https://www.youtube.com/watch?v=2CleOqLUbs4&t=11s

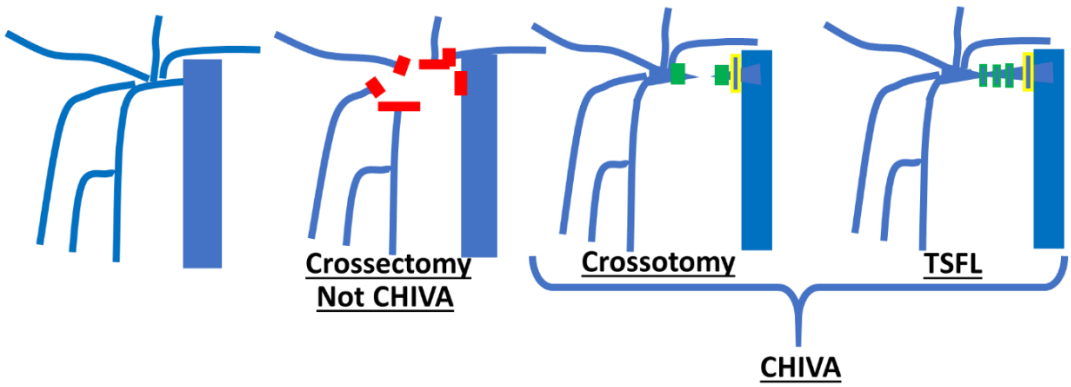
Note that a clip is placed flush with the femoral vein to avoid leaving a stump (possible source of recanalisation).

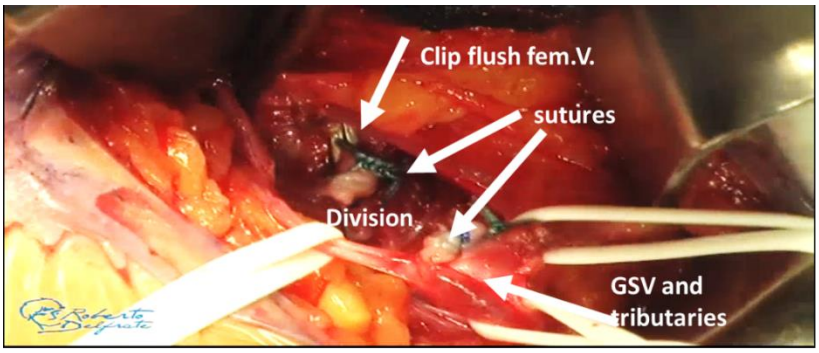
Sapheno-Femoral Junction

Crossectomy: Arch resection +Tributaries ligation **CHIVA: disconnection**

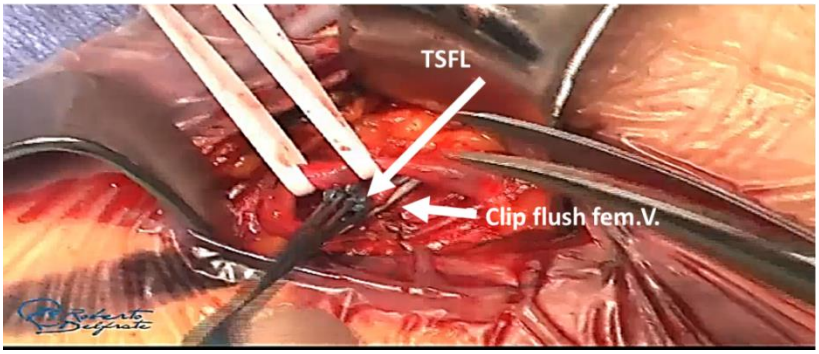
Crossotomy :No tributaries ligation nor Arch resection. Division, non absorabale suture and clip flush the Femoral vein (no stump)

Triple Sapheno-Femoral Ligation: Triple Thick N°2 non absorbable ligation + clip





Crossotomy :No tributaries ligation nor Arch resection. Division, non absorbable suture and clip flush the Femoral vein (no stump)



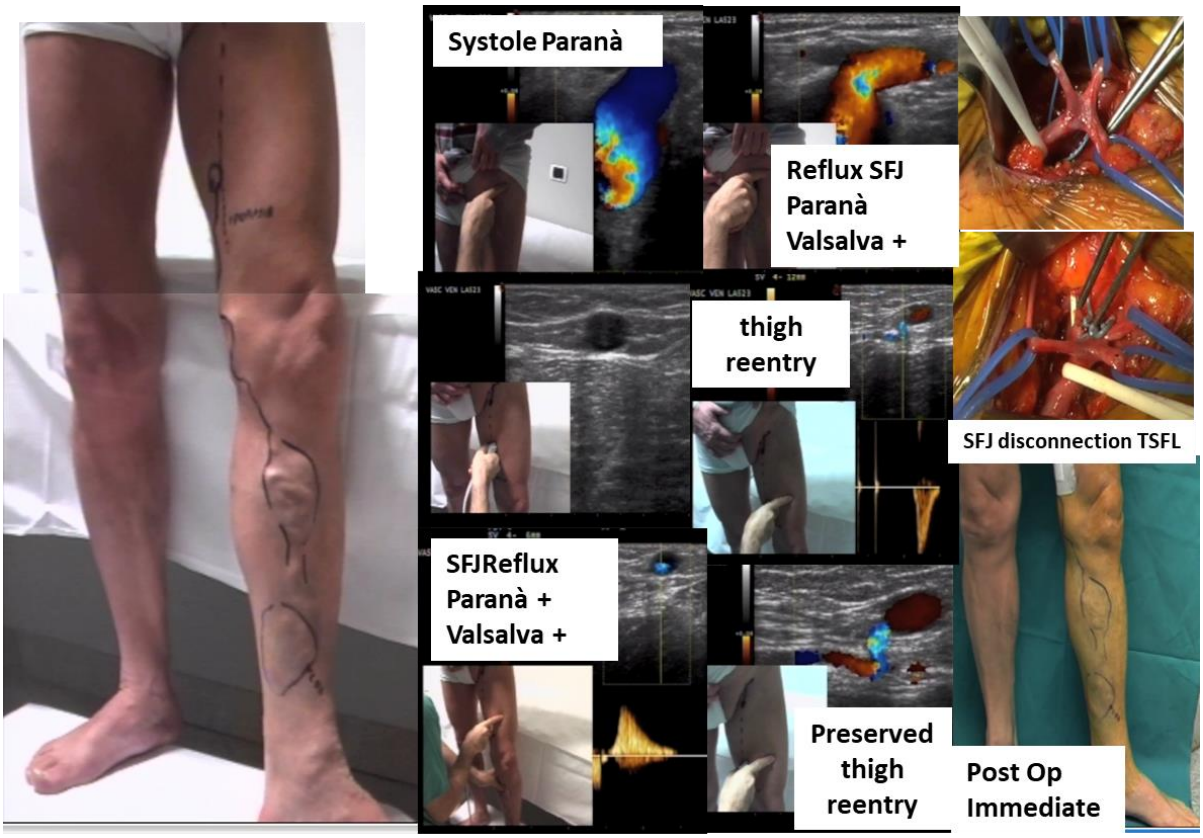
Triple Sapheno-Femoral Ligation TSFL: Triple Thick N°2 non absorbable ligation + clip

The junction is sometimes atypical: *it may be double, or it may by-pass the femoral artery from behind, which makes dissection more difficul****t.***

Cavernomas due to recurrence *after crossectomy and stripping are more difficult and invasive in surgical approach. The****y respond well to injection of sclerosing foam.***

In case of SHUNT III*. Great care must be taken not to leave any N2>N3 stump during the first stage of CHIVA2 of III shunts to* ***reduce as much as possible the risk of thrombus with pulmonary embolism and recurrence N2>N3.***

The endovenous procedure of the first centimetres below the junction, called ***CHIVA Laser, is not CHIVA because it doesn't disconnect the closed shunts flush the saphéno-femoral junction, but below the arch tributaries and reduces the length of saphenous vein eligible for by-pass surgery. Notice that the same result can be achieved by triple ligation, few centimetres below the saphenofemoral junction, without any risk for greater efficiency and lower cost.***



83332- ***Saphenopopliteal junction***

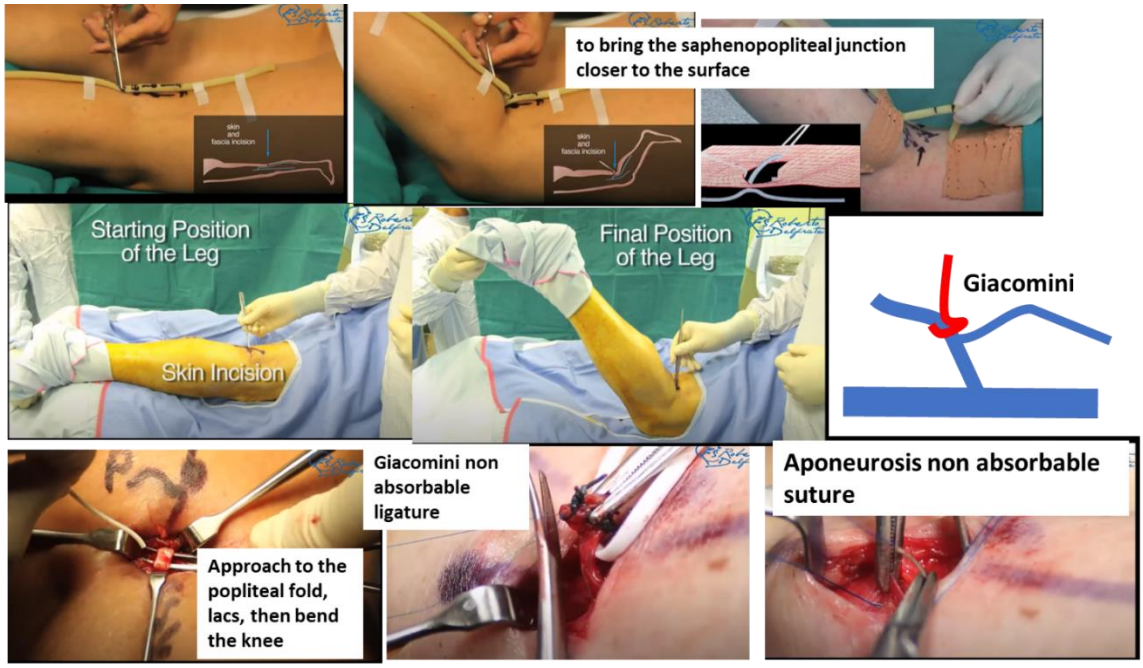
833321- The location of the sciatic nerve has been previously identified by ultrasound.

833322- Disconnection of the small saphenous vein

Section-ligation flush with the popliteal vein is unnecessary *in most cases. VIDEO:* SSV specific technique: https://www.youtube.com/watch?v=coek5QYpXec&t=137s

When the trunk is refluxing, disconnection is most often possible flush below the junction of the small Saphenous vein with the Giacomini, *which will "wash" the saphenofemoral junction.*

It is totally inadvisable in open vicarious shunts OVS and mixed shunts MS that compensate a femoral block, *due to the high chance of recurrence by* ***cavernoma****.*



The bent-knee position allows the saphenopopliteal junction to be brought closer to the surface and the high and deep junctions to be better approached by incising lower, at the level of the popliteal fold, which avoids crossing the muscles

833323- ***In the absence of Giacomini, disconnection, when necessary, is performed flush with the popliteal vein, by section ligation or triple ligation as for the great saphenous vein (see above TSFL).***

833324- The position of the saphenopopliteal junction is often high and deep.

The operative position, which I have called the "technique du petrolier", consists of bending the patient's knee to 45-60° in procubitus. The incision in the popliteal fold moves up "by itself" towards the thigh. Moreover, it facilitates surperficial traction with loops hooking the small saphenous vein, because the popliteal vein is relaxed by this position.

833325- The aponeurosis is always closed with non-absorbable suture.

833326- Popliteal cavernomas, either post-crossectomy, or by OVS or MS, are left in place because of the risks of bleeding, but above all because of the inefficiency of surgical resections. It is preferable to disconnect the supra-fascial varicose tributaries flush with the fascia, which is then sutured with non-absorbable thread.

83333- The popliteal perforator is approached in the same way as the small saphenous vein (petroleum technique) and is treated by resection or triple ligation, then the fascia is sutured with non-absorbable thread.

83334-- Incompetent Saphenous femoral perforators of the thigh

*The Saphenous Femoral perforator is **resected** over the greatest possible length **without cutting the trunk of the saphenous vein. As it is sometimes double the collateral is sought and resected in the same way.***

83335--Pelvic escape points.

As I have already mentioned, pelvic escape points are disconnected without prior embolization of deep pelvic reflux when there are no clinical signs of pelvic congestion. ***Ref:*** Delfrate R, Bricchi M, Franceschi C. Minimally-invasive procedure for pelvic escape points in women. Veins and Lymphatics. 2019; 8:7789, 10-16. VIDEO : Inguinal Pelvic Escape point: I Point: https://www.youtube.com/watch?v=z3tSXAfMqnc&t=116s Perineal Pelvic Escape/ P Point: https://www.youtube.com/watch?v=ThLN8ApPTOc&t=87s

833351--Perineal escape point. P-Point.

VIDEO: Perineal Pelvic Escape/ P Point: https://www.youtube.com/watch?v=ThLN8ApPTOc&t=87s

Disconnection of the P-point is sufficient to collapse varicose veins of the greater when they depend solely on the P-point.

Patient in gynecological position.

Local anesthesia.

Short incision** approach at the level of the marking in the **¼ posterior vulvo-perineal fold, sometimes more anterior.

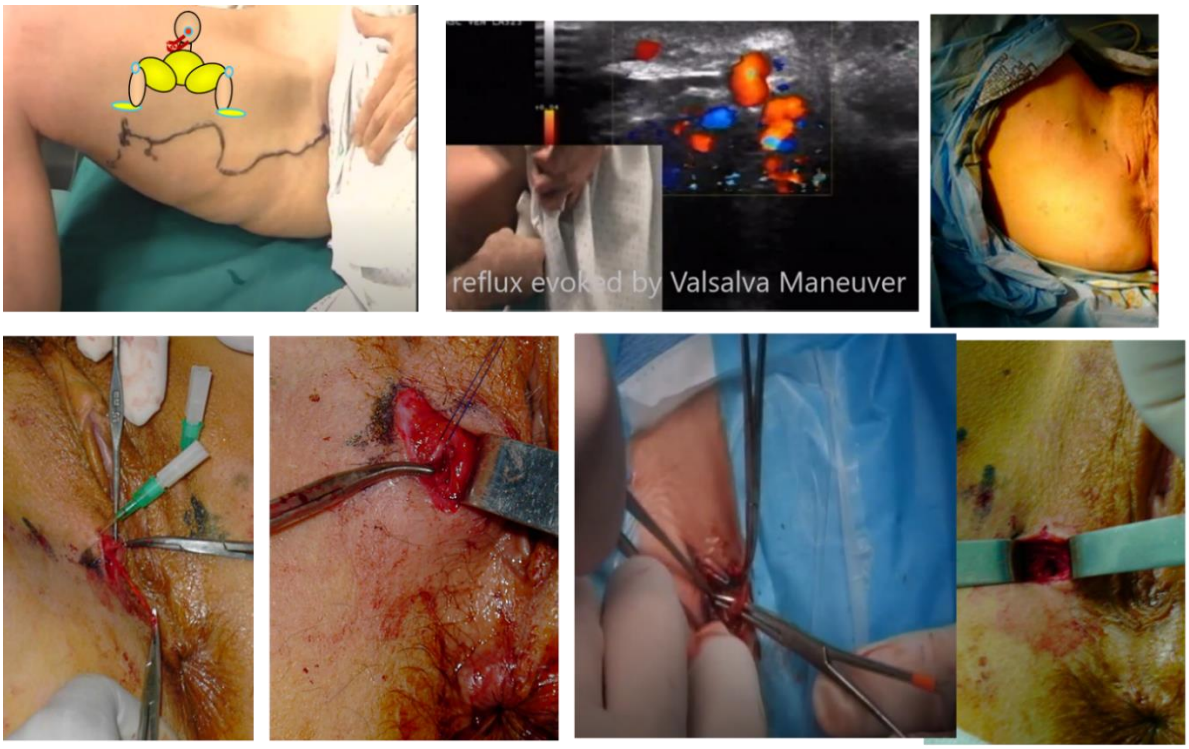
*The perineal vein **appears thin in this position.** It is often **very fragile.***

It is resected and ligated with non-absorbable thread.

Local antibiotics

Non-absorbable closure of the fascia

***Ultrasound-guided sclerosis may be offered instead of surgery**, bearing in mind that it may require several sessions and recanalisation may occur later.*



Perineal point: approach in gynecological position. Local anesthesia. Suture resection of the perineal vein and fascia closure with non-resorbable thread. Local antibiotic..

833352*-. *Inguinal escape point: I point

VIDEO : Inguinal Pelvic Escape point: I Point: https://www.youtube.com/watch?v=z3tSXAfMqnc&t=116s

Patient in supine position.

Local anesthesia

Approach at the marked point, in front of the superficial ring of the inguinal canal.

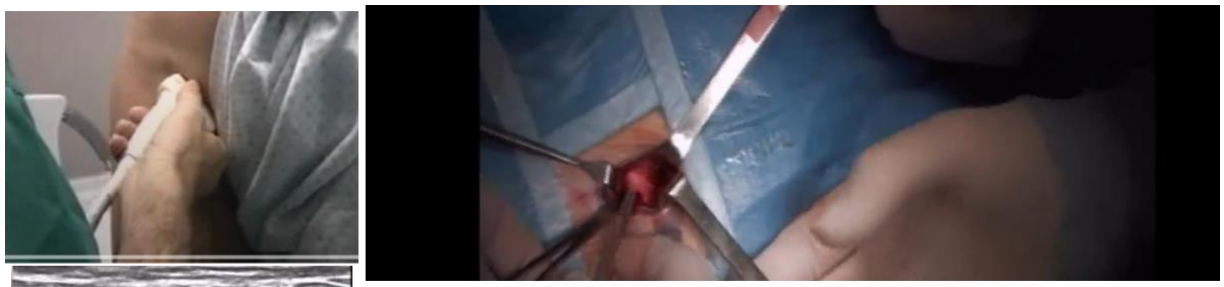
Dissection of the vein of the round ligament of the uterus, respecting the round ligament and the genitocrural nerve.

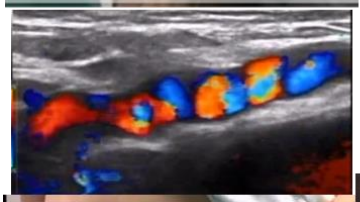
First ligation to avoid ***accidental intra-pelvic bleeding.***

then short resection of the vein.

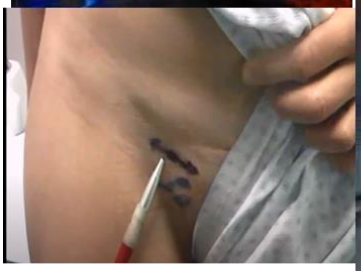
Closure of the canal while minding not to tight the genitocrural nerve.

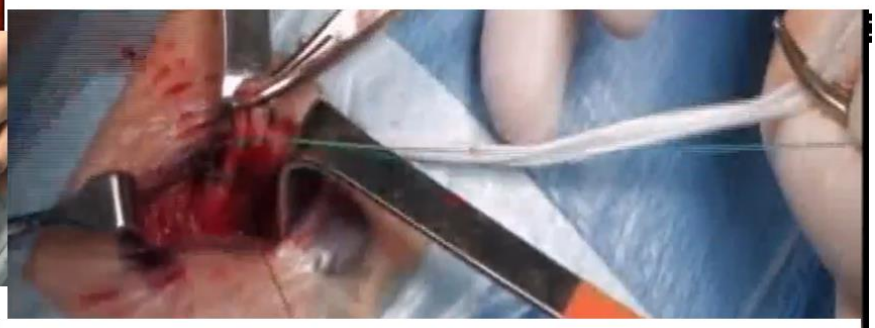
Ultrasound-guided sclerosis may be offered instead of surgery, *bearing in mind that it may require several sessions and recanalisation may occur later.*





Opening of the fascia. Dissection of the round ligament vein, avoiding trauma to the genito-crural nerve





First non-absorbable ligation of the round ligament vein before sectioning to avoid pelvic hemorrhage!

833353- *Obturator escape point: O Point*

The approach to the saphenous-vein obturator connection is very close to the saphenofemoral junction. It can be approached as for a disconnection of the saphenofemoral junction or selectively under echodoppler control.

833354-*CHIVA of SHUNT III*

CHIVA 2: 2 steps or CHIVA 1: 1 step

Simultaneous disconnection of the saphenofemoral N1>N2 and spahenotributary N2>N3 escape points in type III shunts is hemodynamically incorrect.

Due to the absence of an intermediate draining re-entry N2>N1 between N1>N2 and N2>R, this procedure blocks the drainage of the saphenous vein, which favours its thrombosis and varicose recurrence.

I remind you the test to assert the absence of interposed truncal reentry which consists of manual compression-release of the calf or, better still, the Paranà manoeuvre + the Valsalva manoeuvre, while the refluxing tributary is effectively compressed with the finger (see details chapter 7).

3 solutions are offered.

8333541-1. Disconnection only of the saphenofemoral escape N1>N2. ***It ablates the N3 reflux overload from N1 but leaves behind the overload from N2 (type II shunt).*** *May be sufficient to improve skin trophicity in patients not disturbed by the aesthetics of their varicose veins*

8333542-2. CHIVA 2, i.e., CHIVA in 2 steps:

First step: N3>N2 disconnection.

In the absence of intermediate re-entry, *the blood of the great saphenous vein no longer flows back and the* ***antegrade flow is restored. Though the closed type III shunt (N1>N2>N3>N1) has been deactivated by closing the N2>N3 escape****, the pressure column has not been fractioned at the level of the saphenofemoral junction.*

Most of the time, in the following months ***a perforator that was inactive during tests to visualize intermediate re-entries, opens****, which reactivates the saphéno-femoral reflux and* ***creates a N1>N2>N1 closed shunt.***

Second step. N1>N2 disconnection.

When a N1>N2 reflux reappears, a shunt I has been created *which can be disconnected at the saphenofemoral junction.* ***This is the second step of CHIVA 2.***

<u>The first step can be dangerous</u> because it leaves the way free via N2>N1 for a possible postoperative thrombus of the saphenous trunk. This risk increases with the calibre of the Great Saphenous vein and the proximity of N2>N3 to the saphenofemoral junction.

<u>This risk can be eliminated, and the double operation can be replaced by CHIVA in 1 step: Disconnection + Devalvulation</u>.

8333543-. CHIVA of SHUNT III in 1 step.

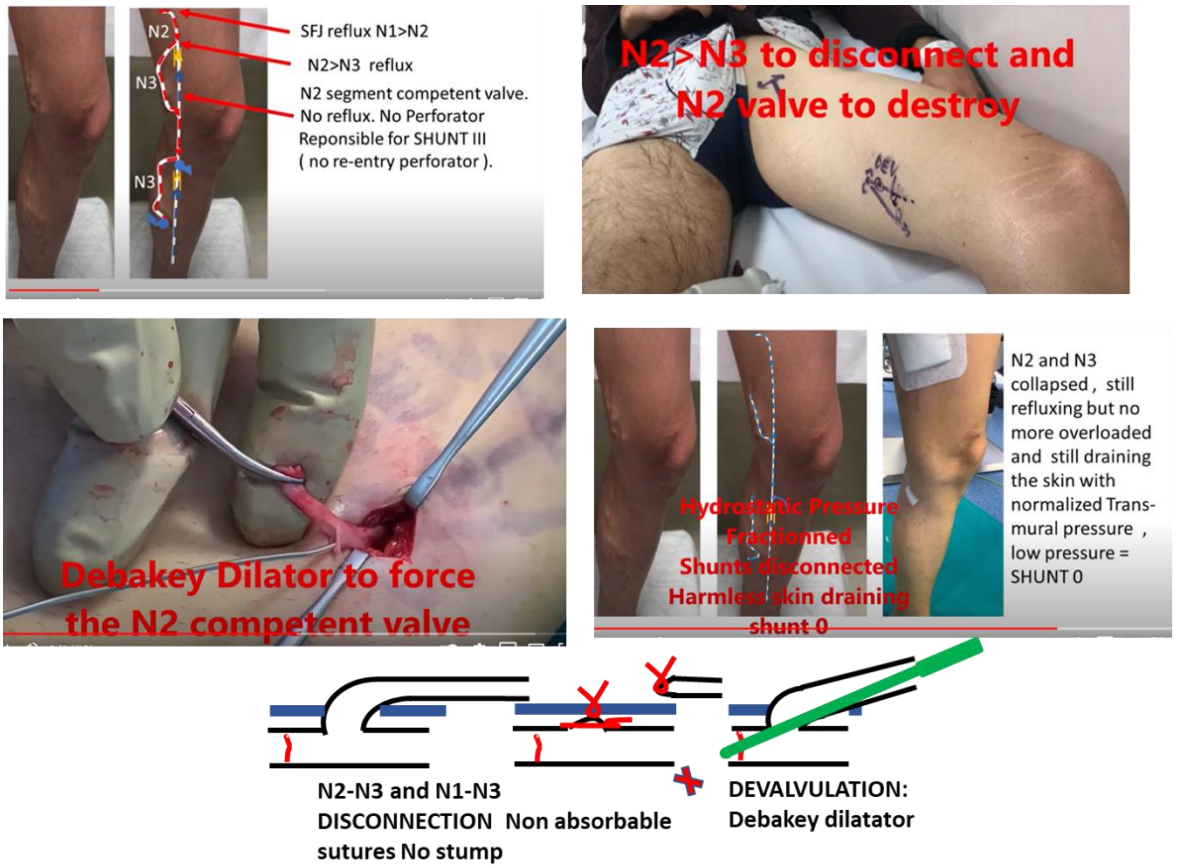
VIDEO: Shunt III devalvulation: https://youtu.be/T01AXcdomkM

Disconnection + Devaluation.

1-Disconnection of the saphenofemoral junction as described above

2-Approach of the N2>N3 junction.

Passing a tool as Debakey Dilatator N°2 through the N3 stump to force the saphenous competent valve previously located by echodoppler so that the breach makes the reflux reach and enter below the re-entry perforator great saphenous trunk.



83336-*Deep escape points.*

Under local or loco-regional anaesthesia.

Here, <u>intraoperative ultrasound is particularly useful</u>. It facilitates recognition of the <u>cleavage planes</u> and <u>bloodless</u> passage between the muscles and flush with the bones, to reach the veins to be disconnected.

The <u>disconnection takes 2-3 cm of incompetent veins</u> below the escape point between 2 non-absorbable ligations.

<u>Effective dose anticoagulation for 2 months postoperatively</u>

VIDEO deep CHIVA https://www.youtube.com/watch?v=t6vGDEwx9XI&t=230s

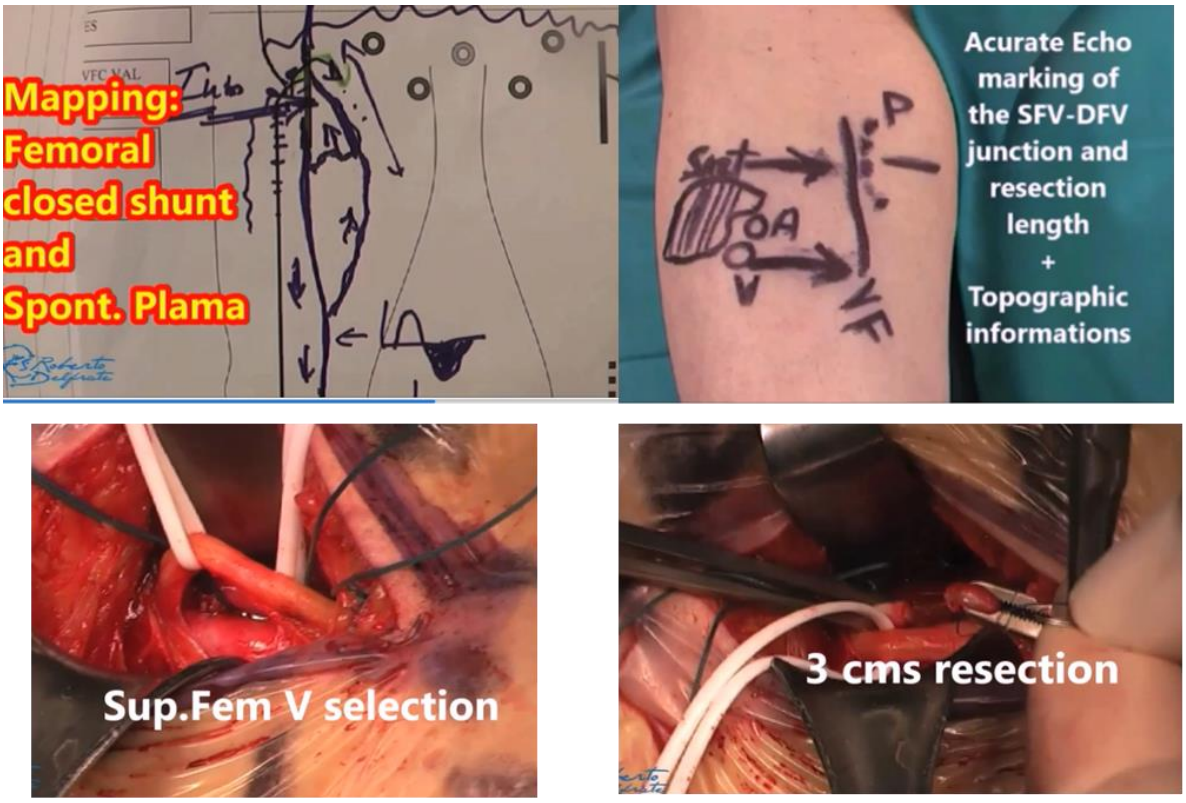
833361- Deep closed femoral shunt:

8333611- Closed SHUNT superficial femoral-deep femoral

Disconnection, resection of 2 to 4 cm and non-absorbable ligation of the incompetent superficial femoral vein at its junction with the deep femoral vein,

but only <u>when it has been ascertained by echodoppler that the latter communicates with the popliteal vein</u>. The echodoppler must show a deep femoral flow activated by manual compression of the calf in the standing position. Manual compression of the calf is more selective here than the Parana manoeuvre, as the latter also mobilizes the thigh muscle pump, which does not allow assessment of the proportion of calf blood via the deep popliteal-femoral connection. ***VIDEO*** deep CHIVA Femoral closed shunt
https://www.youtube.com/watch?v=t6vGDEwx9XI&t=230s

Postoperative anticoagulants in effective doses for 2 months.



Deep CHIVA, Superficial femoral closed shunt fed by the deep femoral vein which also drains the calf via a good connection with the popliteal vein.

8333612*-Duplicated Superficial Femoral- SHUNTS.*

Disconnection resection 2 to 4 cm and non-resorbable ligation of the incompetent vein of the incompetent branch of a double superficial femoral vein.

Postoperative anticoagulants at effective dose for 2 months.

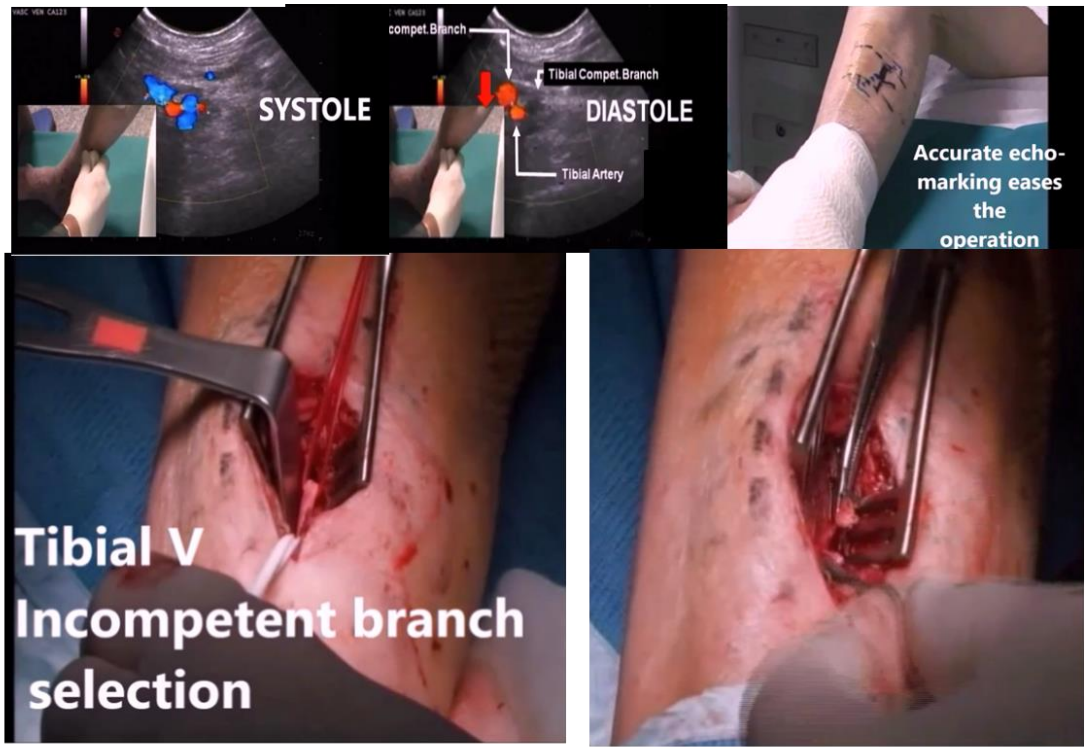
833362- Closed SHUNT of the tibial veins.

VIDEO deep CHIVA Tibial closed shunt https://www.youtube.com/watch?v=t6vGDEwx9XI&t=230s

Marked and approached in little healthy, above hypodermitis and/or ulcer.

Disconnection resection 2 to 4 cm and non-absorbable ligation of the incompetent trunk or both when both are incompetent.

Postoperative anticoagulants in effective doses for 2 months.



Déconnexion par résection 2 cm d'un shunt profond par incontinence de l'une des 2 veines tibiales postérieures. Ulcère et hypodermite. Marquage et abord en peau saine.

84- Results of the CHIVA Cure

CHIVA recurrence is different from post destructive methods. They are made of

recanalization of previous disconnections

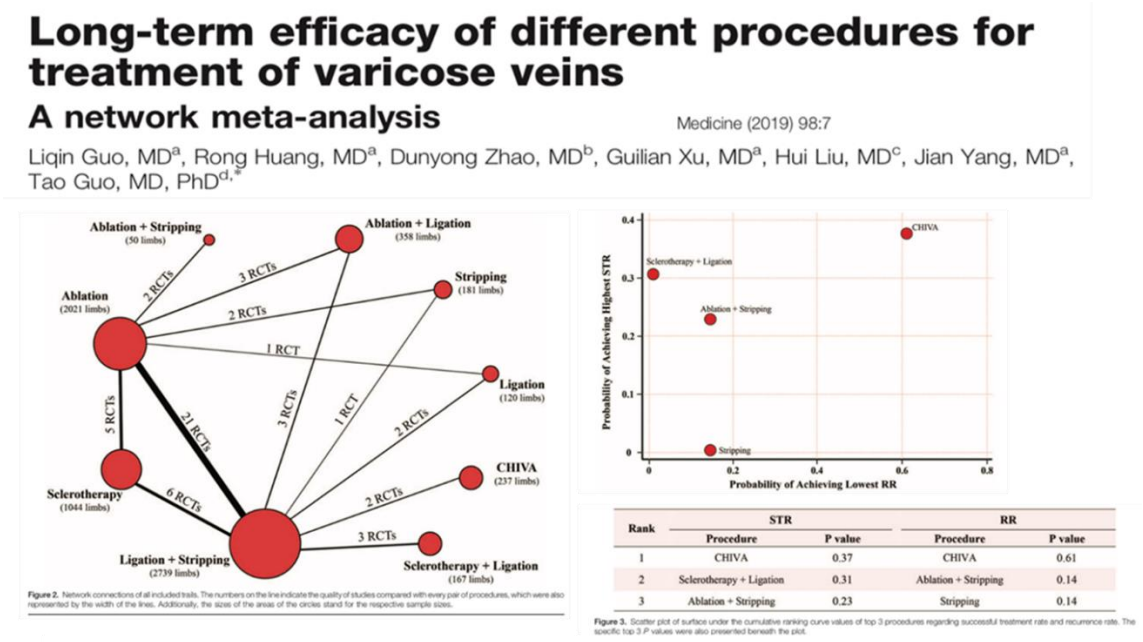
and/or easy to treat left behind or new escape points (Closed shunts CS et open deviated shunts ODS,

. Whereas , most recurrences post destructions are made of many superficial Open vicarious shunts forces by the residual pressure upstream the destroyed N3 and or N3 veins. This aspect was particularly analysed by Carandina **Ref:** CARANDINA S., MARI C., DE PALMA M., MARCELLINO M.G., CISNO C., LEGNARO A., LIBONI A., ZAMBONI P., Varicose vein stripping vs hemodynamic correction (CHIVA): a long-term randomized trial, Eur. J. Vasc. Endovasc. Surg., 2008 Feb, 35(2): pp. 230–7. ***These OVS recurrences are doomed to recur each time they are ablated.***

This first RCT shows a post CHIVA recurrence rate 50% less than other methods and moreover saves the venous capital for by-pass.

The Last review:

Ref: Guo L. et Al.: Long-term efficacy of different procedures for treatment of varicose veins A network meta-analysis. Medicine (2019) 98:7



Last review. CHIVA rated best treatment for the superficial venous insufficiency

2 Cochrane reviews:

- ***CHIVA vs stripping:*** *ref* ***Bellmunt-Montoya S, Escribano JM, Dilme J, Martinez-Zapata MJ.Cochrane Database CHIVA method for the treatment of chronic venous insufficiency Syst Rev. 2013 Jul 3;(7):CD009648. . Conclusion: CHIVA better than Stripping***

CHIVA vs Stripping and endovenous ablation: *ref* ***Bellmunt-Montoya S, Escribano JM, Dilme J, Martinez-Zapata MJ.Cochrane Database CHIVA method for the treatment of chronic venous insufficiency Syst Rev . 2021 Sep 30;9(9):CD009648. Conclusion: CHIVA equal or slightly superior to other methods***

Unfortunately, no study, review or guideline emphasizes or incorporates GSV salvage as a potential life-saving arterial shunt, provided in all cases by CHIVA alone. Ref Paolo Zamboni, Claude franceschi***" Regarding the review and commentary of CHIVA Method for the Treatment of Chronic Venous Insufficiency," Manuscript Number JVSVL-D-22-00224. In press.***

- ***CHIVA must be performed by "experts" i.e. the ones who apply strictly the diagnosis, strategy and tactics rules. Otherwise, most disappointments will result.*** ***Ref****:* Milone M, Salvatore G, Maietta P, Sosa Fernandez LM, Milone Recurrent varicose veins of the lower extremities after surgery. Role of surgical technique (stripping vs. CHIVA) and surgeon's experience. G Chir. 2011 Nov-Dec;32(11-12):460-3. ***This should be obvious since vascular specialists are expected by professional ethics to be experts in the best available treatments.***

The results of the CHIVA cure have been the subject of more than 120 publications (studies, RCTs and Cochrane reviews) cited with their abstract at the end of this chapter.

I report here the conclusion of Paolo Zamboni on this bibliographic work that he conducted with Massimo Cappelli (see chapter 9).

The following elements rise up from the analysis of the articles:

The results of different CHIVA studies about clinical data, recurrence rates and quality of life, comparing them with other methods of treatment without randomization, are superimposable on each other. So, they are not sporadic cases, referring to individual studies.

All randomized studies as well as the two Cochrane reviews and the meta-analysis demonstrate the superiority of CHIVA compared to other treatments in terms of recurrence and quality of life at 5 and 10 years.

The biochemical analysis of the pre- and post-chiva inflammatory markers, together with the demonstration of a regression of the saphenous wall alterations after

treatment, extremely confirm the possible use of a post-CHIVA saphenous trunk for arterial by-pass. Anyway, the incompetent great saphenous vein has been always used for by-pass, especially in case of infra-inguinal arteriopathies.

The low spread of CHIVA treatment and the learning curve cannot be criteria influencing the levels of evidence. Indeed, they must be a stimulus to optimize the own work.

Therefore, CHIVA represents the treatment of superficial venous insufficiency which gives the best results over time compared to all the other methods applied: stripping and endovascular procedures; with the big advantage of being able to preserve a saphenous trunk for a possible use as an arterial by-pass. About this aspect, I would like to underline, two concepts:

The age of population increases, therefore the probability of finding phlebopathic patients with arteriopathies increases patients, awareness of venous problems, approach earlier their own phlebological problems so the probability of finding saphenous veins not involved or less alterated is high.

Pr Paolo Zamboni 2020 »

ARTICLES COMPARING CHIVA RECURRENCES / CLINICAL DATA WITH OTHER PROCEDURES EMPLOING RANDOMIZED STUDIES (RCT)

1) ZAMBONI P., CISNO C., MARCHETTI F., MAZZA P., FOGATO L., CARANDINA S., DE PALMA M., LIBONI A., Minimally invasive surgical management of primary venous ulcers vs. compression treatment: a randomized clinical trial, Eur. J. Vasc. Endovasc. Surg., 2003 Apr, 25(4): pp. 313–8.

This prospective randomized study compared CHIVA strategy associated with compression with the use of compression alone in the treatment of venous ulcers associated with chronic superficial venous insufficiency of the lower extremities (C6 in the CEAP classification). 24 patients were treated with compression, advanced wound dressings (and treatment antibiotic if necessary) the dressings were changed every 3 to 5 days during the first month and every 7 days thereafter.

The CHIVA group included 21 patients, 16 extremities had a hemodynamic presentation similar to type I shunts and were treated with crossectomy and further tributary ligatures, 7 extremities had a type III shunt and were treated with type CHIVA 2 procedure.

The study assessed:

- the healing process expressed in 2 mm per day;

- the functionality of the venous system based on air plethysmography data before treatment, 6 months and 3 years after treatment;

- quality of life through SF-36 questionnaire before treatment

and 6 months after treatment. In addition to the clinical evaluation, an eco-Doppler examination was performed

every 6 months for a total of 3 years. The results are presented in table 10.7. (Comment by Paolo Zamboni)

2) CARANDINA S., MARI C., DE PALMA M., MARCELLINO M.G., CISNO C., LEGNARO A., LIBONI A., ZAMBONI P., Varicose vein stripping vs haemodynamic correction (CHIVA): a long term randomised trial, Eur. J. Vasc. Endovasc. Surg., 2008 Feb, 35(2): pp. 230–7.

This randomized comparative study aimed to compare the long-term results of stripping with respect to CHIVA in the treatment of chronic superficial venous insufficiency.

180 consecutive patients underwent clinical evaluation, including CEAP classification, and duplex examination performed by expert operators. 30 patients were excluded second because they did not meet the study inclusion criteria, while 150 patients were randomized to

two groups, 75 were treated with stripping and 75 with CHIVA. All operated extremities were examined by three independent assessors who had not been involved in previous surgical procedures. The results were evaluated according to the Hobbs criteria and are presented in tables 10.8 and 10.9.

The relative risk of recurrence in the Stripping group doubled to 10 years compared to the CHIVA group (OR 2.2; 95% CI 1–5, p <0.04).

No significant difference was found between the two 3-year techniques. During the 3 to 10-year period the different recurrence rates in the two groups become evident and significant, so it is concluded that at 10 years the risk of recurrence is double in the ablative group (Figure 10.1). (Comment by Paolo Zamboni)

J Mal Vasc. 2009 Feb; 34 (1): 65. doi: 10.1016 / j.jmv.2008.10.002. Epub 2008 Dec 4.

[Correspondence: letter by P. Zamboni about the analysis of the article "Varicose vein stripping versus haemodynamic correction (CHIVA): a long-term randomized trial"].

[Article in French]

Zamboni P.

3) IBORRA–ORTEGA E., BARJAU–URREA E., VILA–COLL R., BALLÓN–CARAZASH., CAIROLS–CASTELLOTE M.A., Estudio comparativo de dos técnicas quirúrgicas en el tratamientode las varices de las extremidades inferiores: resultados tras cinco años de seguimiento,

ANGIOLOGÍA, 2006, 58(6): pp. 459–468.

Iborra and his team published a prospective randomized study in Spanish in 2006 that included 100 legs treated with CHIVA or Stripping with a 9-year follow-up. 62 women and 38 men with an average age of 49 years were selected following the Spanish guidelines for the treatment of varicose veins. The patients included did not have a history of venous surgery, thrombosis, were not overweight or older than 70 years. 49 patients were randomized to the Stripping group and 51 to the CHIVA group. There were no differences in age, gender, weight and CEAP

between the 2 groups. All patients underwent Doppler examination and after the surgery, they received the same dose of prophylactic heparin. The follow-up with questionnaires and ultrasonography was performed 1 week after the intervention and then after 1, 3, 6 months and every year for 5 years. All patients in the stripping group were hospitalized (44 spinal and 5 under general anesthesia)

While of the CHIVA group 9 patients remained in hospital for one night, the rest were treated on an outpatient basis (6 spinal, 3 general, 42 local anesthesia), table 10.10.

The average working disability in the stripping group was 19 days while in the CHIVA group 8 days ($p < 0.001$). Neither group experienced serious complications, 11 patients in the stripping group reported ankle paresthesia, while in the CHIVA group 4 patients reported symptomatic superficial venous thrombosis (table 10.11).

Despite the best recovery after CHIVA, the 5-year results for the

outcomes considered were not significantly different (table 10.12).

(Comment by Paolo Zamboni)

4) PARÉS J.O., JUAN J., TELLEZ R., MATA A., MORENO C., QUER F.X., SUAREZ D., CODONY I., ROCA J., Varicose vein surgery: stripping versus the CHIVA method: a randomized controlled trial,

Ann. Surg., 2010 Apr, 251(4): pp. 624–31.

The aim of this study was to compare the effectiveness of the CHIVA method for the treatment of varicose veins compared to the standard stripping treatment. The study design was randomized and controlled monocentric, and 501 patients with primary varices were included. Patients were randomly assigned to the CHIVA procedure (experimental group n = 167) or stripping without duplex mapping (control group 1, n = 167) or stripping with duplex mapping (control group 2, n = 167). The outcome measure was 5-year clinical recurrence, examined by independent evaluators previously trained in the procedures. Duplex ultrasonography has also been used to evaluate the causes of relapses. The results are summarized in table 10.13.

The odds ratio for the presence of 5-year relapses between the stripping group with clinical marking and the CHIVA group was 2.64, (95% confidence interval [CI]: 1.76–3.97, $P < 0.001$). The odds ratio for relapses after 5 years of follow-up, between stripping with duplex mapping and CHIVA group, was 2.01 (95% CI: 1.34-3.00, $P < 0.001$).

The conclusion was that the CHIVA surgical treatment had

fewer side effects and less recurrence after 5 years compared to both stripping groups. No statistical differences were found between the two stripping groups (with and without duplex mapping). (Comment by Paolo Zamboni)

Reviews COCHRANE e Metanalisis

1) BELLMUNT–MONTOYA S., ESCRIBANO J.M., DILME J., MARTINEZ–ZAPATA M.J., CHIVA method for the treatment of chronic venous insufficiency, Cochrane Database Syst. Rev., 2013 Jul 3, (7): CD009648.

2) BELLMUNT–MONTOYA S., ESCRIBANO J.M., DILME J., MARTINEZ–ZAPATA M.J., CHIVA method for the treatment of chronic venous insufficiency, Cochrane Database Syst. Rev., 2015 Jun 29, (6): CD009648.

The first review was published in 2013 and aimed to compare the effectiveness and safety of the CHIVA method with alternative therapeutic techniques for the treatment of chronic superficial venous insufficiency. Randomized controlled trials (RCTs) have been included to compare the CHIVA method compared to any other treatment. The primary endpoint was clinical recurrence, the studies included in the review had a follow-up of 3 to 10 years, and showed more favorable results for the CHIVA method compared to stripping (721 people, RR 0.63, 95% CI 0.51 to 0.78).

Only one of the studies included in the review reported data related to

quality of life (presented graphically) and these results also significantly favored the CHIVA method.

The stripping group had a higher risk of side effects than the CHIVA group; in particular, for the presence of hematomas (RR 0.63 95% CI from 0.53 to 0.76;) for nerve damage (RR 0.05 95% CI from 0.01 to 0.38).

No statistically significant differences were reported between the groups regarding the incidence of infection and superficial venous thrombosis. (Comment by Paolo Zamboni)

) Guo L. et Al.: Long-term efficacy of different procedures for treatment of varicose veins A network meta-analysis

Medicine (2019) 98:7

Abstract

Background: Various procedures for the treatment of varicose veins have been shown to have long-term effectiveness, but research has yet to identify the most effective procedure. The aim of this study was to investigate the long-term efficacy of different procedures based on Bayesian network meta-analysis and to rank therapeutic options for clinical decision-making.

Methods: Globally recognized databases, namely, MEDLINE, Embase, and Cochrane Central, were searched for randomized controlled trials (RCTs). Quantitative pooled estimation of successful treatment rate (STR) and recurrence rate (RR) was performed to

assess the long-term efficacy of each procedure with more than a 1-year follow-up. The surfaceunder the cumulative ranking (SUCRA) probabilities of the P values regarding STR and RR were calculated to rank various procedures. Grades of Recommendations Assessment, Development and Evaluation (GRADE) criteria were utilized for the recommendation of evidence from pairwise direct comparisons.

Results: A total of 39 RCTs encompassing a total of 6917 extremities were eligible and provided relative raw data. After quantitative

analysis, the CHIVA procedure was determined to have the best long-term efficacy, as it had the highest STR (SUCRA, 0.37).

Additionally, the results revealed that CHIVA possessed the highest probability of achieving the lowest long-term RR (SUCRA, 0.61).

Moreover, the sensitivity analysis with inconsistency approach clarified the reliability of the main results, and the evidence of most

direct comparisons was ranked as high or moderate.

Conclusion: CHIVA seemed to have superior clinical benefits on long-term efficacy for treating varicose veins. However, the

conclusion still needs additional trials for supporting evidence.

Abbreviations: CHIVA = Ambulatory Conservative Hemodynamic Management of Varicose Veins, Development and Evaluation,

GRADE = Grades of Recommendations Assessment, PRISMA = Preferred Reporting Items for Systematic Reviews and Metaanalyses,

RCT = randomized controlled trial, RR = recurrence rate, STR = successful treatment rate, SUCRA = surfaceunder the cumulative ranking**.**

Find the rest of 120 publications and books in the dedicated chapter 9

85- CHIVA cure by sclerotherapy:

Sclerosis is an option when surgery is not technically possible, particularly for varicosities and certain popliteal and groin cavernomas secondary to excision surgery. It must be carried out considering the haemodynamic requirements, and in particular the respect of drainage.

Chpater 9 is dedicated to this. It is written by Massimo Cpapelli.

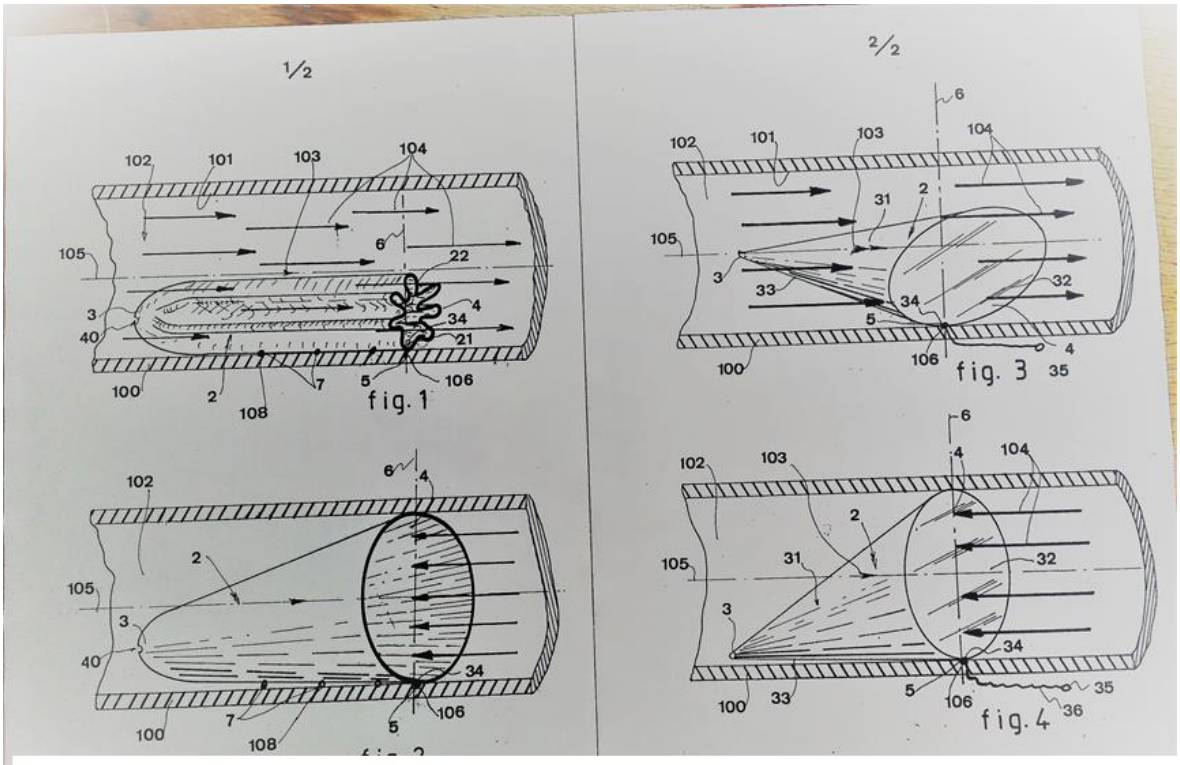
86- Reconstruction methods and valve Prostheses.

I have no personal experience, as the deep CHIVA cure is the alternative that I have developed. I would remind you that prostheses and valve repairs are performed at the level of the common femoral vein, which leaves a significant incompetent column below.

The ideal would be to succeed in making the popliteal vein competent. The deep CHIVA cure fractions the pressure column further down, at the level of the superficial or deep femoral vein or the tibial veins, depending on the hemodynamic configurations of the patients.

In the absence of a deep closed shunt, I have designed and patented a prosthetic valve consisting of a collapsible cone making the role of a single-nest valve easy to introduce with a catheter under ultrasound guidance.

Ref : Une Valve artificielle pour vaisseaux sanguins French Patent Claude Franceschi INPI N° 94 15391.
Today, the patent is free and could help for further realizations.



prosthetic valve consisting of a collapsible cone making the role of a single-nest valve easy to introduce with a catheter under ultrasound guidance. Une Valve artificielle pour vaisseaux sanguins French Patent Claude Franceschi INPI N° 94 15391

87- Deep revascularization

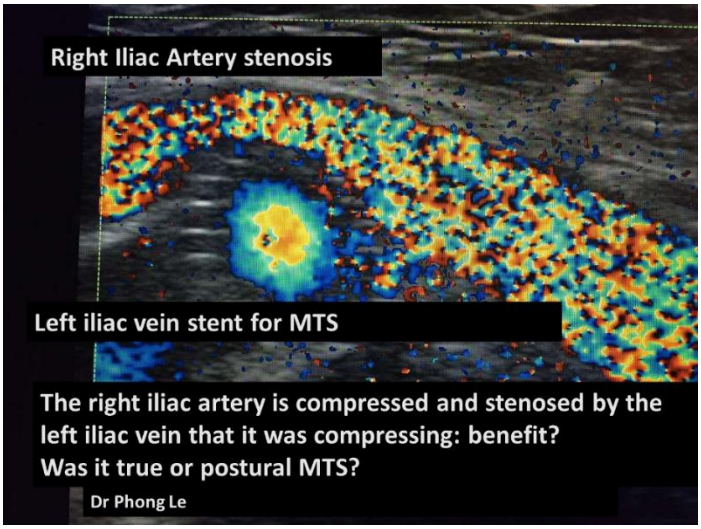
Deep revascularization is nowadays essentially carried out by endo-venous route and stabilized with stents. <u>Its successes are indisputable</u>**, but it must be avoided when it is unnecessary, and therefore with a negative benefit/risk. **This is the case when the** indication is based on **anomalies without hemodynamic confirmation, i.e., the impact of obstructions on the upstream pressure at rest and during exercise.

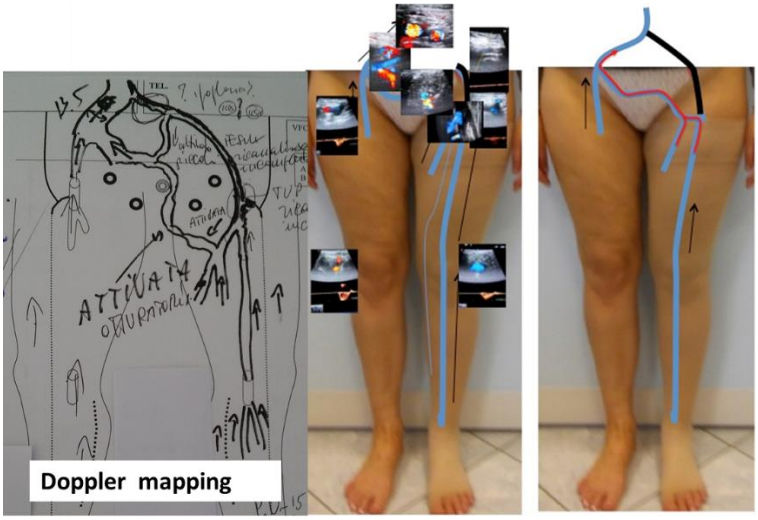
871- Therapeutic excesses

In chapter 7 I showed how postural artefacts can cause false Nutcracker and May Thurner syndromes. Ref: Paolo Zamboni, Claude Franceschi,Roberto Del Frate.The overtreatment of illusory May Thurner syndrome Veins and Lymphatics 2019; volume 8:8020

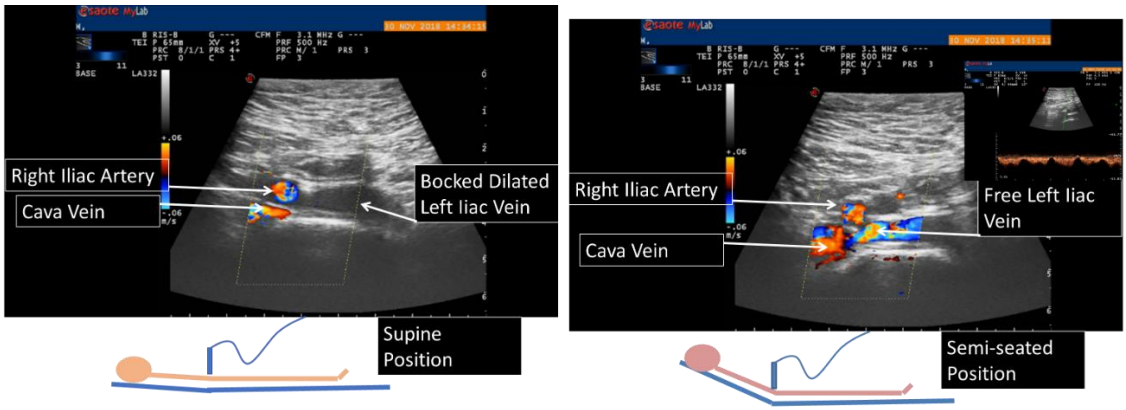
I insisted on the need to distinguish between incompetence and obstruction, particularly in post thrombotic diseases and venous malformations, by means of the <u>Perthes test and the measurement of posterior tibial vein pressures</u> with the Doppler in the decubitus position.

<u>The indication must therefore be based not on anatomical data, but on hemodynamic data.</u>





Compensated Iliac Vein Occlusion: clinical and hemodynamic: Doppler Left Posterior Tibial Vein Pressure = 20 mmHg



May Thurner Syndrome at phlebography but
Pseudo May Thurner Syndrome on Echodoppler: only postural

872- *Stent length and size can also be assessed using Poiseuille's law.*

When collaterals develop, they establish a resistance R2 in parallel with the resistance R1 of the obstacle, which reduces the overall resistance R according to Ohm's law R= (R1+ R 2)/ R 1x R 2 and decreases the pressure P1 accordingly. *The reduction of R1 by recanalisation/stenting over the entire extent L of the stenosis, results in the normalisation of P1. The same result is obtained if the calibre of the collaterals is sufficiently large, which makes recanalisation unnecessary.*

The Poiseuille equation measures the stent size and length sufficient to hemodynamically recanalize an occluded or hemodynamically occluded vein.

The useful flow rate for each patient is difficult to measure directly in the veins. In ***fact, it is sufficient to measure the resting and stress flow in the common iliac artery*** *to assess the flow required by the corresponding iliac vein of each patient. This measurement is easy to perform* ***with the echodoppler.*** *It is then sufficient to* ***integrate this value to measure the dimensions of the corresponding stent,*** *with the least possible loss of load and the smallest possible calibre to avoid stasis and aggression of the iliac artery in May Thurner syndrome.*

According to Poiseuille, the pressure gradient P1-P2 = $Q.8L\mu/\pi r^4$

The measurement ot the designed stent is performed with the following international units:

DP=P1-P2=pressure gradient= PA(Pascal) 1PA= 1/98.0638 cmH^2O = 0.74/ 98.0638 mmHg

Q=flow rate: m3/s L=length in metres r=radius =metres μ=Viscosity of blood = 6.10-3

Values in laminar (Newtonian) regime in a cylindrical vessel, in the absence of turbulence (Reynolds < 2500). The shear stress and Reynolds number as well as the shpae of the zone interfere but only for very large flows....

The value of Blood Viscosity, depending on the source, varies from 4 to 25 (usually 6) × 10-3 for T°= 37°.

Here is an example of a stent measurement.

Example of a measurement for a requested flow rate = 120 ml/min.

Resistant back pressure DP =0.05mHg for a flow rate of 120 ml/min with a stent of 16 mm diameter and 10 cm length.

Diameter = 16 mm, L=10mm, Flow = 120 ml/minute, v=25.6 cm/s

Q= 0.000002 m3

x 8μ (8 x 6. 10-3= 42. 10-3)= 0.000002 x 0.042 = 0.000000084

x L= 0.01 m = 0.00000084

r = 0.008 m N4= 0.000000004 m

X π= 0.00000001256

DP= 0.0000000084/ 0.00000001256 = 0.67 PA= 0.005mmHg

Diameter = 16 mm, L=100mm, Flow = 120 ml/minute, v=25.6 cm/s

DP=0.05 mmHg

Resistant back pressure DP =0.5mHg for a flow rate of 700 ml/min with a stent of 16 mm diameter and 10 cm length.

88- *Venous Malformations.*

The hemostator is a fast, efficient and haemostasis tool.

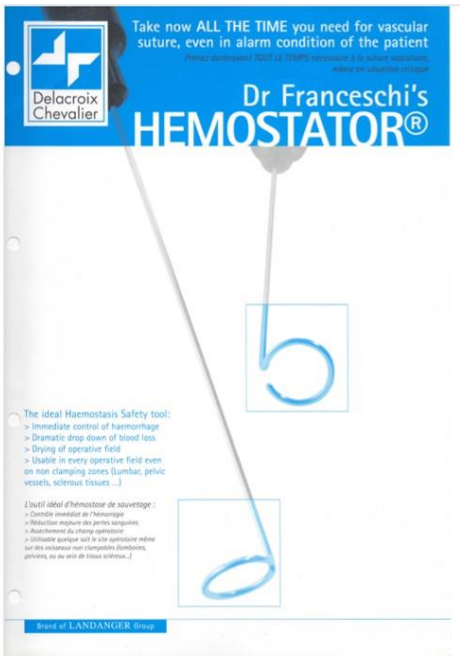
In case of bleeding that is difficult to control, it is sufficient to press the ring (variable diameter: 1,2,3,4cm) against the source of the bleeding.

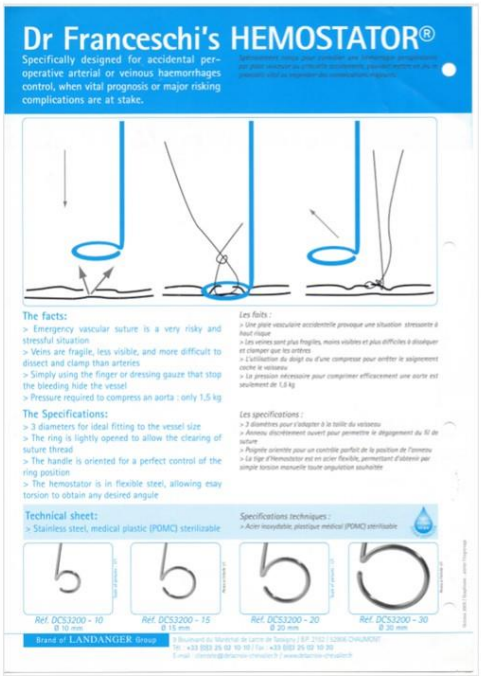
The vessels surrounding the vascular wound are clamped by the ring which compresses them, thus stopping the bleeding.

The vascular wound is dried in the centre of the ring where it can be easily sutured.

A small opening in the ring allows hemostator to be removed without cutting the thread. This tool is also very useful in any vascular or non-vascular surgery, to control bleeding that is difficult to access or that floods the operating field. For example, it can be

used to reduce bleeding in vascular malformations surgery.

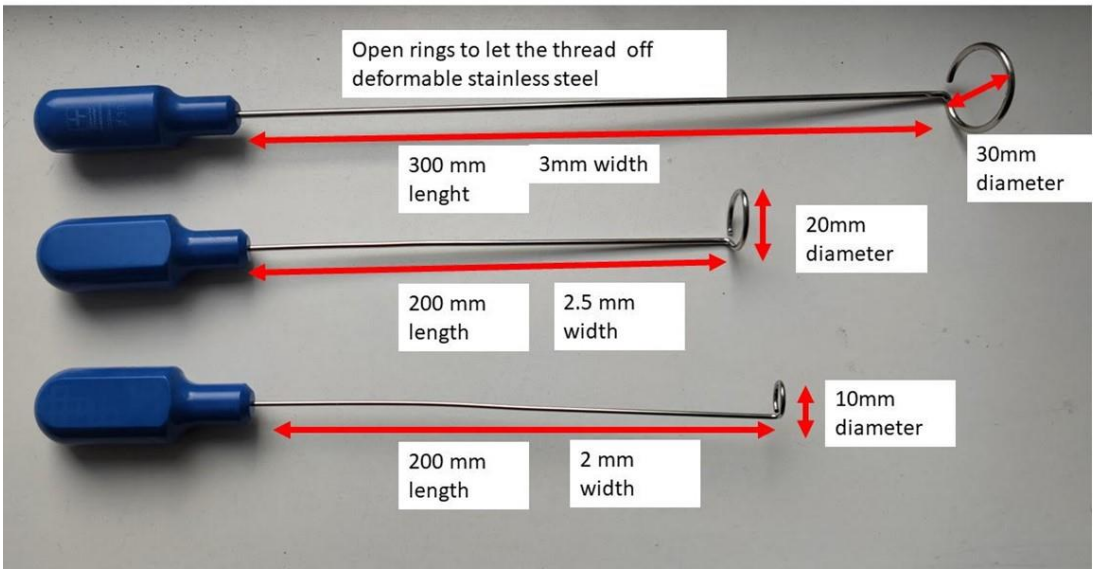




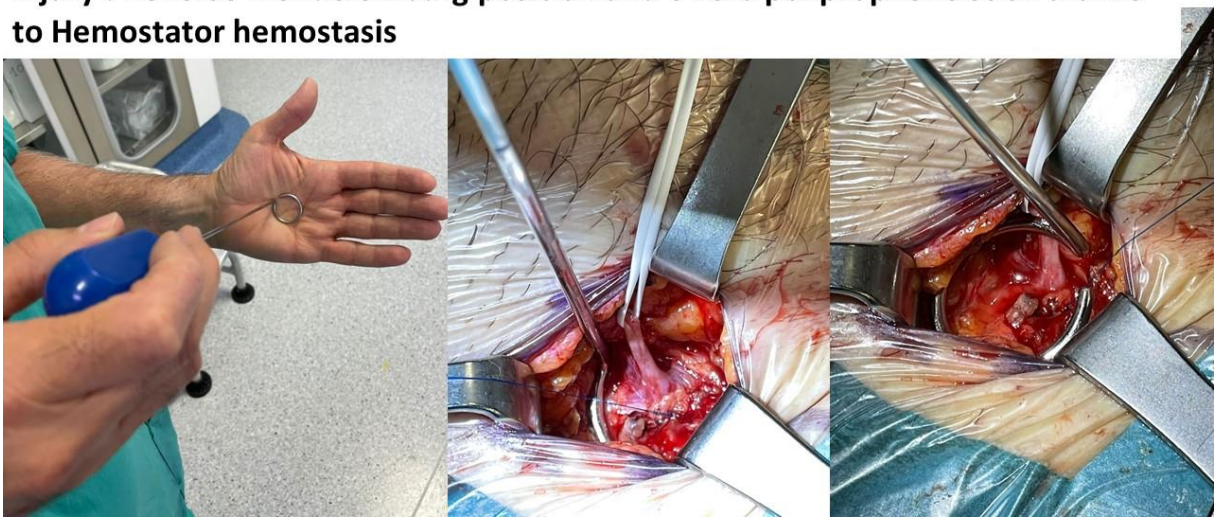
<u>Note that I have released the patent and that the hemostator can therefore be easily copied</u>

.

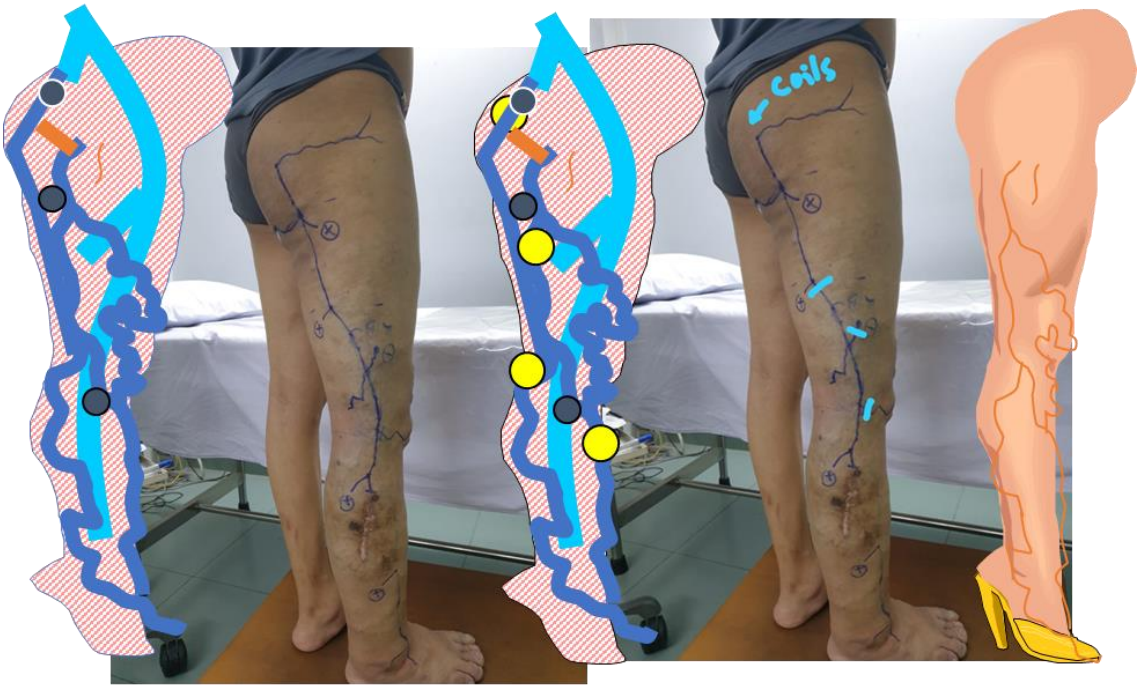
Patent free. Easy to copy.



About hemostator:
Redoo surgery for groin recurrence with systolic valsalva refux through the terminal valve. Previous anterior saphena disconnection. Bleeding from stump injury . Reverse Trendelemburg position and 6 zero polipropilene stich thanks to Hemostator hemostasis

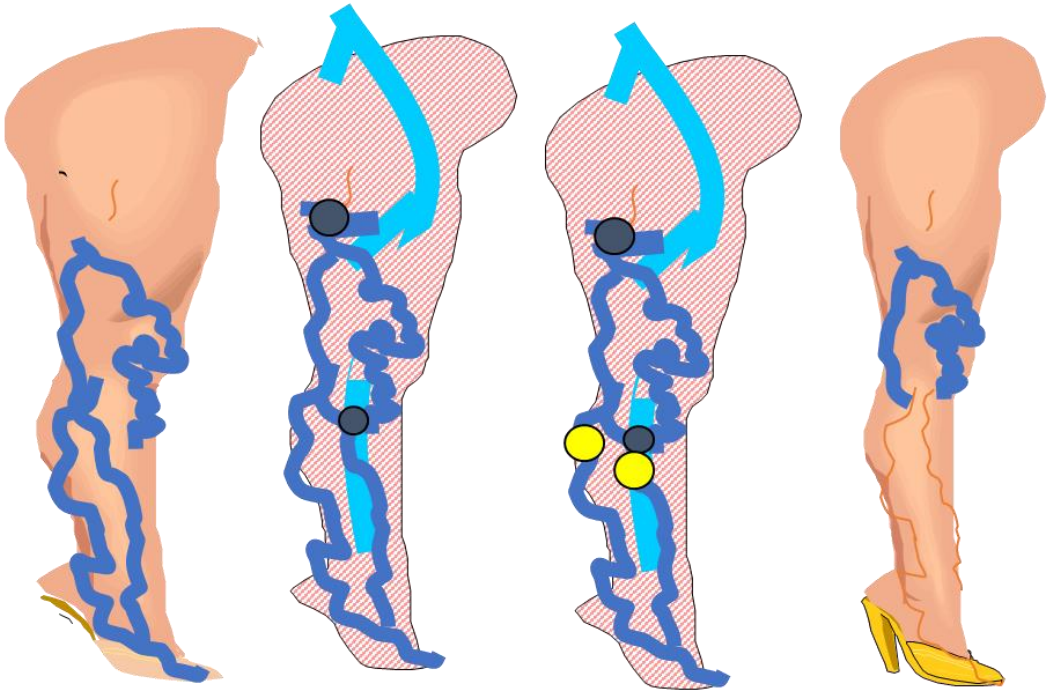


Same clinical aspect, but different hemodynamic conditions identified by echodoppler and different treatments

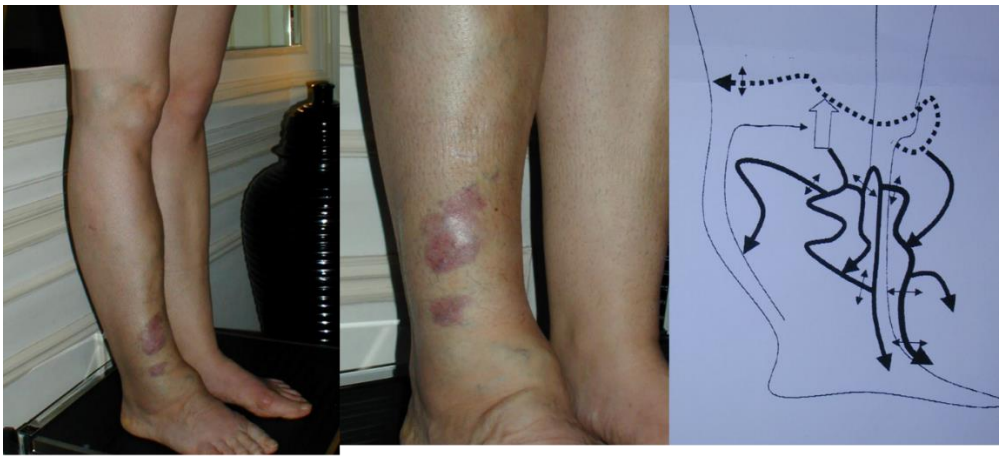


Dr Le Thanh Phong Hô Chi Minh-Ville Vietnam

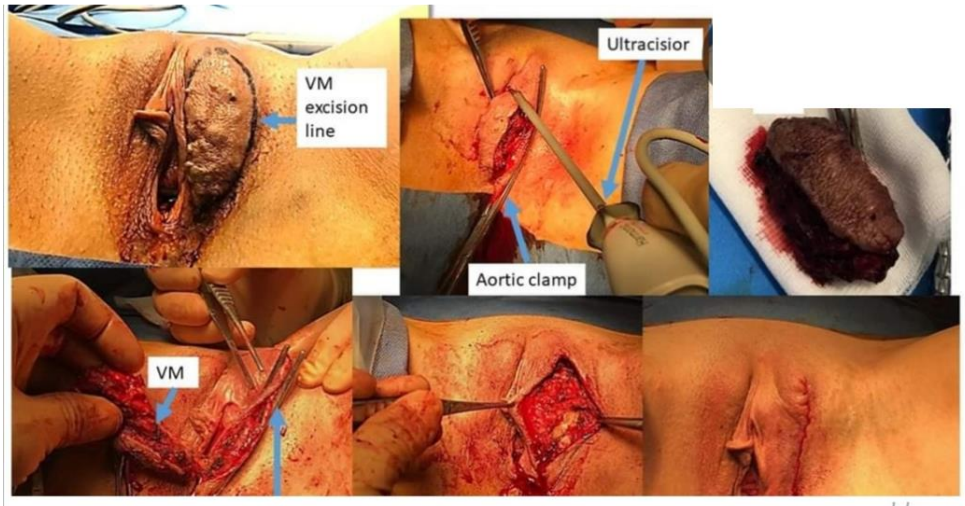
Klippel Trenaunay Weber syndrome without vicarious varicose veins . Staged Shunt VI to disconnect. No phlébectomy

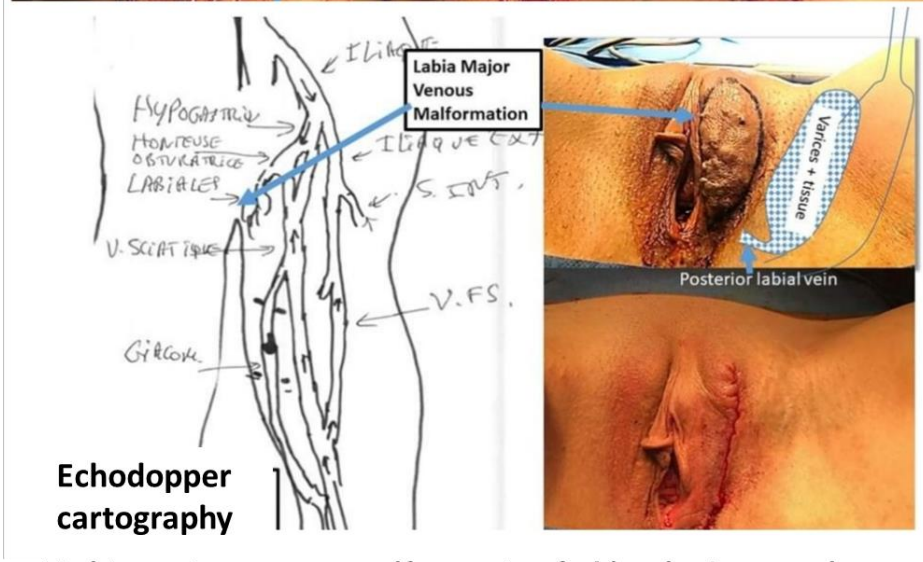


Klippel Trenaunay Weber syndrome, same clinical aspect BUT different hemodynamic configuration. The varicose Marginal vein is an OVS that compensate a femoral vein hypoplasia. Shunts VI are disconnected but the varicose SOV is preserved in order to drain the leg.



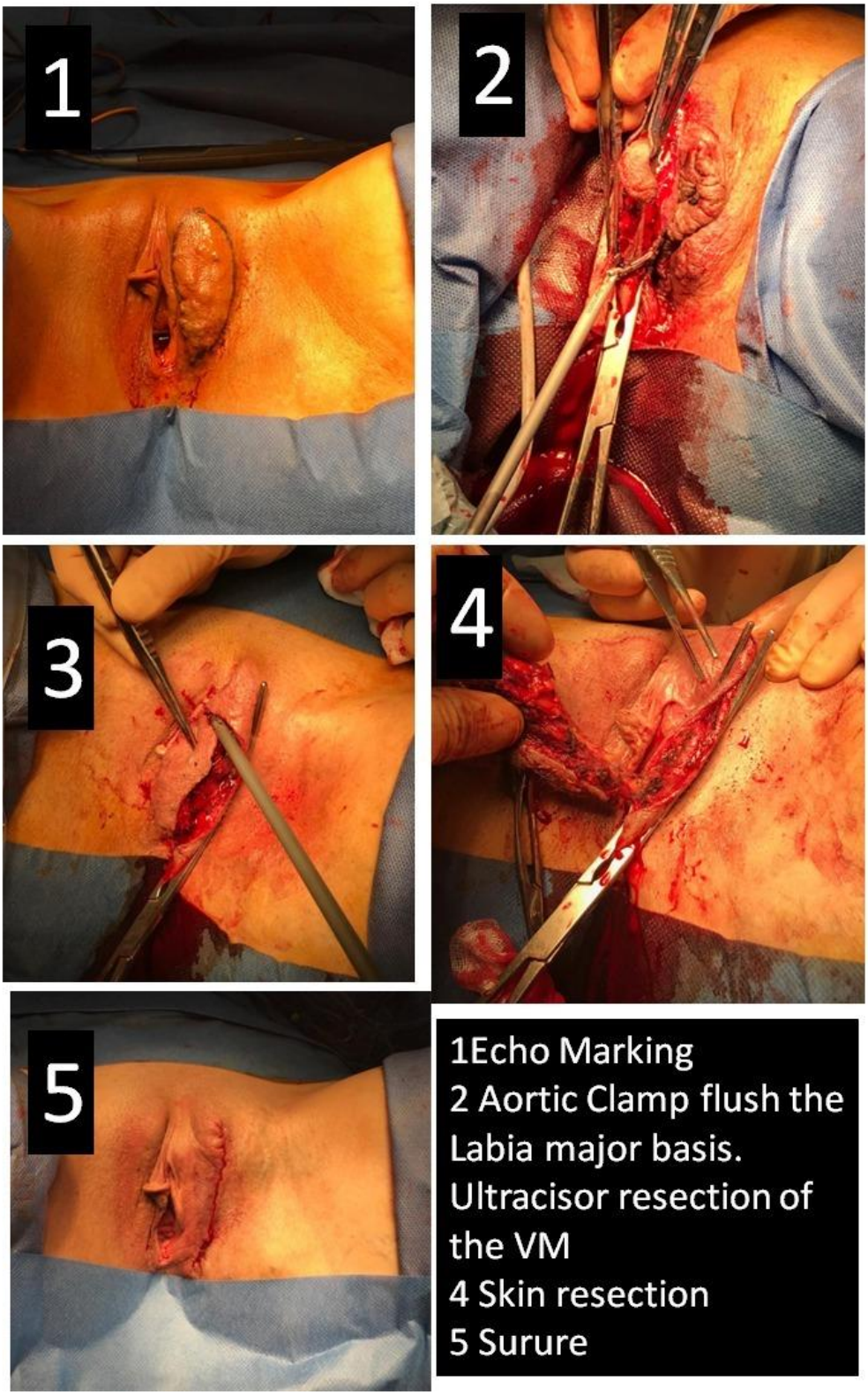
Venous malformation fed by varicose veins closed shunts VI+ shunts II to be disconnected without phlebectomy.





Echodopper cartography

Labia major venous malformation fed by the interrnal pudendal vein via the posterior labial vein + Giacomini shunt II. Hemostatic Aortic clamp flush the root of the maformation to prevent hemorrhage during the exeresis.

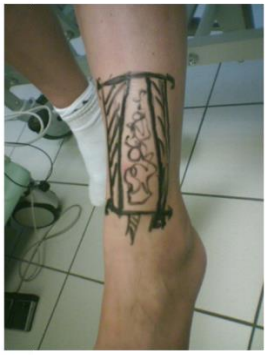

1
2
3
4
5
1Echo Marking
2 Aortic Clamp flush the Labia major basis.
Ultracisor resection of the VM
4 Skin resection
5 Surure

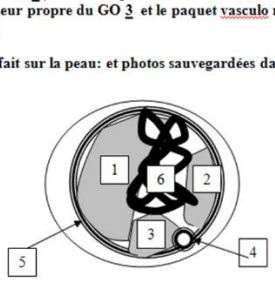
Les TRONCS VEINEUX PROFONDS des deux membres inférieurs sont normaux, sans incontinence, ni thrombose,ni altération pariétale et sans séquelles post-phlébitiques.

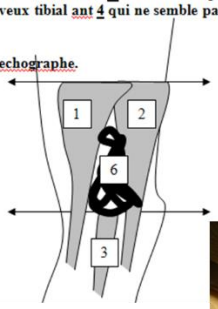
Les TRONCS VEINEUX SUPERFICIELS des deux membres inférieurs sont normaux, sans incontinence, ni thrombose,ni altération pariétale et sans séquelles post-phlébitiques.

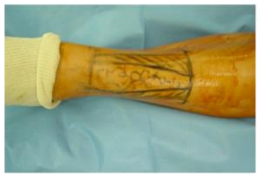
Les TRONCS VEINEUX PROFONDS des deux membres inférieurs sont normaux, sans incontinence, ni thrombose,ni altération pariétale et sans séquelles post-phlébitiques. **SAUF:**
ANGIOME VEINEUX 6 capillaire intrant partiellement caverneux (partiellement compressible et liquide) , sous-aponevrotique 5 , large de 17 mm et profond de 16 mm au 1/4 inf de la loge ant de jambe gauche (78 mm de haut) limité en dedans par le muscle et tendon jambier ant.1 , en dehors par le muscle et tendon extenseur commun 2 et en arrière par l'extenseur propre du GO 3 et le paquet vasculo nerveux tibial ant 4 qui ne semble pas être infiltré.

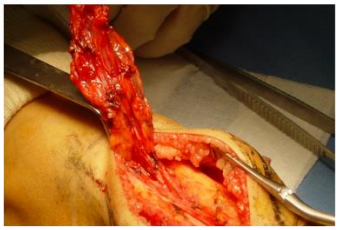
Dessin fait sur la peau: et photos sauvegardées dans echographe.



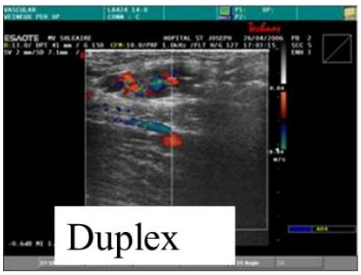


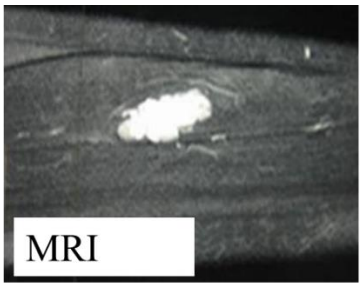




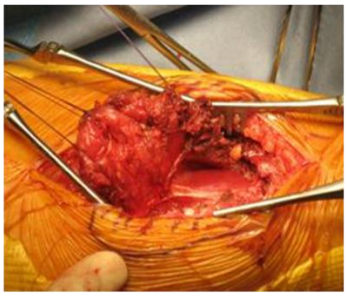


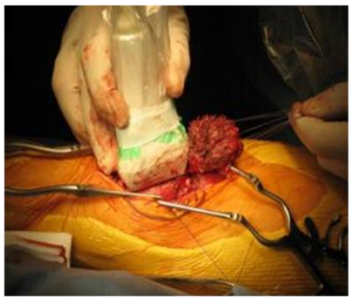
Acurate Echodopper superficial and deep cartography and marking in order to ease the exeresis of a capillar veous malformation

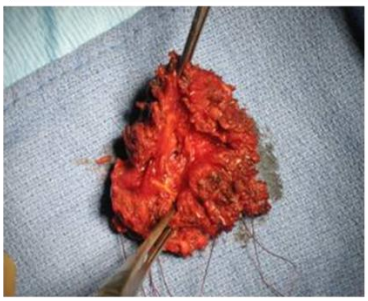


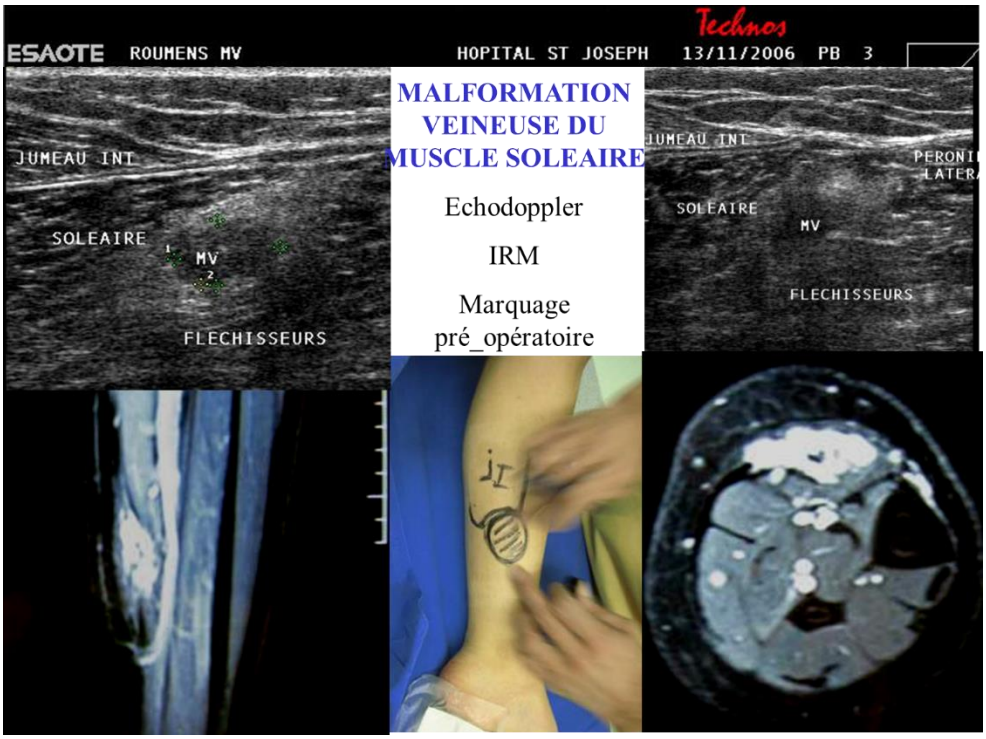


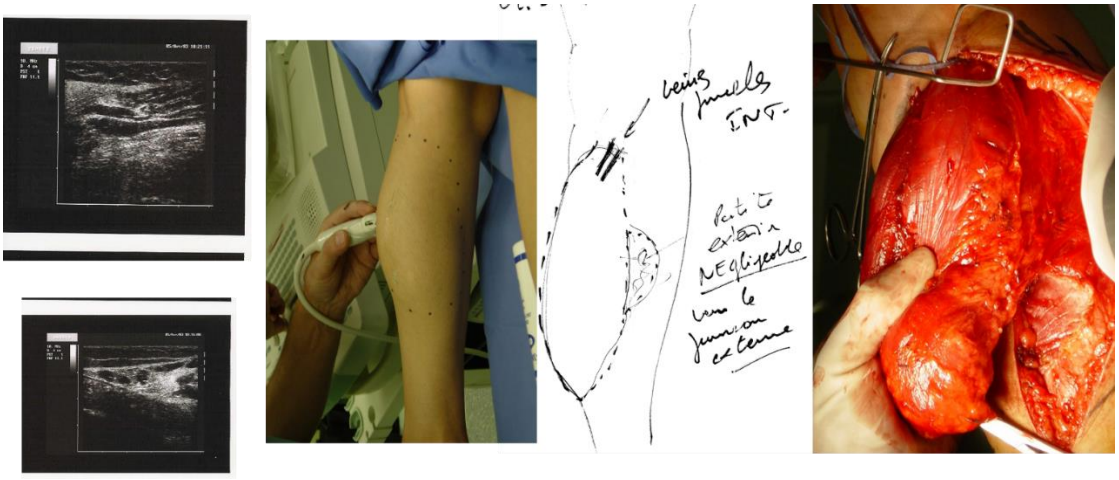
Duplex marking











Venous malformation of all the veins of the medial gastrocnemous muscle no more functionnal (no voluntary nor reflex contraction). Echodoppler Muscle exeresis.

VM sub aponeurotic Ant. Tibial compartment.Previous foam treatment.Pain increased after foam (1 year) .VM unchanged

Echo guided surgical exhaustive ablation

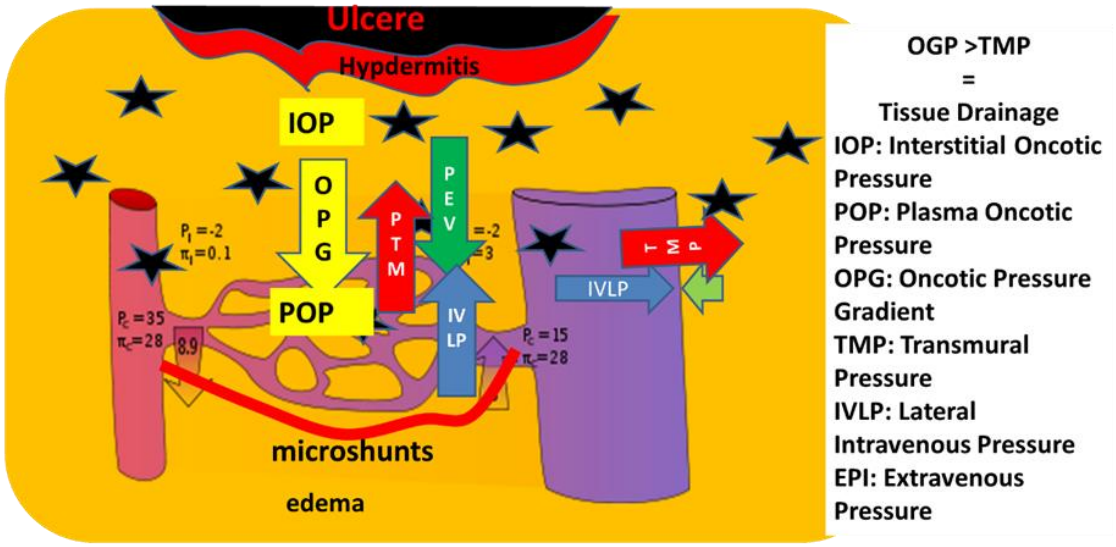
30 days post Op. Swelling dramatically reduced. No pain.

89- *Venous ulcers.*

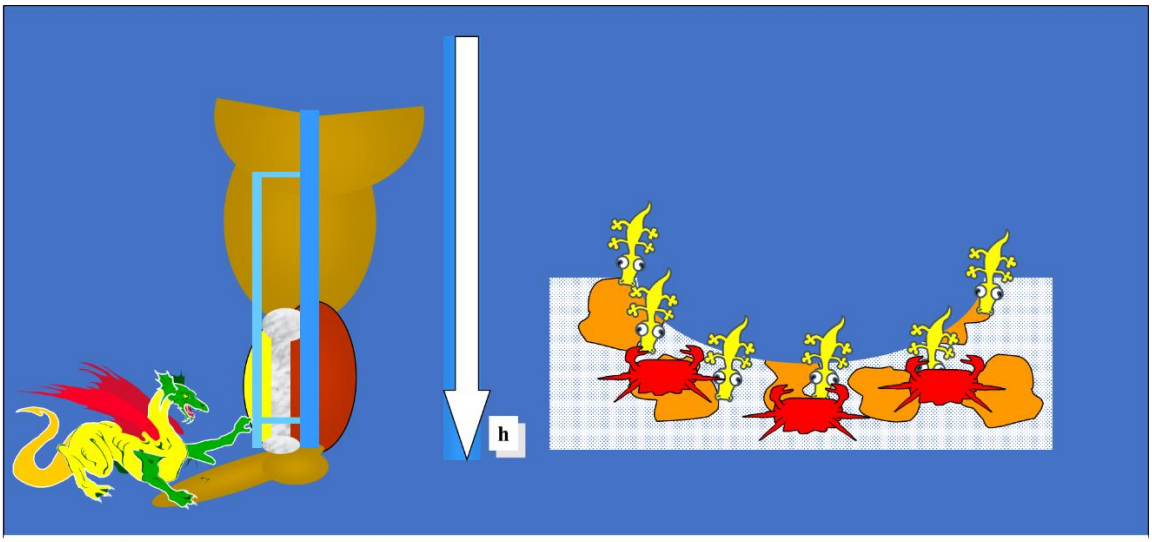
Physiopathological reminder:

An ulcer is a wound that does not heal in the usual time. There are many causes, arterial, venous, capillary, infectious, Paraneoplastic, neoplastic

As we have just seen, the starting point of the venous ulcer is usually at the ankle, where drainage conditions are the most precarious, opposite the re-entry perforators. It then spreads mainly due to superinfection. It is also remarkable that the bleeding is "red" with a normal or even elevated venous oximetry, which could be due to an opening of the micro-shunts that steal the capillaries.



Drainage failure due to Transmural Pressure TMP Excess
Edema, accumulation of toxic catabolites, Hypodermitis
Opening of micro-shunts



Venous ulcer begins at the ankle promoted by: Hydrostatic pressure + Bony subsoil + Poor drainage pathways
Aggravated and extended by superinfection

Note that these re-entry perforators are not the cause of the ulcer, but only the point of excessive pressure/flow overload of the closed shunts. Removing them not only removes the cause but also the drainage pathway. Therefore, disconnection of the responsible shunt removes the cause and preserves the drainage, which ensures a good and lasting healing of the ulcer. BIBLIO Discussions and controversies about the pathophysiology of venous ulcers (peri-capillary fibrin sheath (Browse and Burnand theory, release of inflammatory mediators by white blood cells.), have no Practical meaning for treatment, when it is understood that any venous ulcer only occurs if the TMP is excessive and heals if the TMP is normalized and this is all the faster if the infectious complication is effectively treated.

The effective and long-lasting treatment is the correction of the cause of the excess of lateral intravenous pressure (IVP). Release of venous obstructions (stents and bypasses), and/or correction of the effects of incompetence by CHIVA

Ref: P.Zamboni and al Minimal Invasive Surgical Management of Primary Venous Ulcer vs. Compression Treatment: a randomized Clinical Trial .Eur J Vasc Endovasc Surg °°,1-6 (2003) ***and/or valve repair/Prosthesis. The long maintained supine position with the leg raised is not sustainable in terms of its personal and socially disabling effects.***

Increasing extra venous pressure by compression is an excellent adjunct to the reduction of IVLP.

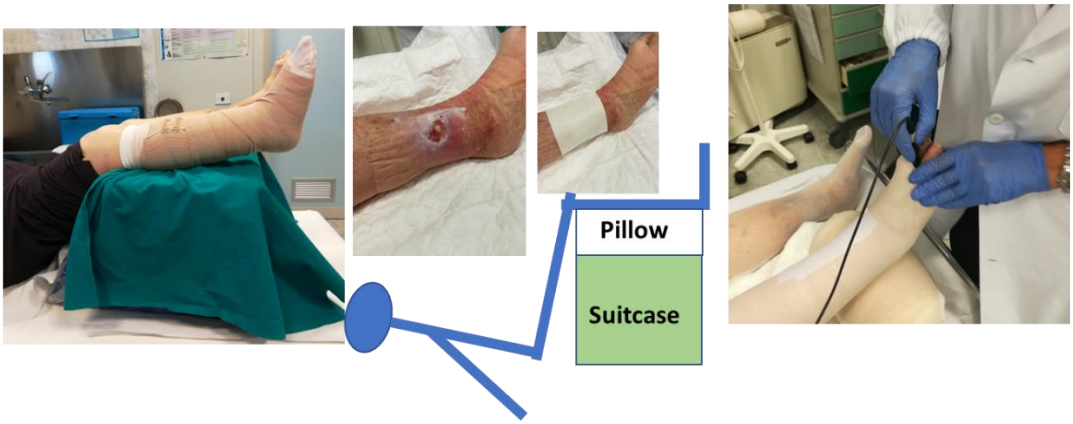
Treatment of secondary infection is effective in accelerating hemodynamic recovery. For several decades I have successfully used a mixture of 60% Vaseline 40% sugar applied once

a week, without debridement or antibiotics but with a non-elastic restraint applied after 2 hours of supine posture with legs raised to drastically reduce oedema.

Ref: Claude Franceschi,Massimo Bricchi, Roberto Delfrate. Anti-infective effects of sugar-vaseline mixture on leg ulcers. Veins and Lymphatics 2017; volume 6:6652.

Non-elastic compression after 2 hours of elevation + light elastic compression

Distal arterial Doppler control



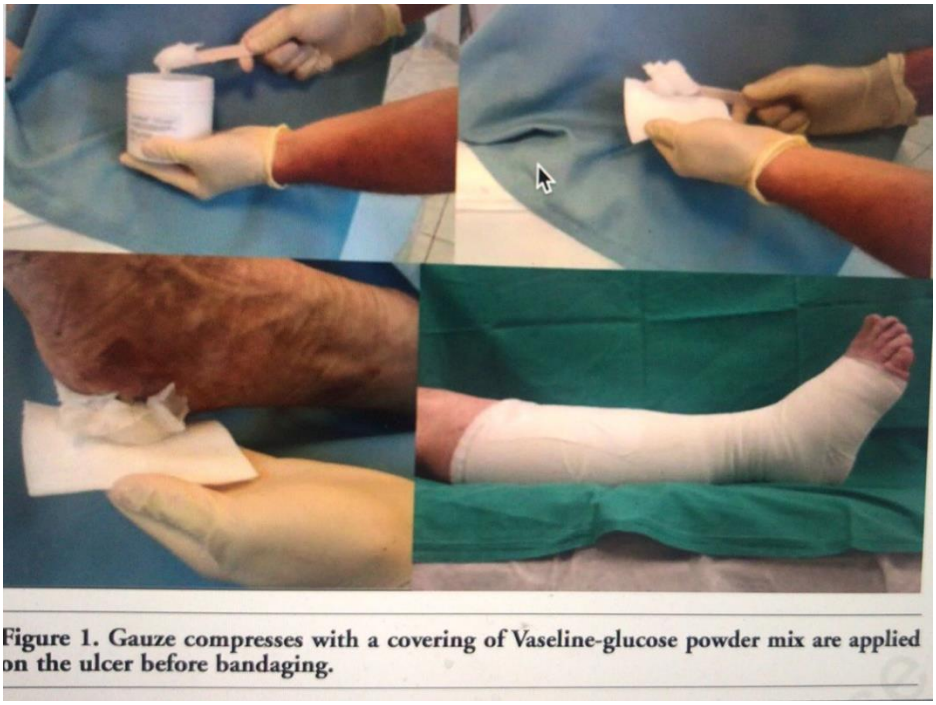


Figure 1. Gauze compresses with a covering of Vaseline-glucose powder mix are applied on the ulcer before bandaging.

80A- Haemorrhoids

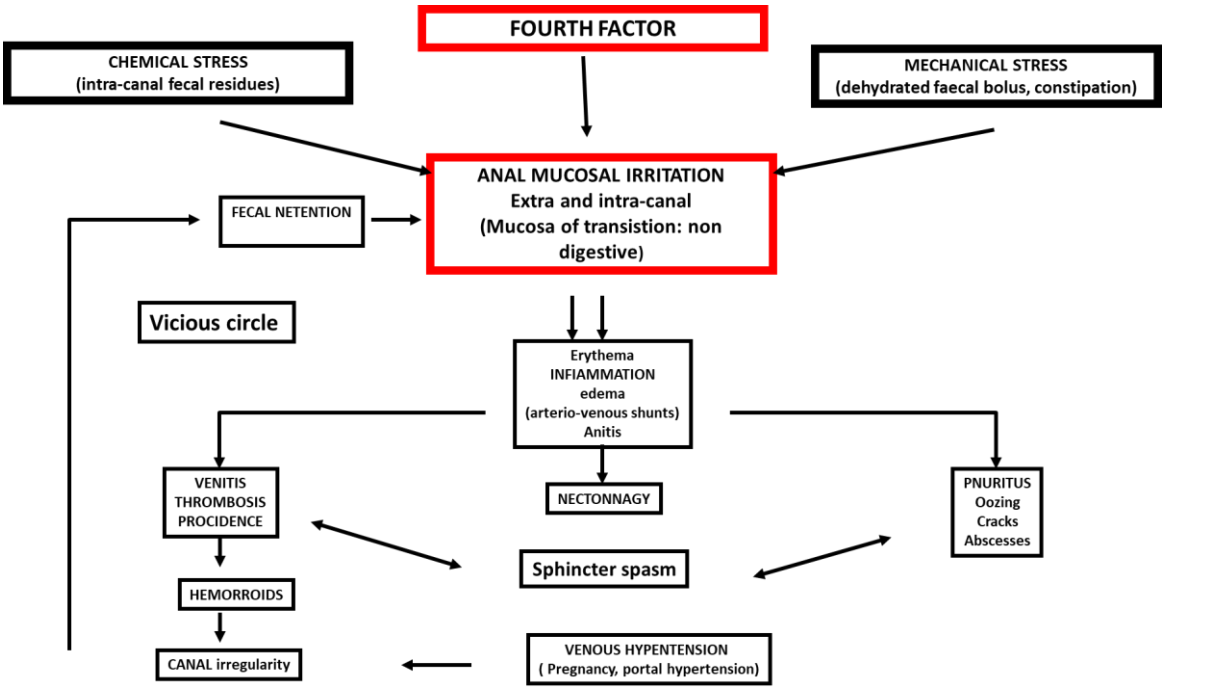
Ref: ***C.***Franceschi. Hémorroïdes : maladie des veines ou d'un quatrième facteur. Essai d'analyse physiopathologique. Conséquences thérapeutiques. Actualités Médicales Internationales. Angiologie (8), n° 145, décembre 1991 VIDEO https://youtu.be/1FoYynLlb98**.**

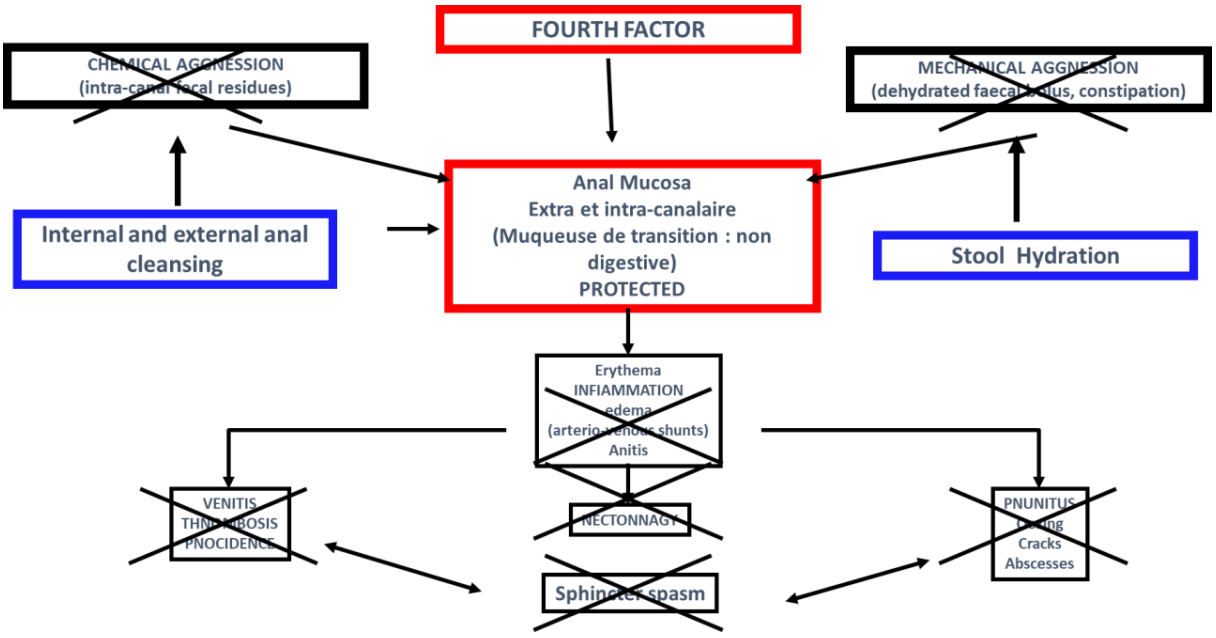
Hemorrhoidal thrombosis is a venous complication but not the cause of "hemorrhoids". The cause is infection and chemical and traumatic irritation of the anal canal, the transitional mucosa.

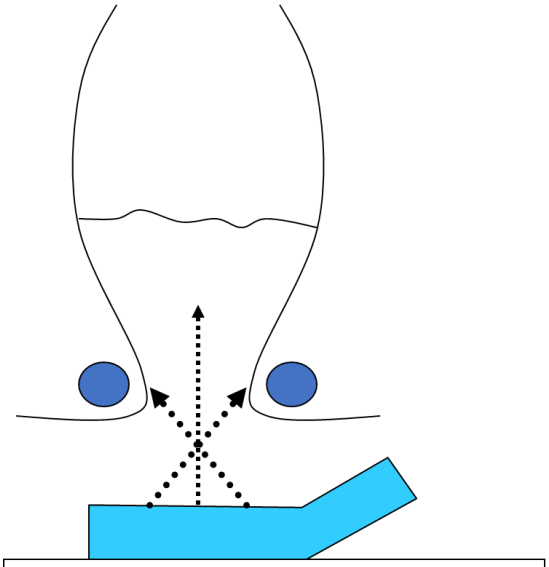
Keeping this canal unaffected mechanically by constipation and chemically by fecal residues stops or prevents the evolution towards complications. This has been demonstrated by an RCT study which showed that atraumatic pre and post evacuation rinsing by a jet of water penetrating the canal and the rectum emitted by a conduit without contact with the perineum.

I have released the patent. Intrajet can therefore be freely copied.

Anorectal varicose veins, internal, external, retractable or not, depending on their stage, thrombosed or not, are rarely due to venous hypertension. They are most often secondary to the 4th factor as I described it in 1995 (see article below) This factor is the intolerance of the mucous membrane of the anal canal (transitional mucous membrane, not digestive, like the oropharynx) which becomes inflamed, traumatized and infected by the mechanical and chemical contact of faecal matter. The hemorrhoidal veins, just under this mucosa, would suffer the consequences. The hemorrhoidal crisis of the pregnant woman is facilitated by the dilation of the hemorrhoidal veins which deform the mucous membrane of the canal. This deformation favours the pathogenic contact with faecal matter. An RCT study demonstrated the effect of pre and post defecation washing of the anal canal by a simple jet of water penetrating without contact with the cannula which remains at a distance from the anus.

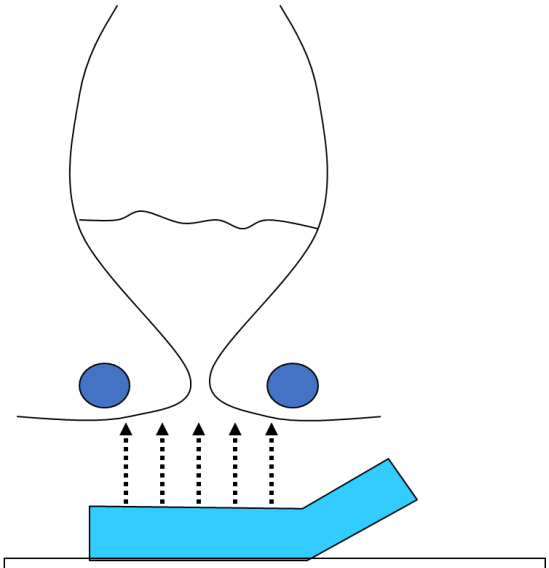

FOURTH FACTOR
CHEMICAL STRESS
(intra-canal fecal residues)
MECHANICAL STRESS
(dehydrated faecal bolus, constipation)
ANAL MUCOSAL IRRITATION
Extra and intra-canal
(Mucosa of transistion: non digestive)
FECAL NETENTION
Vicious circle
Erythema
INFIAMMATION
edema
(arterio-venous shunts)
Anitis
VENITIS
THROMBOSIS
PROCIDENCE
NECTONNAGY
PNURITUS
Oozing
Cracks
Abscesses
Sphincter spasm
HEMORROIDS
CANAL irregularity
VENOUS HYPENTENSION
(Pregnancy, portal hypertension)


FOURTH FACTOR
CHEMICAL AGGNESSION
(intra-canal fecal residues)
MECHANICAL AGGNESSION
(dehydrated faecal bolus, constipation)
Internal and external anal cleansing
Anal Mucosa
Extra et intra-canalaire
(Muqueuse de transition : non digestive)
PROTECTED
Stool Hydration
Erythema
INFIAMMATION
edema
(arterio-venous shunts)
Anitis
VENITIS
THROMBOSIS
PROCIDENCE
NECTONNAGY
PNUNITUS
Oozing
Cracks
Abscesses
Sphincter spasm

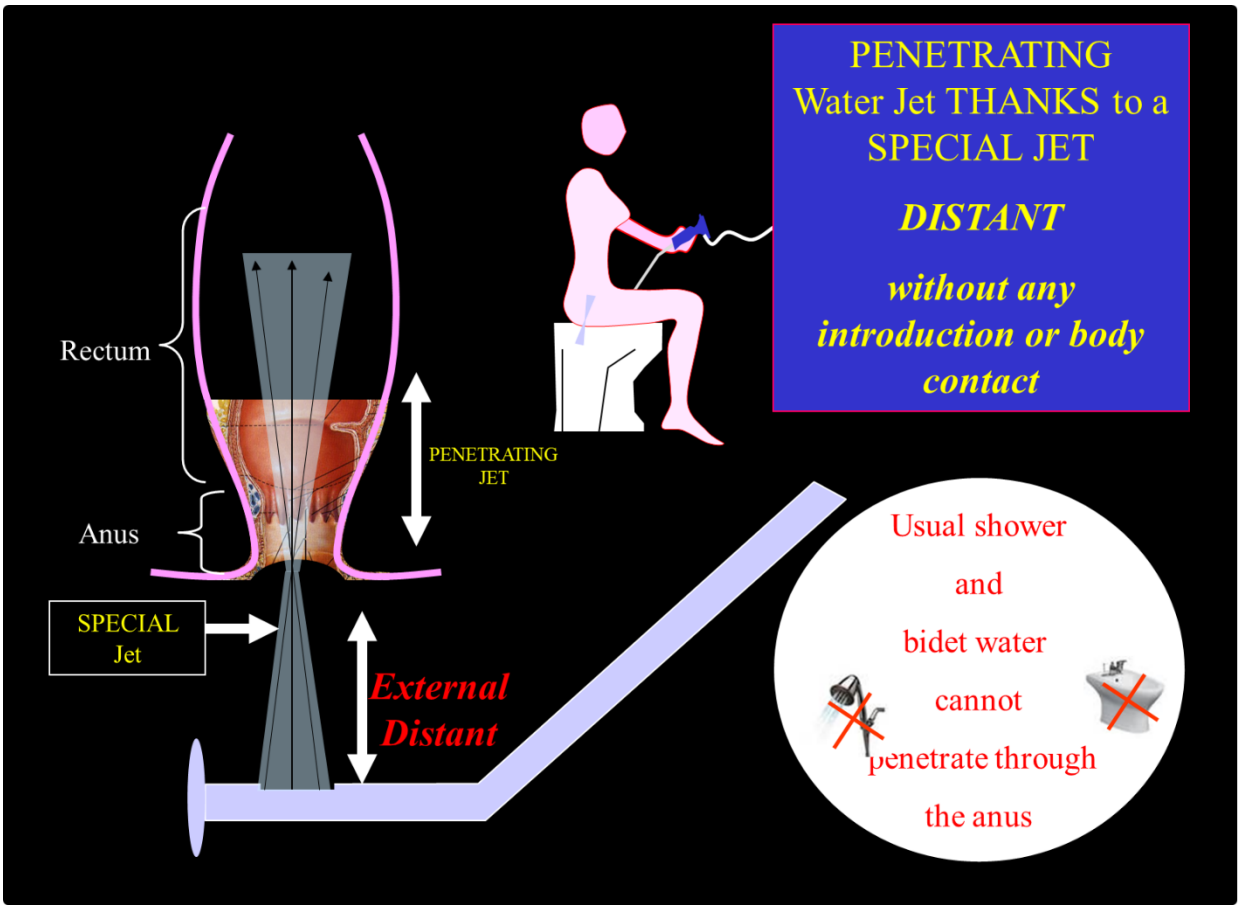


EXTERNAL focused water jet,
Penetrates without cannulation or contact

Intrajet system



EXTERNAL NOT focused water jet,
does not penetrate



MEDECINE ET CHIRURGIE DIGESTIVES
Tome 24 - N°2
Mars - Avril 1995

Med. Chir. Dig. 1995 – 24 - 109-111

ACTUALITES THERAPEUTIQUES

Intrajet®

Evaluation de l'efficacité et de la tolérance d'un nouveau procédé de traitement des hémorroïdes symptomatiques : Intrajet®*

*B. VERGEAU**, R. CLEMENT**, M. MASSONNEAU***, C. FRANCESCHI*****

(Vincennes, Paris)

Introduction

Les hémorroïdes feraient souffrir un sujet sur trois et constituent une véritable maladie sociale. L'étude IJ 301 avait pour but d'évaluer l'efficacité et la tolérance d'Intrajet® dans le cadre des hémorroïdes symptomatiques. Intrajet® est un dispositif qui permet l'introduction d'eau dans le canal anal au moyen d'un jet dont la particularité principale est d'être pénétrant sous pression modérée sans canulation ni contact de l'appareil avec le périné. Cette action est rendue possible grâce aux caractéristiques de focalisation et d'orientation du jet.

Cette étude était fondée sur une approche physiopathologique privilégiant l'intolérance de la muqueuse du canal anal aux résidus même minimes de matières fécales (C. Franceschi).

Matériel et méthode

Principe d'Intrajet®

Nous avons élaboré un système permettant d'une part de contrecarrer l'agression mécanique en ramolissant le bol fécal distal avant la défécation et d'autre part de supprimer les résidus fécaux consécutifs à la défécation sans agression chimique ou mécanique ni risque de contamination. Il fallait enfin que le système soit d'un emploi simple, quotidien, non contraignant et peu onéreux. Le principe d'Intrajet® consiste à faire pénétrer dans le canal anal, un jet d'eau, émis à distance de l'anus par un appareil externe, donc sans contact et non contaminant, de pression assez faible pour ne pas traumatiser ni remonter au-delà du bas du rectum, de forme et direction particulières afin de pouvoir être pénétrant. Ce jet est émis avant et/ou après la défécation pendant 4 à 6 secondes. Le dispositif Intrajet® est constitué d'une canne vectrice reliée à l'alimentation d'eau par un tuyau souple et munie d'un robinet poussoir en son manche, recourbée de 40° à son extrémité, de sorte que tenu entre les cuisses par le patient assis sur la cuvette des W.C, l'orifice de sortie du liquide aménagé dans cette extrémité se trouve en face et dans la direction du canal anal. Cet orifice est constitué d'une fente particulière en ce qu'elle génère un jet plat et triangulaire, dont la pointe se forme à 25mm de l'orifice pour se repartir en un léger éventail.

Protocole IJ 301

L'étude IJ 301 a obtenu l'accord du CCPPRB de la Pitié Salpétrière en 1992, a duré 16 mois et s'est interrompue en novembre 1993. Cette étude a été placée sous la responsabilité scientifique du Dr Bertrand Vergeau, chef de service d'endoscopie digestive de l'Hôpital d'Instruction des Armées Bégin. Il a été nécessaire d'utiliser une méthodologie originale car reposant sur un matériel d'hydrothérapie et non sur un médicament, il n'était pas possible d'utiliser un placebo. Il a donc été décidé de tester deux jets d'eau différents :

- Un jet sous pression modérée, orienté et non focalisé, assimilé dans le protocole à un placebo, qui est une douchette anale améliorée. Les douchettes n'ont jamais fait la preuve d'une action thérapeutique dans un protocole de ce type.
- Un Intrajet®, qui lui est un jet sous pression modérée, orienté et focalisé et permet un lavage externe équivalent au précédent et y associant un lavage interne du canal anal.

L'expérimentation IJ 301 a été réalisée en double aveugle contre placebo, ni le médecin ni le patient ne pouvant savoir quel était des deux jets celui qui était à sa disposition. Pour cela le protocole imposait au médecin lors de la première consultation une présentation de l'étude ne spécifiant pas la notion de pénétration. Après accord signé du sujet, un Intrajet® ne disposant pas de la canne terminale était installé dans les 24 heures. Le praticien revoyait le patient le troisième jour, lui remettait une enveloppe scellée et randomisée contenant soit une canne de jet externe soit une canne Intrajet® à effet

* *Intrajet® est distribué par Médi-Santé Recherche, 11 rue Ferdinand Duval, 75004 Paris, Tél. : 44 78 82 64, Fax : 44 78 82 61.*

** *Hôpital d'Instruction des Armées Bégin, Vincennes.*
*** *Société IODP, 11 rue Ferdinand Duval, 75004 Paris.*
**** *12 avenue de Wagram, 75008 Paris.*

externe et interne. L'examen de départ était très complet et le patient acceptait de subir un examen proctologique et anuscopique complet à J-3, J+1, J+15 et J+90 jours.

■ Population

La population étudiée comprenait 31 patients présentant des hémorroïdes symptomatiques qui avaient donné leur accord pour participer à cette étude. L'un des patients est revenu sur sa décision dans le délai de réflexion de 3 jours. Le choix d'une consultation hospitalière avait pour but de tester l'efficacité d'Intrajet® auprès d'une population sévèrement atteinte et ayant subi de nombreux traitements antérieurs. Il apparaît que 20 patients présentaient une gêne quotidienne importante ou très importante à l'inclusion, et que 26 patients souffraient d'hémorroïdes depuis plus de 10 ans.

Le groupe bénéficiant du jet externe seul comprenait 16 patients.

Le groupe bénéficiant d'Intrajet® comprenait 15 patients.

Les deux groupes étaient équivalents en ce qui concerne tous les critères d'âge, de sexe, de poids et de taille et ne présentaient pas de différences statistiquement significatives.

Résultats

Sur le critère principal qui était l'amélioration globale ressentie par le patient, 53 % des patients sous Intrajet® (8/15) ont estimé dès le 15ème jour que l'amélioration globale était importante ou très importante contre seulement 25 % (4/16) dans le groupe sous jet externe, $p < 0.001$, (Fig. 1). L'un des patients bénéficiant de ce seul jet externe décrit déjà son action comme plus efficace qu'une simple douchette anale qu'il utilisait auparavant. A 90 jours, la satisfaction globale à l'égard d'Intrajet® se maintient.

En ce qui concerne les critères secondaires d'étude, aucun n'est statistiquement significatif car il s'agit de sous-groupes avec un nombre de patients trop petit. Cependant la diminution de la douleur est remarquable dans les deux groupes puisque sur l'ensemble des patients, l'intensité de la douleur est globalement divisée par quatre, la réduction du prurit est également importante dans les deux groupes puisque le prurit est globalement divisé par trois. L'amélioration du suintement est très en faveur d'Intrajet®, pouvant être considérée comme statistiquement significative, puisque le suintement est globalement divisé par cinq dans le groupe Intrajet® alors qu'il n'est divisé que par deux dans le jet externe seul (Fig. 2).

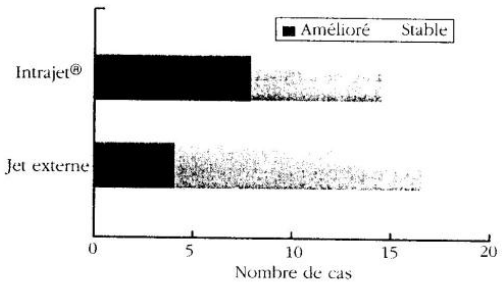


Fig. 1 - Critère principal d'étude : amélioration globale ressentie par le patient
Amélioration avec Intrajet® 53% (8cas) versus Jet externe 25% (4 cas) (p<0.001).

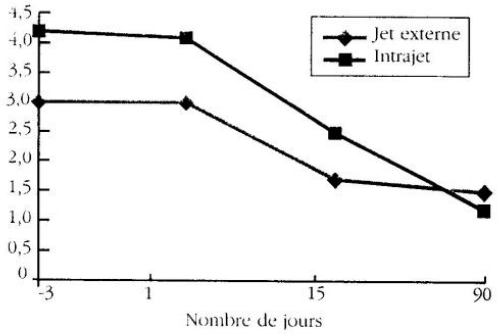


Fig. 2 - Evolution moyenne du suintement au cours du temps Echelle analogique visuelle horizontale (p non significatif)

Les patients présentant des diarrhées et bénéficiant d'Intrajet® n'en ont plus décrit à trois mois, l'un des patients sous jet externe seul présentait toujours des diarrhées à trois mois. Les patients présentant une constipation importante et bénéficiant d'Intrajet® n'en ont plus ressenti dès le quinzième jour, ce qui est confirmé à trois mois, l'un des patients sous jet externe seul présentait toujours une constipation à trois mois. La disparition totale des saignements lors de l'essuyage était observée dans le groupe Intrajet® dès J15 chez les six patients concernés ; chez les huit patients concernés sous lavage externe, quatre en souffraient encore à J15 et un à J90.

En ce qui concerne l'évolution des hémorroïdes, Intrajet® comme le lavage externe, entraîne une diminution modérée en taille des hémorroïdes et même une action étonnante sur les marisques qui ne peut s'expliquer que par une action anti-inflammatoire péri-hémorroïdaire.

Intrajet® réduit plus rapidement le prolapsus hémorroïdaire avec disparition des cas de prolapsus II B dès le quinzième jour contre trois mois pour le lavage externe.

Les hémorroïdes compliquées semblent bénéficier d'Intrajet® puisque trois cas sur sept ne sont plus compliqués à trois mois contre aucune amélioration des six cas sous lavage externe seul. Enfin on note une disparition des cas de thrombose dès J15 dans les deux groupes et surtout

CHAPTER 9

CHIVA literature

By Massimo Cappelli and Paolo Zamboni

The aim of this reviw of CHIVA is to organise the articles in 10 groups accordig to the different gools of the articles:

1° ARTICLES FOCUSING ON THE PRESENTATION OF CHIVA THERAPY

2° ARTICLES CONCERNING SUBJECTES OF CHIVA PROCEDURE NOT IN TERMS OF RECURRENCES / CLINIC DATA BUT OF BIOCHEMICAL, HEMODYNAMIC PARAMETERS, THROMBOSES AND COMPLICATIONS.

3° ARTICLES CONCERNING THE RESULTS OF CHIVA PROCEDURE IN TERM OF RECURRENCES / CLINIC DATA WITHOUT COMPARISON WITH OTHER METHODS

4° ARTICLES COMPARING CHIVA RECURRENCES / CLINICAL DATA WITH OTHER PROCEDURES EMPLOING NOT RANDOMIZED STUDIES

5° ARTICLES COMPARING CHIVA RECURRENCES / CLINICAL DATA WITH OTHER PROCEDURES EMPLOING RANDOMIZED STUDIES

6 ° COCHRANE REVIEW AND METANALYSIS

7° ARTICLES OF GENERAL REVIEW

8° CHIVA AND PELVIC ESCAPE POINTS

9° ARTICLES ABOUT SPARING SAPHENOUS TRUNK AND USE FOR ARTERIAL BY-PASS

10° BOOKS AND CHAPTERS ABOUT CHIVA

Added: Franceschi C Dynamic Fractionizing, of Hydrostatic pressure, Closed and Open Shunts, Vicarious Varicose Evolution, : Have these concepts made the treatmentof varices evolove? Phlebologie 2003 , 56, N°1 61-66

ARTICLES FOCUSING ON THE PRESENTATION OF CHIVA THERAPY

1) FRANCESCHI C.: The conservative and hemodynamic treatment of ambulatory venous insufficiency

Phlebologie. 1989 Nov-Dec;42(4):567-8.

2) C FRANCESCHI, G FRANCO : La cure CHIVA Discussion

Phlébologie, 1989

3) MANDOLESI S, Ballo M, Galeandro I, Filippo S, Migaldi D, Spinelli F, Nasso C, Carbone P, Scaramuzzino L, Passariello F.: The 1st national multicenter study of the CHIVA "Conservative Therapy and Hemodynamics in Venous Insufficiency in Outpatient Departments method of treatment of varices. One-year follow-up"

Ann Ital Chir. 1990 Jul-Aug;61(4):425-7.

Abstract

The authors present clinical and instrumental results of N. 543 operations executed by CHIVA system. These cases are the result of trial performed in seven SIOC (Italian Society of CHIVA Operators) centers executed from November '87 to July '89. Functional and aesthetic results had been very good on over 85% of all cases; superficial thrombosis were verified on 10% of all cases but almost completely asymptomatic. The aa. propose to start a deeper trial on 500 patients choose by rigorous criteria of inclusion.

4) CONSIGLIO L., GIORGI G.: Terapia di exeresi o conservativa?

Minerva Ang. 1991;16, sup.1: pp. 442–3.

5) MELLIERE D, Cales B, Martin-Jonathan C, Schadeck M.: Necessity of reconciling the objectives of the treatment of varices and arterial surgery. Practical consequences. J Mal Vasc. 1991;16(2):171-8.

Abstract

It is unwise to treat patients with varicose veins without thinking about the possibility of atherosclerotic disease occurring later on. The various procedures of stripping, as well as cryosurgery and sclerosis injections in the saphenous veins destroy veins which are at present the best material for femoro-tibial, femoro-popliteal and coronary bypass. Every year, a great deal of limb salvages cannot be achieved because saphenous veins have been previously removed. As arterial disease occurs one or several decades after the venous complaint, every patient with varicose problems may be concerned. Further more, contrary to a frequent opinion, great saphenous veins of varicose patients are often suitable for arterial bypass. As Doppler combined to duplex scan allow to draw a precise map of the superficial venous channels with their endings, amount of flow back, and calibre of the saphenous veins, it is now possible to propose to most patients conservative procedures: ambulatory phlebectomy or sclerosis injections of peripheral veins in case of minor reflux, crossectomy or CHIVA (Ambulatory Hemodynamic Cure of Venous Insufficiency) in case of major reflux, or association of the various technics. Thus, destructing treatments of saphenous veins should be only proposed to patients whose veins are obviously unsuitable for arterial bypass.

6) FRANCESCHI C.: Conservative hemodynamic ambulatory treatment of venous insufficiency Soins Chir. 1992 Mar;(133):29-31.

7) FRANCESCHI C.: Ambulatory and hemodynamic treatment of venous insufficiency (CHIVA cure).J Mal Vasc. 1992;17(4):291-300.

Abstract

Contrasting with the destructive methods of treating varicose veins, the CHIVA cure (Cure Conservatrice et Hémodynamique de l'Insuffisance Veineuse en Ambulatoire) technique is a conservative and hemodynamic approach of this problem. Based on coherent physiological principles, it proposes rigorous analysis followed by effective correction of the hemodynamic disorders, resulting in lasting benefits on the esthetic, functional and tropic changes associated with varicose veins. The results of the CHIVA technique in several french and european-centers, including over 10,000 procedures performed between 1987 and 1991, confirm the value of the method first described by the author in 1988. They confirm the necessity of respecting the strategic and tactical rules of this new approach and the need for specific theorical and practical training.

8) BAILLY M.: Ambulatory and hemodynamic treatment of venous insufficiency (CHIVA cure). Report of an atypical case

J Mal Vasc. 1992;17(3):241-9.

Abstract

After a brief introduction describing the principle, strategy and tactics of hemodynamic treatment of venous insufficiency in ambulant patients, the following atypical case is described. Ms P., aged 38 years, presented a functional symptomatology including waking at night and was obliged to get out of bed. Varices were moderately visible but she asked for treatment for esthetic reasons. A future pregnancy was discounted. Clinical examination and the pulsed ultrasound-Doppler cartogram showed incontinence of the internal perineal, opening from the genitofemoral fold and rejoining the

summit of Jacomini's vein arch. The dorsal segment of Jacomini's vein was incompetent with a retrograde flux and rejoined an incompetent short saphenous continuing as a type III shunt. The cartogram obtained, the strategy decided, and the intervention carried out on 19 June 1990 are illustrated by photographs. At 4 month follow up and despite the heat wave of summer 1990, all functional signs had disappeared and a sufficiently esthetic result had been obtained. Unexpectedly, the patient announced that she was pregnant.

9) HUGENTOBLER JP, Blanchemaison P.: Ambulatory and hemodynamic treatment of venous insufficiency (CHIVA cure). Study of 96 patients operated on between June 1988 and June 1990.J Mal Vasc. 1992;17(3):218-23.

Abstract

96 patients were treated in two years by Ambulatory and Hemodynamic Treatment of Varicose Veins (CHIVA cure), representing 131 legs that underwent surgery. 71 patients (74%) representing 102 CHIVA cures in the long saphena territory were followed up, with a maximum of 28 months of follow-up. The CHIVA cure represents a new and interesting therapy: ambulatory, painless, it allows a very early resuming of normal life. The follow-up shows that the aesthetic and functional results are especially satisfying and seem to be steady. The CHIVA cure certainly neglects the histological and parietal aspects of venous incompetence, but the treatment of the hemodynamic factor is effective. It can be applied on every type of varicose veins concerning the long saphena territory, provided that the deep venous system is normal. The interest of CHIVA cure concerning the short saphena territory remains to be demonstrated. Arteritic patients, patients with coronary arteries diseases, sportmen, young patients with a brief evolutive potential, especially women, are the best indications. The CHIVA cure is a reliable strategy in the short and medium term, offering excellent aesthetic and functional results that still have to be confirmed in the long term and or a large scale.

10) BAILLY M.: Cartographie CHIVA

Encyclopédie Médico–Chirurgicale, Paris 1993, pp. 43–161 – B, pp. 1–4.

11) FRANCESCHI C.: La cure Chiva et la critique: 14 réponses et 1 conclusion STV.

Sang thrombose vaisseaux, 1993

12) ZAMBONI P. et Al.: Video-assisted venous surgery.

Ann Ital Chir. 1995 May-Jun;66(3):379-86.

Abstract

The use of intraoperative angioscopy, till now utilized exclusively in arterial surgery, is now used also in venous surgery. From January 1992 54 patients underwent to video-guided venous surgery: 23 cases of external valvuloplasty of the sapheno-femoral junction (EV-SFJ), 25 cases of hemodynamic correction of varicose veins (French acronyms CHIVA), 5 cases of high ligation plus long saphenous vein intraoperative sclerotherapy (HL-IS) 1 case of sub-fascial perforators interruption (SPI), the only extraluminal videoguided procedure. We have used 3 different video-angioscopes: a 1 mm monofibroscopy let in a 6 Fr Fogarty catheter, a disposable 2,8 mm colangioscope and a 2,2 mm operative angioscope. For the perforators interruption we have utilised the thoracoscope. EV-SFJ: the angioscopy has confirmed the presence of normal valvular cusps in a dilated vein wall in 21 cases, so excluding 2 patients from the planned treatment. At the end of the operation the angioscope has

verified the reapproach of valvular cusps. CHIVA: the angioscopy has allowed to identify the exact points of the superficial venous system which should be interrupted, according to the Franceschi's theory. This procedure can avoid the technical errors due to intraoperatory misleadings of the duplex mapping. HL-IS: consists of a classic high ligation followed by long saphenous vein intraoperative sclerotherapy. The angioscopy has allowed a complete deconnection of the long saphenous vein from tributaries and perforators. Furthermore, has facilitate the proportional distribution of the sclerosing agent along the long saphenous vein. SPI: the videoassistance have permitted the identification of the insufficient perforating veins reducing their surgical exposures.

13) ZAMBONI P. et Al: Haemodynamic correction of varicose veins (CHIVA): An effective treatment?

Phlebology 1996, 11, pp. 98-101.

14) LEONI V., MISURI D.: IL TRATTAMENTO DELLE VARICI DEGLI ARTI INFERIORI MEDIANTE CHIVA 2. NOSTRA ESPERIENZA

UO di Chirurgia Generale Ospedale S.M.N Firenze - academia.edu 1996

15) A BANHINI, C Franceschi, X Mouren, P Caillard et Al.: surfacevenous insufficiency

JOURNAL DES MALADIES VASCULAIRES, 1996

16) FRANCESCHI, C.: Physiopathologie hémodynamique de l'insuffisance veineuse des membres inférieurs

(1997) Actualités Vasculaires Internationales, 22, pp. 17-27

17) FRANCESCHI C. La Cure Hemodynamique de l'Insuffisance Veineuse en Ambulatoire.:

Journal des Maladies Vasculaires. 1997 ; 22 (2) :91-95

18)CAPPELLI M. et Al.: Criteri di scelta della Strategia CHIVA

Arch. Soc. Ital. Chirurgia 4, 118, 1998

19) CAPPELLI M.: POSTERS-Conservative surgery of the saphenous trunks

Journal des Maladies Vasculaires, 1999

20) E MENDOZA: Einteilung der Rezirkulationen im Bein: anatomische und physiologische Grundlagen der CHIVA-Methode Phlebologie, 2002

21) CRIADO E. et Al.: Conservative hemodynamic surgery for varicose veins. Semin Vasc Surg. 2002 Mar;15(1):27-33.

Abstract

Conservative hemodynamic surgery for varicose veins is a minimally invasive, nonablative technique that preserves the saphenous vein and helps avoid excision of varicosities. It represents a physiologic approach to the surgical treatment of varicose veins based on knowledge of the underlying venous pathophysiology gained through detailed duplex scanning. A change in venous hemodynamics is attained through fragmentation of the blood column by interruption of the refluxing saphenous trunks, closure of the origin of the refluxing varicose branches, and preservation of the

communicating veins that drain the incompetent varicose veins into the deep venous system. After surgery, varicose veins regress through a reduction in hydrostatic pressure and efficient emptying of the superficial system by the musculo-venous pump. Obvious advantages of this technique are that it is done in an ambulatory setting, minimizes the risk of surgical complications, and permits a rapid return to full activity. The long-term hemodynamic improvement and recurrence rate of this technique remain to be established.

22) MENDOZA, E.: CHIVA - Alternative oder Ergänzung zum Stripping? (2002) Vasomed, 14, pp. 6-17.

23) HACH W. : What is CHIVA? [Was ist CHIVA?] (2002) Gefasschirurgie, 7 (4), pp. 244-250.

Abstract

The French phlebologist Claude Franceschi introduced the "Cure conservatrice et hémodynamique de l'insuffisance veineuse en ambulatoire" (CHIVA; ambulatory conservative and hemodynamic treatment of venous insufficiency) in 1988. It is based on Perthes' observation (1895) that varicose veins fill on standing and empty on walking when a tourniquet is applied to the thigh. This hemodynamic situtation is intended to be mimicked in CHIVA by graded surgical corrections of the varices. Franceschi's method is based on the theory of the four venous networks differing in the degree of harm they cause when affected. Different shunting patterns are referred to this theory, a shunt being a connection between one venous network and the next. Recirculation R1 designates the intrafascial leading veins. The R2 network comprises the stem veins. They, too, are thought to be situated intrafascially within a special saphenous fascia, which is visible on ultra-sound imaging. The R3 network comprises the epifascial collateral veins in the subcutaneous fat layer regardless of diameter; and reticular veins and capillaries and starburst varices make up R4. The surgical principle consists in flush ligation and division of the great or small saphenous vein junction without crossectomy. The effect of this is that a retrograde stream of blood is still fed into the preserved varicose stem vein, but it is reduced by that part of the retrograde flow that comes from the common femoral vein. Ultrasound diagnosis of the competent perforating veins and conservation of drainage into the deep venous system are considered very important.

24) MENDOZA E.: Classification of the recirculations in the leg: Anatomic and physiologic bases of the CHIVA-method .Phlebologie 2002, 31 (1), pp. 1-8.

Abstract

Recirculation in varicose veins was firstly thought of by Trendelenburg and further developed by Hach. This idea is also the basis on which Claude Franceschi founded his treatment for insufficient veins - CHIVA (in English ACHM). He divided the veins of the legs in the nets R1, R2 and R3. Deep veins correspond to R1, saphenous veins to R2 and epifascial tributaries to R3. Depending on the participation of these nets in the recirculation of a varicosity, Franceschi divided the recirculations in four types, which he called shunts (type I to IV). On these shunts he based the therapeutic decisions for the CHIVA-method. Most of the shunts are type I or III. In this cases the reflux fills the saphenous veins directly from the deep veins via a crosse or a perforator vein. The study of these models of recirculation throws a new light on the understanding of the distally dilated perforator veins, as well as on the direction of blood-flow in the different segments of the veins. Therefore it is interesting not only for persons that perform the CHIVA-method. Without having understood these concepts, it is impossible to judge upon CHIVA.

25) JUAN-SAMSO' J.: Venous haemodynamic surgery in the treatment of varicose syndrome.(2003) Angiologia, 55 (5), pp. 460-475.

Abstract

Aims. The objective of this study is to update our knowledge of the different aspects of this subject, i.e. the rationale behind the method used, the anatomical-functional terminology employed, strategic principles and ways they can be applied. The results reported from the different series available are also analysed. Development. The CHIVA cure technique (ambulatory and haemodynamic treatment of venous insufficiency) was described by Franceschi in 1988. After the initial expansion of the procedure, its use diminished because it had not been submitted to adequate testing. Later standardisation of the method has led to different groups' adopting the strategy with satisfactory results. The terminology put forward by the European CHIVA Association in 2002 allows the different types of strategies in this therapy to be applied accurately. It must be noted that in the register of activities of the Spanish Society of Angiology and Vascular Surgery (SEACV) for the year 2002, a third of the varicose veins submitted to surgery in Angiology or Vascular Surgery units or services in Spain were performed using venous haemodynamic surgery. Conclusions. No definitive evidence exists (randomised prospective clinical trials are under development) in favour of the CHIVA cure, yet the data available do support this procedure as an alternative to stripping in the treatment of varicose veins.

26) **CAPPELLI M.; Molino Lova R.; Ermini S. Franceschi C.:**

Nouvelle approche de la physiopathologie de l'insuffisance veineuse superficielle: conséquences thérapeutiques" Phlébologie 2002, 55, N° 1, 27 – 31

27) E CRIADO, J Juan, J Fontcuberta and J M Escribano: Haemodynamic surgery for varicose veins: rationale, and anatomic and haemodynamic basis. Phlebology Vol 18 No. 4 2003 pag 158-166

Abstract

The treatment of varicose veins has traditionally been ablative in nature and implemented without intent to improve the haemodynamic condition of the lower extremity veins. Haemodynamic surgery attempts to treat varicose veins by changing the reflux pattern while preserving the most efficient venous drainage channels. To implement this treatment modality it is necessary to have a clear understanding of the physiologic principles and the different reflux patterns that form the basis of haemodynamic surgery. Haemodynamic surgery is an emerging treatment for varicose veins, and has received little attention in the English literature. The rationale, and functional and anatomic basis of haemodynamic surgery for varicose veins are herein described.

28) J JUAN J M Escribano E Criado: Haemodynamic surgery for varicose veins: surgical strategy

Phlebology 2005 Vol 20 No. 1 pag: 1-13

Abstract

The haemodynamic approach for the treatment of varicose veins is a minimally invasive, non-ablative procedure that preserves the saphenous vein. The strategic principles for the implementation of this treatment include fragmentation of the venous pressure column, interruption of the venous

segments where reflux originates, preservation of the superficial venous outflow channels to allow adequate drainage of the residual superficial system, and excision of the superficial varicose veins that remain undrained. This treatment modality requires a thorough understanding of the haemodynamic and anatomic rationale on which haemodynamic surgery is construed to tailor a treatment plan individually for each patient. The principles for the implementation of this strategy for the treatment of varicose veins are described here and the results are discussed.

29) C FRANCESCHI C., Bahnini A.: Reponse a article-Mise au point concernant les commentaires sur la cure CHIVA dans l'article des Drs P. Pittaluga et S. Chastanet. Commentaire de P. Pittaluga.Phlebologie, 2008 .

30) F. PASSARIELLO: Suppression of the sapheno-femoral reflux by pure non-saphenous phlebectomy and anatomical structure of the reflux. ACTA PHLEBOL 2008;9:105-7

Aim. Recently, several hypotheses on varicose veins origin were pro- posed. Accordingly, a non-saphenous foam procedure was shown to achieve successful results in the suppression of the sapheno- femoral reflux, being its persistence limited to a small percentage of treated cases. The aim of the present note is to illustrate the hemodynamic basis of these reflux suppressing failures. Methods. Two hemodynamic theoretic simulations were desi- gned to represent adequately this hemodynamic condition. The sapheno-femoral reflux can be classified according to Teupitz into two kinds of different shunts: ShI and ShIII. The abla- tion of the varicosity's of the non-saphenous superficial network can achieve the disconnection of Sh III and VI, but in no way of Sh I. So that, if the main reflux is a ShI the sapheno-femoral reflux does not disappear after the phlebectomy.

Results. The thorough study of the patient's cartography makes the choice of the surgical strategy easier and more precise. Conclusion. Depending on the structure of the reflux and as to immediate results, GSV ablation can sometimes solve comple- tely the varicose pathology, while in other cases it is completely unsatisfying.

31) C. FRANCESCHI C.: Hémodynamique de la maladie postphlébitique : conséquences diagnostiques et thérapeutiques . Journal des Maladies Vasculaires 2008 Volume 33, numéro S1

ABSTRACT

Keywords: Venous insufficiency, Postphlebitic disease

Goal. - To show how recent knowledge of venous hemodynamics makes it possible to advance not only the classic concepts of pathophysiology, but also the diagnosis and treatment of postphlebitic disease.

Problematic. - Postphlebitic disease is a mixture in variable proportions of obstacles to flow and reflux, both responsible for tissue drainage disorders whose clinical functional and trophic consequences are of uneven severity. The advances in conceptual tools should allow a more detailed understanding of these phenomena, with the result that the diagnostic means of investigation can be optimized and therapeutic strategies rationalized.

Description. - Difference between the internal pressures PI and external atmospheric pressure, altitude (PE) which are exerted, on both sides, of the wall of the vein and its capillaries, the

transmural pressure PTM is the cardinal variable of the function venous. It thus modulates tissue drainage and venous gauges. Too high due to either too low PE or too high PI (PR residual pressure too high due to lower arteriolocapillary resistance or obstruction of flow, hydrostatic pressure orthostatic PHS not reduced due to lack of dynamic fractionation PHS FDPHS). This fractionation is obtained by the systolodiastolic action of the VMP which alternately closes the upstream and downstream valves. The ineffectiveness of VMP results either from muscular inactivity (immobile standing or lying posture, paralysis), or from direct or indirect valve incontinence (deep and superficial closed shunts). Postphlebitic disease combines the effects of FDPHS defect and excess RA in varying proportions. Its treatment must logically reduce PTM by increasing PE (compression), reducing PI (PHS by decubitus, FDPHS by repairing defective elements of VMP, PR by respect and / or creation of open vicarious shunts).

Conclusion. - Thanks to these concepts, treatments for venous insufficiency can now be rationally adapted to the different hemodynamic configurations mapped by ad hoc diagnostic methods.

32) FRANCESCHI C.: So as to avoid any misunderstanding about Cure Conservatrice et Hemodynamique de l'Insuffisance Veineuse en Ambulatoire (CHIVA). Phlebology. 2010 Aug;25(4):212; author reply 213.

Comment on: Venous haemodynamics: what we know and don't know. [Phlebology. 2009]

33) MOWATT-LARSSEN, Shortell C.: CHIVA. Semin Vasc Surg. 2010 Jun;23(2):118-22.

Abstract

Based on a theoretical hemodynamic model, CHIVA (conservative hemodynamic cure for venous insufficiency) is an ultrasound-guided, minimally invasive surgical strategy performed under local anesthesia for the treatment of patients with varicose vein disease. After careful duplex mapping, the surgeon performs flush ligation procedures at the proximal origin of key points of reflux while meticulously maintaining superficial venous drainage to prevent varicosity recurrence. The saphenous veins are preserved. The strategy has been shown in studies to be safe and effective.

34) MOWATT-LARSSEN, Shortell CK.: Treatment of primary varicose veins has changed with the introduction of new techniques. Semin Vasc Surg. 2012 Mar;25(1):18-24.

Abstract

New technologies have produced a revolution in primary varicose vein treatments. Duplex ultrasound is now used for preoperative diagnosis, postoperative surveillance, and during many procedures. Ultrasound has also altered our understanding of the pathophysiology of chronic venous disease. Laser and radiofrequency saphenous ablations are common. Classic techniques, such as sclerotherapy, high ligation, stripping, and phlebectomy, have been improved. Magnetic resonance venography, computed tomographic venography, and intravascular ultrasound have improved diagnostic capabilities. New strategies like ambulatory selective varices ablation under local anesthesia (ASVAL) and conservative hemodynamic treatment for chronic venous insufficiency (CHIVA) raise important questions about how to manage these patients.

35) DUMITUR CASIAN: Metode contemporane de tratament al maladiei varicoase. Buletinul Academiei de Ştiinţe a Moldovei. Ştiinţe Medicale Numărul 1(33) / 2012 / Pag. 319-322

ABSTRACT

The long term results of classic surgical treatment of varicose veins are not completely correspond to requirements of physicians and wishes of patients. The modern methods of varicose veins treatment include endovenous thermal or chemical ablation and “conservative” interventions (CHIVA, ASVAL). According to the literature review, these methods provide the high efficacy of treatment and are associated with low rate of complications and recurrence of disease. Widening of spectrum of curative methods contribute to the individualization of varicose veins treatment.

36) ONIDA, DAVIES A.: CHIVA, ASVAL and related techniques--Concepts and evidence. Phlebology. 2015 Nov;30(2 Suppl):42-5.

Abstract

Chronic venous disease (CVD) is a highly prevalent condition with significant effects on patients' quality of life. Despite this, the underlying pathophysiology of venous disease still remains unclear. Two schools of thought exist, explaining the development and propagation of venous disease as an "ascending" and "descending" process, respectively. The descending theory, stating that CVD is secondary to proximal disease (e.g. saphenofemoral/saphenous incompetence), is the most widely accepted when planning treatment aiming to remove or destroy the junction or truncal veins. The ascending theory, describing the disease process as developing in the lower most part of the leg and propagating cranially, aims to re-route the venous circulation via minimally invasive interventions. Classically, superficial venous insufficiency has been treated with the removal of the incompetent trunk, via open surgery or, increasingly, with endovenous interventions. Minimally invasive treatment modalities aiming to preserve the saphenous trunk, such as CHIVA and ASVAL, may also play an important role in the treatment of the patient with varicose veins.

37) GIANESINI S, Occhionorelli S, Menegatti E, Zuolo M, Tessari M, Spath P, Ascanelli S, Zamboni P.: CHIVA strategy in chronic venous disease treatment: instructions for users. Phlebology. 2015 Apr;30(3):157-71

Abstract

Along the years, scientific clinical data have been collected concerning the possible saphenous flow restoration without any ablation and according with the CHIVA strategy. Moreover, in 2013 a Cochrane review highlighted the smaller recurrence risk following a CHIVA strategy rather than a saphenous stripping. Nevertheless, the saphenous sparing strategy surely remains a not-so-worldwide-spread and accepted therapeutic option, also because considered not so immediate and easy to perform. Aim of this paper is to provide an easily accessible guide to an everyday use of a saphenous sparing strategy for chronic venous disease, highlighting how even apparently too complicated reflux patterns classifications can be fastly and successfully managed and exploited for a hemodynamic correction.

38) P. ZAMBONI S.Gianesini: Surgical Technique for Deep Venous Reflux Suppression in Femoral Vein Duplication. (CHIVA in the deep system). EJVES Short Reports Volume 30, 2016, Pages 10-12

Background

Deep venous surgery is a challenging field with limited indications. Femoral vein duplication (FVD) is a frequent anatomical variant (55% prevalence). The aim was to describe a simple technique for managing deep venous reflux in FVD, when just one of the two segments exhibits deep venous reflux.

Methods

The technique consists of closing the refluxing femoral branch with a titanium clip. In this way abolition of reflux along the duplicated vessel is achieved, together with the restoration of femoral vein drainage.

Results

The technique is feasible and associated with improvement in limb haemodynamics.

Conclusions

Thanks to the high prevalence of FVD, the proposed technique provides an opportunity to treat a larger number of cases affected by primary or post-thrombotic deep venous reflux.

39) PUSKÁS A. et Al.:HAEMODYNAMIC MAPPING OF CHRONIC VENOUS INSUFFICIENCY:THE CONCEPT OF SHUNTS. VÉNÁK BETEGSÉGEI Érbetegségek, XXIII. évfolyam 4. szám, 2016/4.

ABSTRAC

It is well-known that the diagnostics of chronic venous insufficiency has been revolutionised by the introduction of duplex ultrasound. Refined manoeuvres and techniques contribute to the clarification of anatomical and haemodynamic details which are necessary for planning different steps in treatment. According to fluid dynamic science, the shunt is a pathway which diverts the flow into another channel system. Veno-venous shunts are venous tubes which diverts the venous blood from the physiological backflow through an insufficient veno-venous connection, which means a large extra amount of blood burdens these shunting venous channels. These shunts cause a flow and pressure overload, which results in high venous transmural pressure in this part of the venous system, which causes varicose dilatations, and symptoms and complaints for the

patient. According to the haemodynamic concept, these phenomena are the consequences of hight transmural pressure. This is because the extra amount of venous blood causes a faster speed of

low which changes the flow from laminar to turbulent. As a consequence of the turbulent flow, the pressure on the venous wall increases, which slowly dilates the lumen of the vein. With the usage of duplex ultrasound the following shunt elements can be identified:

(1) Escape point, where venous blood escapes from the deep to the superficial layers, which is opposite the physiological direction of flow.

(2) Shunt pathway, comprising all venous segments which make up the shunt and drains the superfluous venous blood burdening this system.

(3) Re-entry point drives the extra blood back to

the deep veins.

Four basic shunt types and some further subtypes can be distinguished by venous haemodynamics. Ultrasound mapping of them is a key point of therapeutic strategy. A good map is mandatory for a good treatment.

The basic shunt types are the following:

a) Closed shunt – venous blood starts its way at the escape point and goes to the re-entry point and again to the escape point, which means the recirculation of blood between these venous

segments.

b) Open deviating shunt – in these shunts blood never returns to the escape point. Valsalva manoeuvre is negative and venous blood is deviated by a branch of the saphenous stem.

c) Open bypass shunt – bypasses occlusions and obstacles. Occluded or stenosed or congenitally missing parts of the deep venous system mean a high resistance of flow is bypassed by a

superficial compensatory pathway. This type of shunt can have a vital significance in the circulation of the limb, therefore they must be preserved in any intervention.

d) Mixed shunt – is a combination of open bypass and active closed shunts.

The world of shunts is a great challenge for a practising phlebologist. There are many variations, so duplex ultrasound mapping is time-consuming at the beginning, but with some experience this becomes routine

40) FELIPE PUCINELLI FACCINI et Al.: CHIVA to spare the small and great saphenous veins after wrong-site surgery on a normal saphenous vein: a case report. J. vasc. bras. vol.18 Porto Alegre 2019 Epub Jan 14, 2019

ABSTRACT

CHIVA (Cure Conservatrice et Hemodynamique de l'Insufficience Veineuse en Ambulatoire) is a type of operation for varicose veins that avoids destroying the saphenous vein and collaterals. We report a case of CHIVA treatment of two saphenous veins to spare these veins. The patient previously had a normal great saphenous vein stripped in error in a wrong-site surgery, while two saphenous veins that did have reflux were not operated. The patient was symptomatic and we performed a CHIVA operation on the left great and right small saphenous veins. The postoperative period was uneventful and both aesthetic and clinical results were satisfactory. This case illustrates that saphenous-sparing procedures can play an important role in treatment of chronic venous insufficiency. Additionally, most safe surgery protocols do not adequately cover varicose veins operations. Routine use of duplex scanning by the surgical team could prevent problems related to the operation site.

ARTICLES CONCERNING SUBJECTES OF CHIVA PROCEDURE NOT IN TERMS OF RECURRENCES / CLINIC DATA BUT OF BIOCHEMICAL, HEMODYNAMIC PARAMETERS, THROMBOSES AND COMPLICATIONS

1) CAPPELLI M. et Al.: Considerazioni sul ruolo fisiopatologico delle perforanti nella varicosi essenziale, quale presupposto alla concezione terapeutica dell'intervento CHIVA. Ospedali d'Italia – Chirurgia nov–dic 1991, vol. XLIV n°6, pp. 425–438.

2) ZAMBONI P. et Al.: Alternative saphenous vein sparing surgery for future grafting . Panminerva Med 1995; 37:19 There are two possibilities: External valvuloplasty and CHIVA. Clinical, ecographic parameters, pressure values and R.L.R have been evaluated.

3) PINTOS T., SENIN E., RAMOS R., RODRIGUEZ E., MARTINEZ PEREZ M., Trombosis de safena interna post–CHIVA. Incidencia, factores condicionantes y repercusiones clınicas, Presented at the XLVII National Congress of the Spanish Society of Angiology and Vascular Surgery, Valladolid 2001.

Pintos et al. Studied 165 patients after CHIVA treatment and compared the presence of postoperative superficial venous thrombosis of the GSV in different groups. 101 patients (61%) were treated with CHIVA 1 or 2, 64 patients (39%) with a non-draining method consisting of CHIVA 1+2 (simultaneous closure of the saphenous-femoral junction and CHIVA 2 points with type 3 shunt) . The preoperative mean diameter of the GSV was 0.78 cm (0.28 to 1.70 cm). All patients carried out prophylaxis with low molecular weight heparin for 15 days after the operation, and wore class II compression stockings for 6 weeks. Controls were performed by ultrasonography at 1, 3 and 6 months after surgery.

The incidence of superficial venous thrombosis of GSV in the CHIVA 1 or 2 group was 9 patients (9%) while in the CHIVA group non-drained 25 patients (38%).

The difference between the incidence of superficial venous thrombosis in the two groups was statistically significant ($p < 0.001$).

This shows that the relatively high incidence of superficial venous thrombosis from the first publications on the CHIVA technique it has been negatively affected by the use of the CHIVA 1 + 2 procedure. If the CHIVA 1 + 2 (non-draining) procedure is not used, the incidence of superficial venous thrombosis decreases significantly. (Comment by Paolo Zamboni)

4) ZAMBONI P., CISNO C., MARCHETTI F., QUAGLIO D., MAZZA P., LIBONI A., Reflux elimination without any ablation or disconnection of the saphenous vein. A haemodynamic model for venous surgery, Eur. J. Vasc. Endovasc. Surg., 2001 Apr, 21(4): pp. 361–9.

The aim of this prospective study was to verify the possibility of reflux suppression in GSV without any crossectomy and / or stripping procedure. The authors studied about forty patients with primary chronic venous insufficiency of all clinical classes, with demonstrated Doppler incompetence of both the saphenous-femoral junction and the large trunk of the GSV, with the presence of a re-entry perforator placed along a saphenous tributary. The air plethysmography and duplex data were both

collected before the intervention and at 1 and 6 months after. The duplex investigation showed the presence of an antegrade flow and the disappearance of reflux in the GSV in 100% of cases after 1 and in 85% at 6 months (Comment by Paolo Zamboni)

5) FRANCESCHI C.: CHIVA effectiveness score: the correct one is below. Eur J Vasc Endovasc Surg. 2012 Sep;44(3):351; author reply 352.

Comment on: Validation of a new duplex derived haemodynamic effectiveness score, the saphenous treatment score, in quantifying varicose vein treatments. [Eur J Vasc Endovasc Surg. 2012]

6) MALDONADO-FERNANDEZ et Al.: Postoperative complications of CHIVA technique for the treatment of chronic venous failure. (2010) Angiologia, 62 (3), pp. 91-96.

Abstract

Introduction. The most commonly used technique for varicose vein surgery is saphenectomy, although haemodynamic surgery (CHIVA) has been becoming increasing popular in the last decade in our country, probably, due to its good postoperative recovery and fewer complications. Objective. To describe and quantify postoperative complications of CHIVA technique in our experience as well as that reported in the literature. Methods. Retrospective descriptive study of 269 extremities operated on by our group and analysis of 2,793 audited extremities described in the literature. Results. The main complications in our patients were: 17 cases in 269 extremities (6.33 %), distributed as follows: 11 symptomatic saphenous vein thrombosis, two temporary paresthesias, two groin haematomas, one wound infection, and one headache after spinal anaesthesia. Complications reported in the literature: 208 cases in 2,793 extremities (7.44 %), distributed as follows: 82 symptomatic saphenous vein thrombosis, 70 neuritis and paresthesias, 25 minor skin infections, 9 haematomas, 7 groin infections, 6 lymphatic groin leakages, 4 deep vein thrombosis, and one groin haemorrhage. There is no mortality or major complications associated with this procedure. Conclusions. CHIVA surgical approach to chronic venous insufficiency is accompanied by a rapid recovery and active life with a 7 % complication rate, which are mostly benign and do not hinder recovery. Symptomatic saphenous vein thrombosis is the most common complication after surgery for varicose veins using this technique.

7) MENDOZA E., BERGER V., ZOLLMANN C., BOMHOFF M., AMSLER F., Calibre reduction of great saphenous vein and common femoral vein after CHIVA Phlebologie, 2011, 40(2): pp. 73– 78.

8) MENDOZA E., Diameter reduction of the great saphenous vein and the common femoralvein after CHIVA Long–term results, Phlebologie, 2013, 42: pp. 65–69.

The diameters of the GSV and the common femoral vein (CFV) reflect the hemodynamic overload of venous disease. This study was designed to answer Prof. Hach's question in 2002, who asked if the femoral vein was overloaded after CHIVA.

Hach hypothesized that tributary blood (R3) that flowed through the saphenous vein (R2) into the deep veins (R1) usually through the saphenous-femoral junction could overload the femoral vein. Usually this blood would never circulate through the femoral vein, while after crossotomy in the CHIVA treatment, it will flow retrograde and drain through a perforating vein of the thigh or calf. Therefore, after surgical treatment, the femoral vein and the common femoral vein (CFV) distal to the saphenous-femoral junction would be overloaded by the blood returning from the perforating

vein. The aim of this study was therefore to investigate the long-term effects of CHIVA treatment on CFV diameters. Patients underwent interventions aimed at maintaining drainage (CHIVA 2 in one or two phases, depending on the hemodynamic pattern).

In an initial phase, the evolution of the GSV diameters at the level of the proximal thigh and the CFV diameter in an upright position was measured (Mendoza 2011).

383 patients with 470 treated legs (84.4%) repeated a duplex examination between 8 and 25 weeks after surgery. The GSV and CFV diameters were compared before and after the surgery. The GSV diameter went from 6.1 mm before surgery to 4.5 mm after surgery in the female group and from 6.8 mm to 5.1 mm in the male group. The diameter of the common femoral vein went from 14.0 mm before surgery to 13.7 mm after surgery in the female group and from 16.5 mm to 16.1 mm after surgery in the male group, all of these

results showed a statistically significant difference.

In a second study (Mendoza 2013), the long-term effect on diameter was checked after 5 years in 43 patients included in the first study.

In addition, the clinical class (CEAP) and filling time were

compared with preoperative values and after 8 weeks.

The diameter of the CFV and the diameter of the GSV decreased significantly even after 5 years, the CEAP clinical class decreased from 2.77 _ 0.81 before surgery to 1.72 _ 1.10 after 5 years (p = 0.007). The venous filling time measured with photoplethysmography was significantly longer from 15.24 _ 6.18s to 21.61 _ 9.2s after 5 years from the intervention (p = 0.022).

The authors therefore concluded that not only in the short term, but

also as a long-term result, the CHIVA intervention reduced both the CFV and GSV diameter and the C of the CEAP clinical classification and improved the venous filling time. (Comment by Paolo Zamboni)

9) MENDOZA E.: Crossectomy of the great saphenous vein with the CHIVA method

(2004) Vasomed, 16 (2), pp. 46-48.

Abstract

During the last years a lot of new techniques to treat varicose Veins were introduced in Germany. They question established concepts, specially the treatment of refluxive great saphenous vein with safeno-femoral incompetence. CHIVA treats the groin-region in a different way than the classical accepted crossectomy and stripping of saphenous vein. The saphenous vein and healthy side branches of the crosse are left in situ, just double ligation of the safeno-femoral junction is performed. This technique is explained with fotos and pictures: Disection of safeno-femoral junction, double ligation of safeno-femoral junction, once just at the level of femoral vein and twice just below the side branches.

10) DELFRATE R., BRICCHI M., FRANCESCHI C., GOLDONI M., Multiple ligation of the proximal greater saphenous vein in the CHIVA treatment of primary varicose veins,

Veins and Lymphatics, 2014, 3: pp. 19–22, https://www.pagepressjournals.org index.php/vl/article/view/vl.2014.1919.

The aim of this study was to determine if a crossotomy was needed or if a ligation could be performed for safety reasons on patients operated on an outpatient basis.

199 legs were followed-up after the saphenous-femoral interruption in the CHIVA context with three different techniques.

Common to all techniques was to place a titanium clip (10 mm long and 1 mm thick) flush with the femoral vein in order to prevent the presence of a residual saphenous stump.

- First group: (N = 61) Crossotomy (with interruption of the saphenous-femoral junction, 29 months follow-up);

- second group: (N = 82) triple ligature of the saphenous vein (TSFL performed with a suture covered with non-absorbable thread (14 months follow-up);

- third group: (N = 56) Triple polypropylene ligature (TPL; 12 months follow-up).

In the first two groups the percentage of new refluxes to the Valsalva manoeuvre at SFJ level was 6.1%, in the second (which however had a shorter follow-up) the presence of reflux on the SFJ at Valsalva was 4.9% , without statistically significant differences. In the third group, a percentage of channelization of 37.5% was detected after one year, the difference between group 3 and 1, as well as with group 2 was highly significant with p <0.001 (Comment by Paolo Zamboni)

11) MENDOZA E, AMSLER F., CHIVA with endoluminal procedures: LASER versus VNUS – treatment of the saphenofemoral junction, Phlebologie, 2017, 46: pp. 5–12.

From its description, the CHIVA strategy has always been performed with open surgical techniques.

After the introduction of endoluminal thermal techniques, this first approach aimed to compare LASER and / or Radio Frequency in the obliteration of the saphenous-femoral junction in the CHIVA context.

104 patients were studied before and at 3 and 6 months after GSV treatment with CHIVA strategy using endoluminal thermal techniques to close the inguinal segment (75 patients with VNUS [Closure – Fast], 29 LASER [1470nm, Radial Intros]).

A significant reduction of the GSV diameters at the level of the proximal thigh and of the CFV was detected, as well as an improvement in the clinical results (Table 10.17), the latter comparable to those achieved after surgical lacrossectomy.

The author concludes that it is suitable to apply endoluminal thermal techniques in the context of the CHIVA strategy. (Comment by Paolo Zamboni)

12) PASSARIELLO F. et Al.: The office based CHIVA

Journal of Vascular Diagnostics 26 September 2013 Volume 2013:1 Pages 13—20

Abstract: The cure Conservatrice Hémodynamique de l'Insuffisance Veineuse en Ambulatoire (CHIVA) can be office based (OB). The OB-CHIVA protocol is aimed at transferring CHIVA procedures to specialists rooms. The protocol will check the feasibility of OB-CHIVA, data pertaining to recurrence, and will offer the opportunity to study saphenous femoral junction (SFJ) stump evolution, the role of the washing vessels and the arch recanalization rate, and gather new data about the effect of the length of the treated saphenous vein. A simplified diagnostic procedure will allow an essential ultrasound examination of the venous net while a schematic and easily readable

algorithm guides therapeutic choices. The Riobamba draining crossotomy (RDC) tactic is composed of a set of OB procedures. While some of these procedures are, at the moment, only proposals, others are already applied. Devices generally used in ablative procedures such as Light Amplification by Stimulated Emission of Radiation (LASER), radio frequency, steam, and mechanical devices are used in this context to serve to conservative interventions for CHIVA. New techniques have also been proposed for devalvulation and tributary disconnection. Detailed follow-up is necessary in order to determine the effects of therapy and possible disease evolution. Finally, information is added about the informed consent and the ethical considerations of OB-CHIVA research.

13) GIANESINI S.,MENEGATTI E., ZUOLO M., TESSARI M., ASCANELLI S., OCCHIONORELLI S., ZAMBONI P.: Short endovenous laser ablation of the great saphenous vein in a modified CHIVA strategy, Veins and Lymphatics, 2013, volume 2: e21, https://www.pagepressjournals.org/index.php/vl/article/view/vl.2013.e21.

14) GIANESINI S. et Al. : Mini-invasive high-tie by clip apposition versus crossectomy by ligature: Long-term outcomes and review of the available therapeutic options

Phlebology OnlineFirst, published on May 9, 2016 as doi:10.1177/0268355516648066

Abstract

Objective: The aim of the present study is to compare a mini-invasive (smaller than 2-cm incision) sapheno-femoral

high-tie by clip apposition (HT group) with a traditional high-ligation by ligature (HL group).

Methods: One hundred fifty chronic venous disease patients were included in group HT and compared with 150 cases constituting the group HL. The main outcome was the sonographic detection of saphenous trunk recurrences. Procedural pain, esthetic satisfaction, and disease specific quality of life were assessed.

Results: At 4.5_2.4 years follow-up, 8 cases (5.3%) of Great Saphenous Vein reflux reappearance were reported in group HT vs. 19 cases (12.6%) (odds ratio: 2.6; 95% confidence interval: 1.1–6.1; P.0.04) of group HL. Esthetic satisfaction was scored as high and very high in group HT and HL, respectively (P<.0001).

Conclusions: Proper high-ligation technique provides satisfying outcomes both in terms of recurrence rate and patient

esthetic satisfaction. The different outcomes obtained by the two groups encourage further investigations regarding recurrence pathogenesis.

15) CAPPELLI M. et Al.: Ligation of the saphenofemoral junction tributaries as risk factor for groin recurrence. J Vasc Surg Venous Lymphat Disord. 2018 Mar;6(2):224-229. doi: 10.1016/j.jvsv.2017.09.005. Epub 2017 Dec 28.

Abstract

OBJECTIVE:

The aim of this study was to compare the recurrence rate after high ties performed with or without sparing of the saphenofemoral junction tributaries.

METHODS:

There were 867 lower extremities enrolled. All patients underwent a high tie with (group A) or without (group B) ligation of all the junctional tributaries for a great saphenous vein reflux (C2-5EpAsPr). A duplex ultrasound examination detected recurrences.

RESULTS:

Median follow-up was 5 years (interquartile range, 3-8 years). Group A had a higher recurrence rate than group B (odds ratio, 7.52; P < .001). Group A recurrences (7.4%), compared with group B (1.1%), presented with a more frequent direct stump reconnection (3.7% vs 0.2%; P < .001) or newly developed pelvic shunts (3% vs 0.5%; P < .001). No significant difference was reported between the two groups in newly incompetent perforating veins.

CONCLUSIONS:

Ligation of the junctional tributaries is associated with a higher recurrence risk. Further investigations are needed to determine the hemodynamic role of each single junctional tributary.

16) TISATO V. et Al. : Modulation of Circulating Cytokine-Chemokine Profile in Patients Affected by Chronic Venous Insufficiency Undergoing Surgical Hemodynamic Correction. Journal of Immunology Research Volume 2014, Article ID 473765, 10 pages http://dx.doi.org/10.1155/2014/473765

ABSTRACT

The expression of proinflammatory cytokines/chemokines has been reported in in vitro/ex vivo settings of chronic venous insufficiency (CVI), but the identification of circulating mediators that might be associated with altered hemodynamic forces or might represent innovative biomarkers is still missing. In this study, the circulating levels of 31 cytokines/chemokines

involved in inflammatory/angiogenic processes were analysed in (i) CVI patients at baseline before surgical hemody namic correction, (ii) healthy subjects, and (iii) CVI patients after surgery. In a subgroup of CVI patients, in whom the baseline levels of cytokines/chemokines were analyzed in paired blood samples obtained from varicose vein and forearm vein, EGF, PDGF, and RANTES were increased at the varicose vein site as compared to the general circulation. Moreover, while at baseline, CVI patients showed increased levels of 14 cytokines/chemokines as compared to healthy subjects, 6 months after surgery, 11 cytokines/chemokines levels were significantly reduced in the treated CVI patients as compared to the CVI patients before surgery.

Of note, a patient who exhibited recurrence of the disease 6 months after surgery, showed higher levels of EGF, PDGF, and RANTES

compared to nonrecurrent patients, highlighting the potential role of the EGF/PDGF/RANTES triad as sensitive biomarkers in the

context of CVI.

17) GIANESINI S., MENEGATTI E., ZUOLO M., TESSARI M., SPATH P., ASCANELLI S., OCCHIONORELLI

S., ZAMBONI P., Laser–assisted strategy for reflux abolition in a modified CHIVA approach, Veins and Lymphatics, 2015, 4: 5246 doi:10.4081/vl.2015.524, https://www.pagepressjournals.org/index.php/vl/article/view/5246.

18) ZAMBONI P. et Al.:Oscillatory flow suppression improves inflammation in chronic venous disease journal of surgical research _ september 2016 (205) 238-245

a b s t r a c t

Background: To assess if suppression of the oscillatory component of reflux may improve the inflammatory phenotype in chronic venous disease (CVD).

Materials and methods: From 193 CVD patients, we selected 54 (13 males, 41 females, CEAP C2-4EpAsPr) for a blinded, case-control prospective investigation. All of them underwent echo-color-Doppler assessment of reflux parameters. In the same patients a blood systemic assessment of 19 inflammatory cytokines was obtained. Follow-up lasted 6 months. The control group (C) was constituted by 21 homogenous CVD patients, unselected and not operated.

Results: Forty-one of 54 patients were excluded from post-operative evaluation in consequence of reported new other inflammatory episodes. Twenty-three (23) completed

the follow up, showing the suppression of the oscillatory component of venous reflux; 4 of the 19 cytokines decreased significantly after the procedure: Tumor Necrosis Factor-a (TNFa), Granulocyte Colony Stimulating Factor (G-CSF), Interferon gamma-induced Protein

10 (IP-10), Interleukin-15 (IL-15). Particularly, TNFa and IP-10 even returned inside a physiological range: 5.3 _ 2.7 to 4.2 _ 2.2 pg/mL ($P < 0.003$) and from 303.7 _ 168.4 to 254.0 _ 151.6 pg/mL ($P < 0.024$), respectively. Both cytokines showed a weak but significant

correlation with parameters of oscillatory flow correction. Finally, three cytokines implicated in repair and remodeling of tissue, Epidermal Growth Factor, Monocyte Chemoattractant Protein-1 and Platelet Derived Growth Factor-BB (PDGF-BB), significantly

increased. Our findings are further reinforced by the significant changes of the same cytokines when compared to C group.

Conclusions: The surgical suppression of the oscillatory component of reflux modulates the inflammatory phenotype, suggesting a pivotal role of flow among factors concurring to inflammation in CVD.

19) DELFRATE R.: Thanks to the CHIVA strategy may the histoarchitecture of great saphenous vein-sparing, make it suitable as graft for bypasses? Veins and Lymphatics 2019; volume 8:8227

Post-CHIVA regression of anatomical-pathological alterations of the incompetent saphenous trunk

20) ZAMBONI P. et Al.: Alternative saphenous vein sparing surgery for future grafting.

Panminerva Med. 1995 Dec;37(4):190-7.

Abstract

OBJECTIVE:

Evaluation of long saphenous vein sparing surgical procedures alternative to high ligation and distal stab avulsion, in terms of effectiveness and suitability for eventual by-pass surgery.

EXPERIMENTAL DESIGN:

Prospective evaluation of 125 operations for primary varicose veins, 52 external valve-plasties of the sapheno-femoral junction (EV-SFJ) (42 performed using the hand sewing technique and 10 using the Veno-cuff device), mean follow-up 45 months, and 73 hemodynamic correction of varicose veins (French acronyms: CHIVA), mean follow-up 30 months.

SETTING:

Department of General Surgery, University of Ferrara. Institutional practice, one-day surgery.

PATIENTS:

Patients were selected using clinical, Doppler cw, and duplex scanning evaluations. Patients with early varices due to sapheno-femoral reflux with duplex scanning evidence of mobile valve leaflets underwent EV-SFJ. The other patients were operated on using the hemodynamic correction technique. Both groups underwent preoperative ambulatory venous pressure (AVP) and light reflection rheography-refilling time (LRR-RT) measurements.

INTERVENTIONS:

EV-SFJ restores valve function correcting vein wall dilitation by applying an external prosthesis. CHIVA consists of selected ligatures of the superficial veins that allow superficial blood aspiration in the deep veins through the perforators.

MEASURES:

The outcome was evaluated with clinical and ultrasonographic examinations, AVP and LRR-RT measurements.

RESULTS:

Long saphenous vein patency registered after EV-SFJ and CHIVA was 94.2% and 90.4%, respectively. Both treatments preserve the drainage function in the saphenous system. Varicose veins recurrence percentage rate was 9.6% and 10.9%, respectively.

CONCLUSIONS:

Following the proposed selection criteria, these two alternative procedures seem to be more effective in varices treatment than high ligation and have the advantage of preserving saphenous veins suitable for eventual by-pass surgery.

21) E. Mendoza, M. Cappelli : Sclerotherapy technique in CHIVA strategy Phlebologie 2017; 46(02): 66-74

Summary

CHIVA has been developed by Claude France-schi in the 1980-es and was first published in 1988 (1). CHIVA is a strategy to treat venous insufficiency keeping the drainage through the saphenous veins and re-entry perforating veins. Venous recirculations are stratified into shunt types depending on the origin of the reflux and the distal re-entry to the deep veins (2). Originally the method was described with surgical interventions: Flush ligation of the saphenous veins from the deep veins (crossotomy), flush ligation of the tributaries at the saphenous trunk, disconnection of the refluxive saphenous trunk distal to a reentryperforating vein, disconnection of a refluxive perforating vein, if it is the proximal insufficiency point. Techniques in phlebology have developed: in the field of the vein ablating strategies (originally stripping) new possibilities are currently used, as foaming under ultrasound-guidance of the complete saphenous vein, and endoluminal heat techniques, as well as glue. In the same way, the CHIVA strategy has incorporated new techniques. This article highlights the application of foam sclero-therapy in combination with the CHIVA strategy. Sometimes in CHIVA it is useful to start with one step and complete the treatments months later, if a vein did not reduce its caliper or revert its flow after the first step.

22) FERRACANI E.: A Change of a Paradigm Under the Scope of a Cardiovascular Surgeon. Remodeling of the Great Saphenous Vein Instead of Ablation for Preservation of the Patient Anatomical Capital

Cardiology December 30, 2019 ecronicon.com open access.

Abstract

The presented work is an ongoing study by using a combined approach of sparing surgical techniques plus LASER 1470 nm for sparing the Great saphenous vein (GSV) at early hemodynamics stages.

Peak reflux Volume lower than 30cc by second, Total Reflux Volume (TRV) between 10 and 100 cc/s using low LASER LEED and no tumescence anesthesia for preservation of patient anatomical capital and the actual recovery value of this conduit for a future arterial bypass.

23) MENDOZA E. : Does the suture material influence the outcome after high ligation of great saphenous vein?

Vasa (2020), 49 (2), 153–155 https://doi.org/10.1024/0301-1526/a000833

It is a review of the literature concluding that non-resorbable threads give less angiogenetic phenomena.

ARTICLES CONCERNING THE RESULTS OF CHIVA PROCEDURE IN TERM OF RECURRENCES / CLINIC DATA WITHOUT COMPARISON WITH OTHER METHODS

1) FICHELLE JM, Carbone P, Franceschi C.: Results of ambulatory and hemodynamic treatment of venous insufficiency (CHIVA cure) J Mal Vasc. 1992;17(3):224-8.

Abstract

From January 1987 to December 1988, 100 conservative and hemodynamic treatments of superficial venous insufficiency in great saphenous vein territory, have been done on 86 patients. They were 32 men, whose mean age was 53.7 years, and 54 women, whose mean age was 44.5 years. Indication for surgery was mainly functional in 28 cases, esthetic in 26 cases, both in 25 cases and trophic problems in 21 cases. Ligation of the sapheno-femoral junction has been done in 91 cases (62 clips, 9 clips and ligations, 11 ligations, 9 sutures). Distal interruption has been done above knee in 24 cases, below knee in 50 cases, and both in 16 cases. Early postoperative complications have been one septic collection of the groin, one hematoma of the groin, one durable contusion of the saphenous nerve, and 21 superficial venous thrombosis. There were six thrombosis of excluded branches, seven subtotal thrombosis of the saphenous and height partial thrombosis of the saphenous vein. Subtotal thrombosis of the saphenous vein were due either to a mistake in position of distal ligation in three cases, either to a too large saphenous vein in four cases. Five out of height partial thrombosis occurred on saphenous veins larger than ten millimeters. Follow up was obtained, in 1990, so that all patients had at least one year of follow-up. Seven patients have been lost for follow-up. Three patients had recurrence because of failure of the clip. An additional procedure was necessary in 30 patients. Functional results were correct in 89% of patients, and esthetical results in 68% of patients.

2) BAILLY M.: Resultats de la cure Chiva In techniqueset stratégie en chirurgie vasculaire. Jubilé de J.M. Cormier. Edition A.E.R.C.Paris 1992: 255-71.

3) HUGENTOBLER J.P., BLANCHMAISON P.: CHIVA cure. Etude de 96 patients opres de juin1988 a juin 1990 J. Mal. Vasc., 1992, 17: pp. 218–23.

4) QUINTANA F. et Al.:The CHIVA cure of varices of the lower extremities. La Cure Conservatrice et Hemodynamique de l'Insuffisance Veineuse en Ambulatoire Angiologia. 1993 Mar-Apr;45(2):64, 66-7.

Abstract

Presentation of the characteristics of this technique described by the French physician C. Franceschi, in 1988. Our Department began to apply this method on may 1991 and we are the first team in Spain to carry out and systematize this cure. Up to date, **85 patients have been treated with a residual vein percentage of 18%**. Morbidity is low and slight. There is no mortality. This method is considered interesting as it does not require hospitalization, conserves the vein capital of the patient, and has low labour and health care costs.

5) ZAMBONI P.: When CHIVA treatment could be video guided. Dermatol Surg. 1995 Jul;21(7):621-5.

Abstract

BACKGROUND:

Hemodynamic correction (CHIVA) is a conservative, ambulatory, and controversial varicose vein treatment. It consists of selected ligatures of the superficial venous system decided by means of preoperative duplex mapping.

OBJECTIVE:

Prospective evaluation of 80 patients, operated on according to the CHIVA technique described by Claude Franceschi. Mean follow-up length was 30 months.

METHODS:

Fifty-five consecutive patients were operated on after clinical, ultrasonographic, ambulatory venous pressure and light reflection rheography evaluations. After a 3-year follow-up, another 25 consecutive patients were selected applying some exclusion criteria that emerged in the first part of the study. This second series was operated on by means of intraoperative angioscopy. The same preoperative evaluations have been used to study the outcome in all patients.

RESULTS:

CHIVA failed in the short saphenous vein territory varices and when the long saphenous vein and the insufficient perforating veins had a preoperative diameter greater than 10 and 4 mm, respectively. The procedure showed a long saphenous vein patency of 90.4% and registered a total recurrence rate of 18.7%.

CONCLUSIONS:

CHIVA seems to be a more effective varicose vein treatment than high ligation and distal stab avulsion. It also preserves a higher rate of long saphenous veins, suitable for bypass surgery.

Comment in

Video-guided CHIVA treatment. [Dermatol Surg. 1995]

WEISS RA : Video-guided CHIVA treatment.

Dermatol Surg. 1995 Jul;21(7):626.

Comment on: When CHIVA treatment could be video guided. [Dermatol Surg. 1995

6) ZAMBONI P. et AL.: Angiovideo-assisted hemodynamic correction of varicose veins.

Int Angiol. 1995 Jun;14(2):202-8.

Abstract

OBJECTIVE:

Evaluation of the feasibility and utility of angioscopy in the hemodynamic correction (French acronyms is CHIVA) of primary varicose veins disease.

EXPERIMENTAL DESIGN:

Prospective evaluation of 25 patients, undergoing hemodynamic correction of primary varicose disease with intraoperative videoangioscopic guide. Patients have been selected according to criteria emerged from a prospective study that we had previously conducted. Follow-up lasted 1 year (range 8-18 months).

SETTING:

Department of Surgery, University of Ferrara, Italy. Institutional practice. One-day surgery.

PATIENTS:

Their selection has been carried out in our Vascular Laboratory. The adopted clinical criteria of selection were: Primary varicose disease of the long saphenous vein territory, no previous thrombophlebitis and/or sclerotherapy. Doppler cw and Duplex criteria followed were: competent deep venous system, long saphenous vein diameter minor than 10 mm and incompetent perforating veins diameter minor than 4 mm.

INTERVENTIONS:

25 hemodynamic corrections according to the CHIVA method described by Franceschi. An angioscope, introduced through a distal collateral of the long saphenous vein, permitted the precise interruption of the venous-venous shunts and of the superficial venous system, just below the perforators chosen as re-entry points in the deep venous system.

MEASURES:

Clinical: varices and symptomatology reduction. Duplex and Doppler cw: detection of the superficial blood flow re-entry, in the deep venous system, through the perforators and identification of recurrences or new refluxes. Pre and postoperative Ambulatory Venous pressure and Refilling Time have also been measured.

RESULTS:

In 20 patients symptoms and varices relief were recorded (80%), in 5 patients varices reduction was observed only during walking (20%). In 2 of these latter patients there was no re-entry through the perforators, with a recurrent sapheno-femoral reflux in 1 of them. Early complications recorded were: 2 long saphenous vein thrombosis (8%); 7 ecchimosis (28%) when heparine/saline solution had been used for angioscopic clearance.

CONCLUSIONS:

Intraoperative angioscopy is feasible and useful when the hemodynamic situation is complex and the Duplex map is difficult to be interpreted by the surgeon. In this series the second look percentage rate has been minor compared to the percentage rates published so far by other authors.

7) BAHNINI A, Bailly M, Chiche L, Franceschi C.: Ambulatory conservative hemodynamic correction of venous insufficiency. Technique, results.

Ann Chir. 1997;51(7):749-60.

Abstract

Ambulatory conservative haemodynamic correction of venous insufficiency (CHIVA) is a surgical treatment of superficial venous insufficiency designed to correct the pathological haemodynamic effects of superficial venous insufficiency apparent on standing. Surgical treatment is based on precise preoperative anatomical and haemodynamic mapping performed by duplex ultrasound, providing preoperative ultrasound-guided marking. Surgical treatment consists of dividing the hydrostatic pressure column and disconnecting venovenous shunts by ligation-section of the superficial venous network at precise points determined by the preoperative ultrasound-guided

marking. This strategy should achieve a superficial venous circuit draining perfectly into the competent deep venous network. The operation is performed under local anaesthesia as an outpatient procedure and allows immediate resumption of walking, which promotes a good result due to activation of the calf muscle pump. The results of the technique are very good provided a reliable preoperative ultrasound-guided marking and a precise surgical procedure are performed. Failures are due to poor haemodynamic assessment or inappropriate surgical procedure.

8) CAPPELLI M. et Al.: I risultati della cura CHIVA.

Osp Ital Chir 1998; 4: 615-8.

9) ZAMBONI P., MARCELLINO M.G., CAPPELLI M., FEO C.V., BRESADOLA V., VASQUEZ G., LIBONI A., Saphenous vein sparing surgery: principles, techniques and results, J.

J. Cardiovasc. Surg., Torino 1998 Apr, 39(2): pp. 151–62.

ABSTRACT

Follow–up a 4 anni dopo CHIVA (Zamboni 1998). Sono stati studiati 357 pazienti, operati utilizzando la metodica CHIVA e monitorati per 4 anni, non era incluso alcun gruppo di controllo. Nel 94% dei pazienti, alla fine dello studio la vena grande safena (GSV) presentava un flusso di drenaggio per tutta la sua lunghezza (ovvero non si è rilevata la presenza di trombosi venose

superficiali). L'11%dei pazienti ha presentato una recidiva. La reografia a luce riflessa ha mostrato miglioramenti significativi subito dopo l'operazione e dopo 6 mesi rispetto ai valori preoperatori. (PAOLO ZAMBONI)

10) CAPPELLI M. et Al. "Ambulatory conservative hemodynamic management of varicose veins: critical analysis of results at 3 years"

ANNALS OF VASCULAR SURGERY 2000 Vol 14 n°4 pag 376-384

Abstract

This report describes the results of our 3-year experience using ambulatory conservative hemodynamic management (ACHM) for lower extremity venous insufficiency involving the greater saphenous vein (GSV), with specific analysis of recurrence due to neoformation of vessels. We performed 289 ACHM procedures in 259 consecutive patients with GSV-related varicose veins. Follow-up clinical examination and Doppler ultrasound imaging was carried out at 3, 6, 12, 24, and 36 months in all cases to assess formation of neovessels supplied either by the superficial (A) or deep (B) venous system. Our data showed that ACHM achieved excellent improvement, with complete disappearance of varicose veins in 41.2% of cases, good improvement in 43%, fair improvement in 14.1%, and no improvement in 1.7%. The only predictor of outcome was the quality of drainage from the GSV vein. Poor drainage leads to neoformation of vessels supplied by the superficial (A) venous system. In about 50% of cases, drainage appeared spontaneously within 1 year, with a subsequent reduction in formation of neovessels. Neoformation of vessels supplied by the deep (B) venous system (10%) was independent of the quality drainage. This finding suggests that formation of these neovesseis is unrelated to the surgical method used to treat varicose veins. In patients with poor drainage of the saphenous network, neoformation of vessels supplied by the superficial (A) venous system is predictable with regard to both topography and delay. ACHM is a good tool for treatment

of varicose veins, as reliable statistical prediction of mid-term results is possible using available models.

The article focouses on the problem of draining and non-draining systems, and therefore the difference in terms of recurrences and saphenous thrombosis in the two groups

11) ESCRIBANO J.M., JUAN J., BOFILL R., MAESO J., RODRÍGUEZ–MORI A., MATAS M., Durability of reflux–elimination by a minimal invasive CHIVA procedure on patients with varicose veins. A 3–year prospective case study, Eur. J. Vasc. Endovasc. Surg., 2003, 25: pp. 159–63.

José María Escribano and the team of Barcelona Vall d'Hebrón University have published a study on the results of CHIVA in 2 steps in type 3 shunt cases. 58 patients were analyzed during 3 years after performing the first step of "CHIVA 2" in Type 3 shunts with a saphenous tributary below the knee.

The GSV diameter decreased significantly after surgery, although 51 of the patients had the reappearance of reflux after 6 months and 53 after 3 years. In all patients, the presence of a re-entry perforator was found, i.e. the transformation of the type 3 shunt into type 1 shunt.

46 patients underwent a disconnection of the saphenous-femoral junction over the 3 years of the study (crossotomy). The conclusion reported by this study is that the percentage of recurrences after the first half of CHIVA in Type 3 shunts is high. (Comment by Paolo Zamboni)

12) ZAMBONI P, ESCRIBANO JM.: Regarding 'Reflux Elimination Without any Ablation or Disconnection of the Saphenous Vein. A Haemodynamic Model for Venous Surgery' and 'Durability of Reflux-elimination by a Minimal Invasive CHIVA Procedure on Patients with Varicose Veins. A 3-year Prospective Case Study'. Eur J Vasc Endovasc Surg. 2004 Nov;28(5):567.

13) ESTEBAN-GRACIA C. et Al.: Application of the CHIVA strategy. A prospective study at one year Angiologia 2004, 56 (3), pp. 227-235.

Abstract

Introduction. There is a tendency for surgery to become less and less invasive. The CHIVA strategy could be included within the concept of minimally invasive surgery. Aims. Our aim was to perform a prospective evaluation of the clinical results at one year after applying the CHIVA strategy in the treatment of primary varicose veins. Patients and methods. A one-year follow-up of 225 patients (147 females, 78 males). Clinically, 195 of them were in stage 2 (CEAP). A Doppler ultrasound recording was conducted before surgery. Later, at one month and one year, patients were evaluated clinically and the results were classified in four categories. Patients were again submitted to a new Doppler ultrasound recording at one year. The type of strategy employed was in a single intervention in 97.8% of the cases. Results. At one year, the objective and subjective clinical assessment were good in 87.6 and 90.7% of cases, respectively. The mean diameter of the internal saphenous vein changed from 6.4 to 4.0 mm (t test; p = 0.001). Significant differences were observed between the objective assessment at one month and at one year (p = 0.001), as well as in the subjective assessment (p = 0.001), since a third of the patients with a poor evaluation at one month presented a good one at one year. Conclusions. The CHIVA strategy shows good results at one year in our series.

The significant reduction of the diameter of the saphenous vein indicates that the haemodynamic component is important in the pathophysiology of varicose veins.

14) LINARES-RUIZ, P., Bonell-Pascual, A., Llort-Pont, C., Romera, A., Lapiedra-Mur, O. : Mid-term results of applying the CHIVA strategy to the external saphenous vein. Angiologia 2004 , 56 (5), pp. 481-490.

Abstract

Introduction. The anatomical complexity and widely varying distribution of the external saphenous vein (ESV) means that surgical treatment is associated to high rates of relapse and residual varicose veins. Aim. To evaluate the mid-term results of using the CHIVA cure strategy on ESV varicose veins. Patients and methods. Between February 1996 and December 2002 we performed 142 CHIVA interventions to treat ESV. A random sample of 80 interventions was taken and data collected about their factors related to chronic venous insufficiency, pre-operative clinical features (CEAP), primary shunt and the surgical strategy applied. Doppler ultrasound was used to assess competence, patency, flow direction, diameter and neoaortic arch of the ESV in the post-operative period, visible relapses and symptoms. In addition, the relationships between the following parameters were also analysed: Doppler ultrasound recordings, surgical strategy, relapses and symptoms. Results. Competence of the deep vein system (DVS) and ESV patency were found to be > 95% (four ESV thromboses). Haemodynamically favourable situations: 66%. Mean diameter of the ESV: 3.5 cm; neoaortic arch: six patients (7.5%). Clinical features of the post-operative period: 59 asymptomatic patients (73.8%), 16 with a clinical improvement (20%) and five patients with no improvement in their symptoms (6%). Visible relapses: 15 cases, 12 of which were not important enough to require reintervention. There were no cases of DVS thromboses or peripheral neuropathy. There was a statistically significant correlation between the presence of anterograde flow and the absence of relapses and symptoms in the post-operative period, as well as between symptoms and relapses with higher absolute ESV diameters and neoaortic arch. There was a correlation, although statistically non-significant, between relapses and symptoms in the postoperative period and surgical strategy. Conclusion. The best results (i.e. less thromboses and relapses): CHIVA 1 + 2 in the case of ESV.

15) ZAMBONI P., GIANESINI S.,MENEGATTI E., TACCONI G., PALAZZO A., LIBONI A., Great saphenous varicose vein surgery without saphenofemoral junction disconnection, Br. J. Surg., 2010 Jun, 97(6): pp. 820–5.

This case-control study was designed to determine whether preoperative duplex imaging could predict the outcome of varicose vein surgery without disconnecting the saphenous-femoral junction (SFJ).

The duplex protocol included a reflux elimination test (RET-test) and the evaluation of the competence of the terminal valve of the femoral vein. Patients with negative reflux elimination tests were therefore excluded.

One hundred patients with chronic venous insufficiency who had a positive RET test and an incompetent terminal valve were compared with 100 patients, homogeneous by age, sex, CEAP clinical class, duration of disease, who had a positive RET test but a valve competent terminal. All patients underwent proximal ligation of incompetent tributaries from the saphenous trunk without disconnection of the saphenous-femoral junction. Clinical and duplex follow-up lasted for 3 years and included the Hobbs clinical score.

The evaluation with Duplex after 1 and 3 years respectively is reported in table 10.14.

The recurrence rate after 3 years was significantly different depending on the competence or otherwise of the terminal valve. With the competent terminal valve, the recurrence rate was 3% at the sapheno-femoral junction, compared to 71% in case of incompetent terminal valve after 3 years. (Comment by Paolo Zamboni)

16) EVA I. et Al.: CHIVA - ECOGRAPHIC ASPECTS AND SURGICAL RESULTS

Maxilo-facial surgery volume 18 • issue 1 January / March 2014 • pp. 64-70

Abstract

Varices (milk leg) represent pathological dilatations of the superficial veins at the level of the inferior members. Up to now, the strictly anatomical aspect of varix formati- ons inspired only traditional, strictly ablative treatments, generally applied without aiming at improving the hae- modynamic condition of veins. Haemodynamic surgery attempts at modifying the reflux pattern, while preserving the most efficient channels of venous drainage. Implemen- tation of such a treatment requires an exact understanding of the physiological principles and of the reflux patterns on which haemodynamic surgery relies. Ecographic eva- luation of the venous system in patients with varicose dila- tations permits drawing of a detailed map of the venous system, and also of its haemodynamic pattern [1]. Con- sequently, CHIVA appears as a viable therapy, applicable in outpatient services, as well. Post-surgery results are excellent and patients' comfort is appreciated as highly satisfactory. The method is reliable, having produced no incidents, accidents or complications.

17) Claude FRANCESCHI, Massimo CAPPELLI, Stefano ERMINI, Sergio GIANESINI Erika MENDOZA, Fausto PASSARIELLO, Paolo ZAMBONI. CHIVA: hemodynamic concept, strategy and results

International Angiology 2016 February;35 (1):8-30

ABSTRACT

The first part of this review article provides the physiologic background that sustained the CHIVA principles development. Then the venous networks anatomy and ow patterns are described with pertinent sonographic interpretations, leading to the shunt concept description and to the consequent CHIVA strategy application. An in depth explanation into the hemodynamic conservative cure approach follows, together with pertinent review of the relevant literature.

18) MALDONADO-FERNANDEZ et Al.: Clinical results of a new strategy (modified CHIVA) for surgical treatment of anterior accessory great saphenous varicose veins. Cir Esp. 2016 Mar;94(3):144-50.

Abstract

INTRODUCTION:

Traditionally, anterior accessory great saphenous vein insufficiency was managed by crossectomy and resection of varicose veins. The aim of this paper is to show the safety and efficacy of a new therapeutic strategy for anterior accessory great saphenous varicose veins.

METHODS:

This non-randomised prospective study included 65 patients with varicose veins from the anterior accessory great saphenous vein. The novelty of the technique is to avoid the great saphenous vein crossectomy and perform just flebectomy of the visible veins. Venous duplex studies were performed preoperatively, a month and a year postoperatively. The clinical assessment was done by the Fligelstone scale.

RESULTS:

The baseline CEAP clinical classification was: 58% C2, 26% C3 and 15% C4-6. The new strategy was applied to all cases.

COMPLICATIONS:

3 haematomas, 7 cases of asymptomatic partial anterior saphenous thrombosis. Reduction of the initial average diameter was from 6.4 mm anterior saphenous to 3.4 mm by one year ($p < 0.001$). At twelve months a forward flow is maintained in 82% of cases. Recurrence of varicose veins was 8%. All patients improved their clinical status based on the Fligelstone scale. Cases with saphenous diameter bigger than 7.5 mm and obesity were identified as predictors of worse clinical and hemodynamic outcome.

CONCLUSIONS:

This modified surgical strategy for anterior saphenous varicose veins results in better clinical outcomes at one year postoperatively.

19) ZMUDZINSKI M, MALO P, HALL C, HAYASHI A., CHIVA – A prospective study of a vein sparing technique for the management of varicose vein disease, Am. J. Surg., 2017, 213: pp. 967–969.

ABSTRACT

CHIVA. Una tecnica di risparmio del patrimonio venoso nell'insufficienza venosa cronica (Zmudzinski 2017)

Studio prospettico che ha valutato la percentuale di recidiva dovute a reflusso venoso con tecnica CHIVA. Gli autori hanno valutato 150 procedure sia con esami ecografici che clinici pre e post operatori. I pazienti sono stati seguiti a 3 mesi e a 1 anno dopo l'intervento.

La valutazione duplex post–operatoria del reflusso è stata eseguita in posizione supina, l'intervento al sito di giunzione safeno–femorale consisteva in una doppia legatura con 2–0 di seta a 2 cm della giunzione safeno–femorale (distalmente alla confluenza delle vene epigastriche). La recidiva è stata definita come presenza di reflusso nella GSV alla coscia durante l'esame duplex.

Non sono state rilevate recidive al follow–up iniziale, in seguito, 58 gambe hanno completato il follow–up a lungo termine ed il reflusso è stato rilevato all'esame Doppler in 5 gambe con un tasso di recidiva dell'8,6%; CI del 95% (2,4%, 19%).

Nessuno di questi pazienti ha presentato complicazioni cliniche. Gli autori hanno concluso che la percentuale di recidiva utilizzando il metodo CHIVA compete favorevolmente con le tecniche di ablazione venosa. L'elevata soddisfazione del paziente, la bassa percentuale di complicanze ed il basso costo incoraggia a proseguire gli studi con questa tecnica (tabella10.16). (PAOLO ZAMBONI)

20) FRANCESCHI C, Bahnini A, Cappelli M, Cuaranta RL, Dadon M, Delfrate R, Ermini S, Gianesini S, Mendoza E, Passariello F, Puskas A. : Commentary on the article "A prospective study of a vein sparing technique for the management of varicose vein disease" by M Zmudzinski et al. Am J Surg. 2018 Nov;216(5):1035.

21) Felipe Puricelli Faccini, Stefano Ermini, Claude Franceschi : CHIVA to treat saphenous vein insufficiency in chronic venous disease: characteristics and results . J Vasc Bras. 2019;18:e20180099. https://doi.org/10.1590/1677-5449.009918

Abstract

There is considerable debate in the literature with relation to the best method to treat patients with chronic venous disease (CVD). CHIVA is an office-based treatment for varicose veins performed under local anesthesia. The aim of the technique is to lower transmural pressure in the superficial venous system and avoid destruction of veins. Recurrence of varicosities, nerve damage, bruising and suboptimal aesthetic results are common to all treatments for the disease. This paper evaluates and discusses the characteristics and results of the CHIVA technique. We conclude that CHIVA is a viable alternative to common procedures that is associated with less bruising, nerve damage, and recurrence than stripping saphenectomy. The main advantages are preservation of the saphenous vein, local anesthesia, low recurrence rates, low cost, low pain, and no nerve damage. The major disadvantages are the learning curve and the need to train the team in venous hemodynamics.

ARTICLES COMPARING CHIVA RECURRENCES / CLINICAL DATA WITH OTHER PROCEDURES EMPLOING NOT RANDOMIZED STUDIES

1) GORNY PH., BLANCHEMAISON PH., CHAHINED.,HUTINEL B., CHANVALLON C., PAYEN

B., REINHAREZ D., Chirurgie Conservatrice et Ambulatiore: étude comparative entre CHIVA et Crosseectomie chez 321 patients opérés de la saphène interne. Discussion, Phlébologie, 1995, 48, 2: pp. 255–259.

2) CAPPELLI M.,MOLINO LOVA R., ERMINI S., TURCHI A., BONO G., BAHANINI A., FRANCESCHIC.I., La Cure CHIVA dans le traitement de la Maladie Variqueuse: analyse critique des résultats après trois ans, Ann. Chir. Vasc., 1996.

Cappelli et al. they examined 148 patients treated with an average 3-year follow-up CHIVA. The authors compared their own results with the large series of stripping present in the literature (Hobbs 1974, Taulaniemi 1963). The "Hobbs criteria" were chosen as the evaluation method, so that the groups were comparable. Hobbs' criteria were established in 1974

in the first randomized controlled trial in the history of phlebology, which compared surgery to sclerotherapy (Hobbs 1974). (Comment by Paolo Zamboni)

3) MAESO J., JUAN J., ESCRIBANO J., ALLEGUEN.M.,DIMATTEO A.,GONZALEZ E.,MATAS M.: Comparison of clinical outcome of stripping and CHIVA for treatment of varicose veins in the lower extremities

Ann. Vasc. Surg., 2001, 15: pp. 661–5.

Maeso et al., of the university clinic in Barcelona, instead monitored 90 patients operated on with the CHIVA strategy, in a prospective 3-year follow-up study and compared them: with 85 of their historical patients operated on stripping, with data from interventions stripping already present in the literature (see above) and with patients in the Cappelli group. In the Vall d'Hebron university clinic in Barcelona, stripping was completely abandoned in favor of the CHIVA method in

1995, therefore a prospective comparison of the two methods was not possible.

In both the Cappelli and Meso studies, the CHIVA method produced significantly better outcomes than the three stripping groups ($p < 0.001$). The comparison between the two groups CHIVA - Cappelli and Maeso - did not produce significant differences. The results are shown in tables 10.2, 10.3, 10.4, 10.5. (Comment by Paolo Zamboni)

4) NOPPENEY, T., Noppeney, J., Kurth, I.: Results of standard varicose vein surgery

(2002) Zentralblatt fur Chirurgie, 127 (9), pp. 748-751.

Abstract

The principles of modern varicose vein surgery are based on the interruption of the cranial and distal points of venous insufficiency. Especially due to the rise of alternate surgery procedures, we have scrutinized our results of varicose vein surgery. In a retrospective analysis, the results of those patients (pat.) who underwent venous surgery in our institution in 1995 were analysed. In 1995 we performed 1575 varicose vein operations (n = 1019 pat., 16.8 % male, 83.2 % female). 63.5 % were on an out-patient basis. During a follow-up period of 4 to 66 months (av. 38 months) the patients were re-examined, 481 (47.2 %) by Duplex sonography, 94 (9.2%) by clinical examination alone, from 103 (10.1 %) information was obtained through a written inquiry. 341 pat. (33.5%) had just perioperative follow-ups. We didn't find any signs of varicosis in 301 pat. (33.3 %). Minor side branches could be detected in 515 pat. (56.8 %). In 90 pat. (9.9 %) a clearly visible varicosis could be seen. Based on the results of the Duplex examinations, 86 % of the pat. showed no recurrence after ligation of the sapheno-femoral junction and stripping of the LSV, the results after stripping of the short saphenous vein were similar. Analysis of the inquiry forms concluded that 62.3 % of the pat. were satisfied with surgery and the results. The results of the standard varicose vein surgery are satisfactory regarding recurrence rate and patient satisfaction. **Our results are comparable to those**

published in prospective randomised studies. Alternate procedures, for example the CHIVA method, have still to proof their efficiency, especially in view of long-term results.

5) MARIA S. et Al. : Varicose disease of lower extremities: What kind of treatment? Personal experience

Chirurgia 2008, 21 (4), pp. 195-198.

Abstract

Aim. We report the experience over 422 patients admitted and treated for varicose disease on lower extremities, in the U.O. Clinicizzata di Chirurgia D'urgenza of Vittorio Emanuele Hospital of Catania from 01/01/2001 to 12/31/2005. The surgical treatment was modulated by the intensity of the disease. Methods. The 422 patients were treated according to the following surgical techniques: **14 (3,3%) patients with CHIVA method**; 4 (0,9%) patients with reconstruction of saphenofemoral valve (as described by Belcaro); 10 (2,3%) patients with multiple phlebectomies (as described by Muller); 8 (1,9%) patients with crossectomy and multiple phlebectomies; 252 (59,7%) patients with stripping of the great saphenous vein from the groin to the knee (short stripping); 134 (31,7%) patients with stripping of the great saphenous vein from the groin to the ankle (long stripping). Results. **On the 14 (3,3%) patients treated with CHIVA method were noticed the following complications: 6 (42,9%) cases of saphenous vein thrombosis; 8 (57,1%) cases of varicose recurrences during the follow-up (15 months - 3 years) Pochi casi e la maggioranza non drenanti**. On all 4 cases (0,9%) treated with reconstruction of saphenofemoral valve (as described by Belcaro) were noticed saphenous vein thrombosis before the 48-hours following the procedure. On 252 (59,7%) patients treated with the short stripping were noticed: 8 (3,17%) cases of postoperative complications; 3 (1,2%) cases of varicose recurrences. On 134 (31,7%) patients treated with the long stripping were noticed: 9 (6,7%) cases of postoperative complications; no case of varicose recurrences. Conclusion. Finally, according to obtained results, we agree that the gold standard of surgical treatment of the varicose disease of the lower extremities, is represented by the stripping of the saphenous vein, with a low percent of postoperative complicances and/or varicose recurrences.

6) Solís, J.V., Ribé, L., Portero, J.L., Rio, J.: Stripping saphenectomy, CHIVA and laser ablation for the treatment of the saphenous vein insufficiency (2009) Ambulatory Surgery, 15 (1), .

Abstract

Aim: To analyze the results of three different techniques for the treatment of the great saphenous vein insufficiency as the main cause of varicose veins.

Methods: We analyze three groups (Stripping, CHIVA 1 and Endovenous Laser ablation) with 40 patients each. Follow up was done at 1, 3, 9 and 12 months. Results: **The CHIVA and laser ablation had the best aesthetic result and fewer discomfort, but laser ablation had higher economic cost. There was no recurrence after 1 year in any of the groups**. Conclusions: The three techniques proved very good results for the saphenous insufficiency treatment.

7) FRANCESCHI C.: “Stripping versus the CHIVA Method" Angéiologie, 2010

8) MILONE M., SALVATORE G.,MAIETTA P., SOSA FERNANDEZ L.M.,MILONE F., Recurrent varicose veins of the lower extremities after surgery. Role of surgical technique (stripping vs. CHIVA) and surgeon's experience, G. Chir., 2011, p. 32.

This is a retrospective analysis that compared the result after stripping with those of CHIVA method (5-year follow-up) in two different periods: the first group concerned the patients treated in the years immediately following the learning of the CHIVA 1995-2000 method, the second group included patients treated between 2001 and 2005 after the team had completed a sufficient learning curve relative to the surgical strategy used.

The results were evaluated according to the Hobbs criteria. In the first period 223 patients underwent stripping and 88 under CHIVA. Complete success of the treatment was found in 30.9% after stripping and in 12.6% after CHIVA ($p < 0.05$), while treatment failure instead was found in 47.5% stripping and 67 % of CHIVA patients ($p < 0.05$). In the second period 186 patients were treated with Stripping and 208 with CHIVA. The success rate in the Stripping group remained constant at 29.5% while it significantly increased in the CHIVA group at 44.2% ($p < 0.05$). 46.7% of stripped patients were classified as inefficient treatment while in the CHIVA group there was a significant reduction of failed treatments to 30.2% ($p < 0.05$). The authors conclude that training and adequate experience in vascular surgery and ultrasound ultrasound are required to successfully perform the CHIVA method. (Comment by Paolo Zamboni)

9) CHAN CY et Al.: Retrospective comparison of clinical outcomes between endovenous laser and saphenous vein-sparing surgery for treatment of varicose veins. World J Surg. 2011 Jul;35(7):1679-86. doi: 10.1007/s00268-011-1093-8.

Abstract

BACKGROUND:

The purpose of the present study was to compare management of varicose veins by endovenous laser ablation (EVL) and a vein-sparing procedure (CHIVA: Conservatrice et Hémodynamique de l'Insuffisance Veineuse en Ambulatoire) for management of varicose veins.

METHODS:

Data from 82 consecutive patients with great saphenous vein (GSV) reflux and primary varicose veins presenting to the vascular clinic at the Far Eastern Memorial Hospital between June and December 2005 were reviewed. Of these, 74 who met the inclusion criteria were included in this study. CHIVA was performed by a double division of the refluxing saphenous vein (i.e., proximal and distal ligation), and EVL was performed using 10-14 W beginning approximately 4 cm below the saphenofemoral junction to the level of the knee. Phlebectomy for significant branch varicose veins on the leg was routinely performed in all patients. **Outcome measures included postoperative thrombophlebitis, bruising, pain, assessment of ultrasonographic and clinical symptoms (measured by the Venous Clinical Severity Score [VCSS]) and comparison of quality of life survey scores obtained preoperatively and postoperatively (measured by the Aberdeen Varicose Veins Score [AVVQ] and RAND-36). Patients were examined one week post-procedurally and again at 1, 3, 6, and 12 months.**

RESULTS:

Endovenous laser ablation and CHIVA were performed on 54 and 20 patients, respectively. The EVL patients had significantly higher pain scores and bruising than the CHIVA group ($p<0.001$). The VCSS of varicose, oedema, pigmentation, and inflammation were significantly reduced after both EVL and CHIVA; however, patients treated by EVL had significantly more pain postoperatively than those treated by CHIVA ($p=0.003$). Twenty-two of 54 (40.7%) and 3 of 17 (17.6%) patients in the EVL and CHIVA groups, respectively, required sclerotherapy for residual varicosities ($p=0.026$). Both groups benefited significantly from surgery in disease-specific perceptions.

CONCLUSIONS:

The CHIVA patients had less pain postoperatively and a significantly higher sclerotherapy-free period compared to patients in the EVL group. Further follow-up studies to compare long-term results of various approaches to surgically managing varicose veins are needed.

10) D KELLEHER, T R A Lane, I J Franklin and A H Davies : Treatment options, clinical outcome (quality of life) and cost benefit (quality-adjusted life year) in varicose vein treatment

Phlebology 2012;27 Suppl 1:16–22.

Conventional surgery

Standard surgery for varicose veins was firstdescribed over 100 years ago, and is still considered the gold standard against which other treatment modalities are tested. The results of surgery are good and patients are generally satisfied. Surgery is associated with an improvement in QOL in most patients. However, there is a significant rate of minor complications. Rates of morbidity vary from series to series.

New techniques that have arisen interrupt the reflux haemodynamics while preserving the long saphenous vein and include the ASVAL and CHIVA techniques. These provide minimally invasive treatments performed under tumescent local anaesthesia, and have produced good results. One single-centre series has shown that while CHIVA offers improved recurrence rates compared with open stripping in experienced hands, it has a steep learning curve and can lead to worse

Conclusion

Varicose veins have a multitude of treatment options, all of which provide excellent improvements in QOL at a cost-effective level. Overall costs have fallen dramatically despite material requirements, and no patient should be without a treatment option. The treatment of varicose veins is one of the few treatments that offer low morbidity for large improvements in QOL. Importantly, despite the higher incidence of varicose veins in older patients, a high percentage of patients are of working age when health improvements are most cost-effective.

11) DE FRANCISCIS S. et Al. : Hemodynamic surgery versus conventional surgery in chronic venous disease: a multicenter retrospective study. ACTA PHLEBOL. 2013; 14; 109-114

From 1994 to 2012, 11.026 patients were treated surgically, of which 6044 in CHIVA and 4982 in stripping. The average follow up was 9 years.

Compared to stripping, CHIVA seems to improve both clinical and hemodynamic results and reduce the number of recurrences.

12) WANG H., CHEN Q., FEI Z., ZHENG E., YANG Z., HUANG X., HEMODYNAMIC CLASSICATION AND CHIVA TREATMENT OF VARICOSE VEINS IN LOWER EXTREMITIES(VVLE)

Int. J. Clin. Exp. Med., 2016, 9(2): pp. 2465–2471.

Abstract:

Objective: To develop a new method of classifying hemodynamics in varicose veins of lower extremities (VVLE) and make a comparative analysis to determine the efficacy of ambulatory conservative hemodynamic correction of venous insufficiency (CHIVA) treatment. Methods: 150 cases with VVLE in our hospital were selected. Firstly, color doppler ultrasound examination was performed for each patient. Secondly, the hemodynamics of the patients were systematically divided into 6 types: I, II, III, IV, V, and VI. Lastly, complications and recurrence rate were detected to investigate the clinical efficacy, the patients were evenly divided into 3 groups which receiving different treatments: traditional surgery group, endovenous laser treatment group and CHIVA group. Results: Compared with the other two groups, patients in CHIVA group showed significant better performances on clinical efficacy, cure rate, complications and recurrence rate ($P<0.05$ and $P<0.01$). Conclusion: CHIVA treatment has significant better curative effect than traditional surgery and endovenous therapy in the treatment of varicose veins. CHIVA treatment induced less damage, quicker health recovery, high safety factor and lower complications**. Thus, CHIVA treatment can be widely used in clinical restoration than general minimally invasive operations.**

ARTICLES COMPARING CHIVA RECURRENCES / CLINICAL DATA WITH OTHER PROCEDURES EMPLOING RANDOMIZED STUDIES (RCT)

1) ZAMBONI P., CISNO C., MARCHETTI F., MAZZA P., FOGATO L., CARANDINA S., DE PALMA M., LIBONI A., Minimally invasive surgical management of primary venous ulcers vs. compression treatment: a randomized clinical trial, Eur. J. Vasc. Endovasc. Surg., 2003 Apr, 25(4): pp. 313–8.

This prospective randomized study compared CHIVA strategy associated with compression with the use of compression alone in the treatment of venous ulcers associated with chronic superficial venous insufficiency of the lower extremities (C6 in the CEAP classification). 24 patients were treated with compression, advanced wound dressings (and treatment antibiotic if necessary) the dressings were changed every 3 to 5 days during the first month and every 7 days thereafter.

The CHIVA group included 21 patients, 16 extremities had a hemodynamic presentation similar to type I shunts and were treated with crossectomy and further tributary ligatures, 7 extremities had a type III shunt and were treated with type CHIVA 2 procedure.

The study assessed:

- the healing process expressed in 2 mm per day;

- the functionality of the venous system based on air plethysmography data before treatment, 6 months and 3 years after treatment;

- quality of life through SF-36 questionnaire before treatment

and 6 months after treatment. In addition to the clinical evaluation, an eco-Doppler examination was performed

every 6 months for a total of 3 years. The results are presented in table 10.7. (Comment by Paolo Zamboni)

2) CARANDINA S., MARI C., DE PALMA M., MARCELLINO M.G., CISNO C., LEGNARO A., LIBONI A., ZAMBONI P., Varicose vein stripping vs haemodynamic correction (CHIVA): a long term randomised trial, Eur. J. Vasc. Endovasc. Surg., 2008 Feb, 35(2): pp. 230–7.

This randomized comparative study aimed to compare the long-term results of stripping with respect to CHIVA in the treatment of chronic superficial venous insufficiency.

180 consecutive patients underwent clinical evaluation, including CEAP classification, and duplex examination performed by expert operators. 30 patients were excluded second because they did not meet the study inclusion criteria, while 150 patients were randomized to

two groups, 75 were treated with stripping and 75 with CHIVA. All operated extremities were examined by three independent assessors who had not been involved in previous surgical procedures. The results were evaluated according to the Hobbs criteria and are presented in tables 10.8 and 10.9.

The relative risk of recurrence in the Stripping group doubled to 10 years compared to the CHIVA group (OR 2.2; 95% CI 1–5, p <0.04).

No significant difference was found between the two 3-year techniques. During the 3 to 10 year period the different recurrence rates in the two groups become evident and significant, so it is concluded that at 10 years the risk of recurrence is double in the ablative group (Figure 10.1). (Comment by Paolo Zamboni)

Zamboni J "Varicose vein stripping versus haemodynamic correction (CHIVA): a long term randomized trial"].Mal Vasc. 2009 Feb; 34 (1): 65. doi: 10.1016 / j.jmv.2008.10.002. Epub 2008 Dec 4.

[Correspondence: letter by P. Zamboni about the analysis of the article

[Article in French]

P.

3) IBORRA–ORTEGA E., BARJAU–URREA E., VILA–COLL R., BALLÓN–CARAZASH., CAIROLS–CASTELLOTE M.A., Estudio comparativo de dos técnicas quirúrgicas en el tratamientode las varices de las extremidades inferiores: resultados tras cinco años de seguimiento,

ANGIOLOGÍA, 2006, 58(6): pp. 459–468.

Iborra and his team published a prospective randomized study in Spanish in 2006 that included **100 legs treated with CHIVA or Stripping with a 9-year follow-up.** 62 women and 38 men with an average age of 49 years were selected following the Spanish guidelines for the treatment of varicose veins. The patients included did not have a history of venous surgery, thrombosis, were not overweight or older than 70 years. 49 patients were randomized to the Stripping group and 51 to the CHIVA group. There were no differences in age, gender, weight and CEAP

between the 2 groups. All patients underwent Doppler examination and after the surgery, they received the same dose of prophylactic heparin. The follow-up with questionnaires and ultrasonography was performed 1 week after the intervention and then after 1, 3, 6 months and every year for 5 years. All patients in the stripping group were hospitalized (44 spinal and 5 under general anesthesia)

While of the CHIVA group 9 patients remained in hospital for one night, the rest were treated on an outpatient basis (6 spinal, 3 general, 42 local anesthesia), table 10.10.

The average working disability in the stripping group was 19 days while in the CHIVA group 8 days (p <0.001). Neither group experienced serious complications, 11 patients in the stripping group reported ankle paresthesia, while in the CHIVA group 4 patients reported symptomatic superficial venous thrombosis (table 10.11).

Despite the best recovery after CHIVA, the 5-year results for the

outcomes considered were not significantly different (table 10.12).

(Comment by Paolo Zamboni)

4) PARÉS J.O., JUAN J., TELLEZ R., MATA A., MORENO C., QUER F.X., SUAREZ D., CODONY I., ROCA J., Varicose vein surgery: stripping versus the CHIVA method: a randomized controlled trial,

Ann. Surg., 2010 Apr, 251(4): pp. 624–31.

The aim of this study was to compare the effectiveness of the CHIVA method for the treatment of varicose veins compared to the standard stripping treatment. The study design was randomized and controlled monocentric, and 501 patients with primary varices were included. Patients were randomly assigned to the CHIVA procedure (experimental group n = 167) or stripping without duplex mapping (control group 1, n = 167) or stripping with duplex mapping (control group 2, n = 167). The outcome measure was 5-year clinical recurrence, examined by independent evaluators previously trained in the procedures. Duplex ultrasonography has also been used to evaluate the causes of relapses. The results are summarized in table 10.13.

The odds ratio for the presence of 5-year relapses between the stripping group with clinical marking and the CHIVA group was 2.64, (95% confidence interval [CI]: 1.76–3.97, P <0.001). The odds ratio for relapses after 5 years of follow-up, between stripping with duplex mapping and CHIVA group, was 2.01 (95% CI: 1.34-3.00, P <0.001).

The conclusion was that the CHIVA surgical treatment had

fewer side effects and less recurrence after 5 years compared to both stripping groups. No statistical differences were found between the two stripping groups (with and without duplex mapping). (Comment by Paolo Zamboni)

Reviews COCHRANE e Metanalisis

1) BELLMUNT–MONTOYA S., ESCRIBANO J.M., DILME J., MARTINEZ–ZAPATA M.J., CHIVA method for the treatment of chronic venous insufficiency, Cochrane Database Syst. Rev., 2013 Jul 3, (7): CD009648.

2) ———, CHIVA method for the treatment of chronic venous insufficiency, Cochrane Database Syst. Rev., 2015 Jun 29, (6): CD009648.

The first review was published in 2013 and aimed to compare the effectiveness and safety of the CHIVA method with alternative therapeutic techniques for the treatment of chronic superficial venous insufficiency. Randomized controlled trials (RCTs) have been included to compare the CHIVA method compared to any other treatment. The primary endpoint was clinical recurrence, the studies included in the review had a follow-up of 3 to 10 years, and showed more favorable results for the CHIVA method compared to stripping (721 people, RR 0.63, 95% CI 0.51 to 0.78).

Only one of the studies included in the review reported data related to

quality of life (presented graphically) and these results also significantly favored the CHIVA method.

The stripping group had a higher risk of side effects than the CHIVA group; in particular, for the presence of hematomas (RR 0.63 95% CI from 0.53 to 0.76;) for nerve damage (RR 0.05 95% CI from 0.01 to 0.38).

No statistically significant differences were reported between the groups regarding the incidence of infection and superficial venous thrombosis. (Comment by Paolo Zamboni)

3) Guo L. et Al.: Long-term efficacy of different procedures for treatment of varicose veins A network meta-analysis

Medicine (2019) 98:7

Abstract

Background: Various procedures for the treatment of varicose veins have been shown to have long-term effectiveness, but research has yet to identify the most effective procedure. The aim of this

study was to investigate the long-term efficacy of different procedures based on Bayesian network meta-analysis and to rank therapeutic options for clinical decision-making.

Methods: Globally recognized databases, namely, MEDLINE, Embase, and Cochrane Central, were searched for randomized controlled trials (RCTs). Quantitative pooled estimation of successful treatment rate (STR) and recurrence rate (RR) was performed to

assess the long-term efficacy of each procedure with more than a 1-year follow-up. The surfaceunder the cumulative ranking (SUCRA) probabilities of the P values regarding STR and RR were calculated to rank various procedures. Grades of Recommendations Assessment, Development and Evaluation (GRADE) criteria were utilized for the recommendation of evidence from pairwise direct comparisons.

Results: A total of 39 RCTs encompassing a total of 6917 extremities were eligible and provided relative raw data. After quantitative

analysis, the CHIVA procedure was determined to have the best long-term efficacy, as it had the highest STR (SUCRA, 0.37).

Additionally, the results revealed that CHIVA possessed the highest probability of achieving the lowest long-term RR (SUCRA, 0.61).

Moreover, the sensitivity analysis with inconsistency approach clarified the reliability of the main results, and the evidence of most

direct comparisons was ranked as high or moderate.

Conclusion: **CHIVA seemed to have superior clinical benefits on long-term efficacy for treating varicose veins. However, the**

conclusion still needs additional trials for supporting evidence.

Abbreviations: CHIVA = Ambulatory Conservative Hemodynamic Management of Varicose Veins, Development and Evaluation,

GRADE = Grades of Recommendations Assessment, PRISMA = Preferred Reporting Items for Systematic Reviews and Metaanalyses,

RCT = randomized controlled trial, RR = recurrence rate, STR = successful treatment rate, SUCRA = surfaceunder the

cumulative ranking.

ARTICLES OF GENERAL REVIEW

1) Mendoza, E.: CHIVA 1988-2008: Review of studies on the CHIVA method and its development in different countries

(2008) *Gefasschirurgie*, 13 (4), pp. 249-256. Cited 1 time.

ABSTRACT

CHIVA was introduced by Claude Franceschi in 1988. This technique is based on analysis of the venous circulation; a small number of ligatures follow, which cause a volume discharge of the superficial (and deep) veins. The aim is to reduce the circulating volume in these veins, sparing the saphenous trunks and their drainage through perforators. Prospective studies that include 695 patients with 3-year follow-up have been published. In the past 2 years, three prospective randomized studies of CHIVA versus stripping, with follow-ups of 5-10 years, have been done (750 legs) and have been partially published (250 legs). They are summarized and discussed in this article. Treatment costs were lower for CHIVA. Subjective and objective results were either significantly better or equal to stripping, and a lower rate of recurrence was found. In the 10-year follow-up, the recurrence rate was double in the stripping group. CHIVA has become widespread particularly in Spain, where half of the interventions on veins are done using this technique. In France and Italy, CHIVA is covered by health insurance.

2) AGUS G.B.: Thirty years of new venous hemodynamic concept and teaching

Acta Phlebologica 2019 mese;20(0):000–000

DOI: 10.23736/S1593-232X.20.00458-0

Conclusion

Finally, thanks to hundreds of studies, some RCTs and a Cochrane review by various authors over Europe, CHIVA is today validated as more successful than destructive method and the more recent international meta-analysis concluded that CHIVA seemed to have superior clinical benefits on long-term efficacy comparing different therapeutic procedures for treating varicose veins.11-13 The efficacy of this approach was based on a better physiological process, and this revolutionary approach should be widely applied in clinics. However, the conclusion still needs additional trials for supporting evidence.

3) MENDOZA E.: Primum non nocere

Veins and Lymphatics, 2017, 6(2)

https://doi.org/10.4081/vl.2017.6646.

4) C FRANCESCHI C.: CHIVA 30 years later. Scientific and ethical considerations

Veins and Lymphatics, 2019 - pagepressjournals.org

107 ARTICLES ON CHIVA TREATMENT

BOOKS and CHAPTERS OF OTHER BOOKS

1) FRANCESCHI C.

(1988) Théorie et Pratique de la Cure Conservatrice et Hémodynamique de l'Insuffisance Veineuse Ambulatoire,

Précy-sous-Thil: L'Armançon

Edizione: Francese, Italiana, Inglese

2) ZAMBONI P.

(1996) La chirurgie conservativa del sistema venoso superficiale. (1° EDIZIONE)

Gruppo Editoriale Faenza Editrice

3) CAPPELLI M. ERMINI S. MOLINO LOVA R.

(2001) Chapter: La correzione emodinamica o cura CHIVA pag 431-456 book: Trattato di Flebologia e Linfologia Vol 1 Sergio Mancini (1° Edizione)

Masson Editor

4) MENDOZA, E.

(2002) Chiva Handbuch,

Arrien, Wunstorf

5) CAPPELLI M. MOLINO LOVA R. ERMINI S.

(2003) Chapter: Chirurgia conservativa emodinamica pag 177-187 book: Chirurgia delle vene e dei linfatici Giuseppe Genovese

Masson Editor

6) ESCRIBANO J.M.

(2006) Cirugia Hemodinamica en el tratamiento de la insuficiencia venosa superficial

Tesis Doctoral

Universitat Autonoma de Barcelona

7) FRANESCHI C., ZAMBONI P.

(2009) Principles of venous hemodynamics

Hauppauge, NY: Nova Science Publishers

MENDOZA E. CHRISTOPHER R. LATTIMER NICK MORRISON N.

(2014) Duplex Ultrasound of surfaceLeg Veins

Springer Editor

8) ROBERTO DEL FRATE

(2014) A new diagnostic approach to varicose veins: haemodynamic evaluation and treatment

Lorena Dioni publisher

9) ZAMBONI P., MENDOZA E., GIANESINI S.

(2018)Saphenous vein-sparing strategies in chronic venous disease

Springer Editor

10) ZAMBONI P.

(2019) La chirurgie conservativa del sistema venoso superficiale. (2° EDIZIONE)

Aracne editrice

11) JORDI JUAN SAMSO'

(2019) La cura CHIVA en el tratamiento de las varices primarias de las extremidades inferiorores

Aran editor

9 BOOKS PUBLISHED ON CHIVA TREATMENT

CONCLUSION

The following elements rise up from the analysis of the articles:

The results of different CHIVA studies about clinical data, recurrence rates and quality of life, comparing them with other methods of treatment without randomization, are superimposable on each other. So they are not sporadic cases, referring to individual studies.

All randomized studies as well as the two cochrane reviews and the meta-analysis demonstrate the superiority of CHIVA compared to other treatments in terms of recurrence and quality of life at 5 and 10 years.

The biochemical analysis of the pre- and post-chiva inflammatory markers, together with the demonstration of a regression of the saphenous wall alterations after treatment, confirm exetremely the possible use of a post-CHIVA saphens trunk for arterial by-pass. Anyway the incompetent great saphenous vein has been always used for by-pass, especially in case of infra-inguinal arteriopathies.

The low spread of CHIVA treatement and the learning curve cannot be criteria influencing the levels of evidence. Indeed, they must be a stimulus to optimize the own work.

Therefore CHIVA represents the treatment of superficial venous insufficiency which gives the best results over time compared to all the other methods applied: stripping and endovascular procedures; with the big advantage of being able to preserve a saphenous trunk for a possible use as an arterial by-pass. About this aspect, I would like to underline , two concepts:

The age of population increases, therefore the probability of finding phlebopathic patients with arteriopathies increases

Patients, awareness of venous problems, approch earlier their own phlebological problems so the probability of finding saphenous veins not involved or less alterated is high.

Chapter 10

Sclerotherapy and CHIVA

Chapter author: Massimo Cappelli Florence Italy

Sclerotherapy and CHIVA

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SCLEROTHERAPY: DEFINITION AND MECHANISM OF ACTION

The sclerotherapy treatment consists in inducing, through a chemical phlebitis, with consequent thrombosis, a fibrotic process of a vein.

The evolution of this process is represented by: the closure of the vessel, until a possible resorption of the vessel itself, or a marked reduction in its calibre.

The determinant variables, in a probabilistic key, of an evolution compared to another are given by the calibre of the vessel, its tortuosity and how the treatment is conducted.

Chemical phlebitis is induced through an endothelial lesion caused by the intravenous administration of substances, called sclerosing substances.

The extent of the injury caused will depend on:

A) on the state of the venous wall, an already damaged endothelium will be more sensitive to the action of sclerotherapy

B) the concentration of the substance used, but above all the concentration of the substance within the blood volume contained in the vein; this is why the optimal injection should be performed in a vein with the smallest possible blood volume inside.

The extension of the lesion along the treated vessel will instead be conditioned by the volume of the sclerosant injected.Ref: ("La sclérose des varices" 4° édition R. Tournay. Expansion Scientifique Francaise)

Endothelial damage will induce the development of three closely interrelated events:

(a) Thrombosis of the vessel, characterized by a thrombus highly adherent to the wall and confined to the extent of the lesion induced by the percentage and volume of the injected substance.

(b) The inflammatory process of the wall triggered either by the injury or by secondary thrombosis.

c) The activation of fibrinolysis, which is closely related to the size of the thrombus and the extent of inflammation induced.

In fact, there is a direct correlation between the extent of endothelial damage, inflammation, thrombosis and fibrinolysis.

Any fibrotic process, the essence of sclerotherapy, is always the result of a chronic inflammation, whose characteristic element is the macrophage functional variation towards collagen synthesis, expressed by the change of the macrophage, then a phagocyte (Macrophage M1), towards the fibroblast-like phenotyping (Macrophage M2) with

consequent collagen synthesis. Chronic inflammation is in fact characterized by a persistent reduction of the M1/M2 ratio mediated essentially by IL-6 and TGF-B, cytokines typical of this phase. Ref: ("The PNEI and the myofascial system: the structure that connects" Marco Chiera and Al. EDRA publisher).

This element of general pathology is the key point in understanding how sclerosing therapy should be conducted.

The inflammatory process of the wall but, extents to the thrombus, so that it too undergoes fibrosis, with consequent obliteration of the vessel, unless it is lysed first; in this case the fibrotic process would remain confined to the wall of the vein with retraction and reduction of calibre, and complete obliteration would become less likely.

Thus, there is a competition between the fibrosis of the thrombus induced by the chronic inflammatory process and its fibrinolysis. (Fig1)

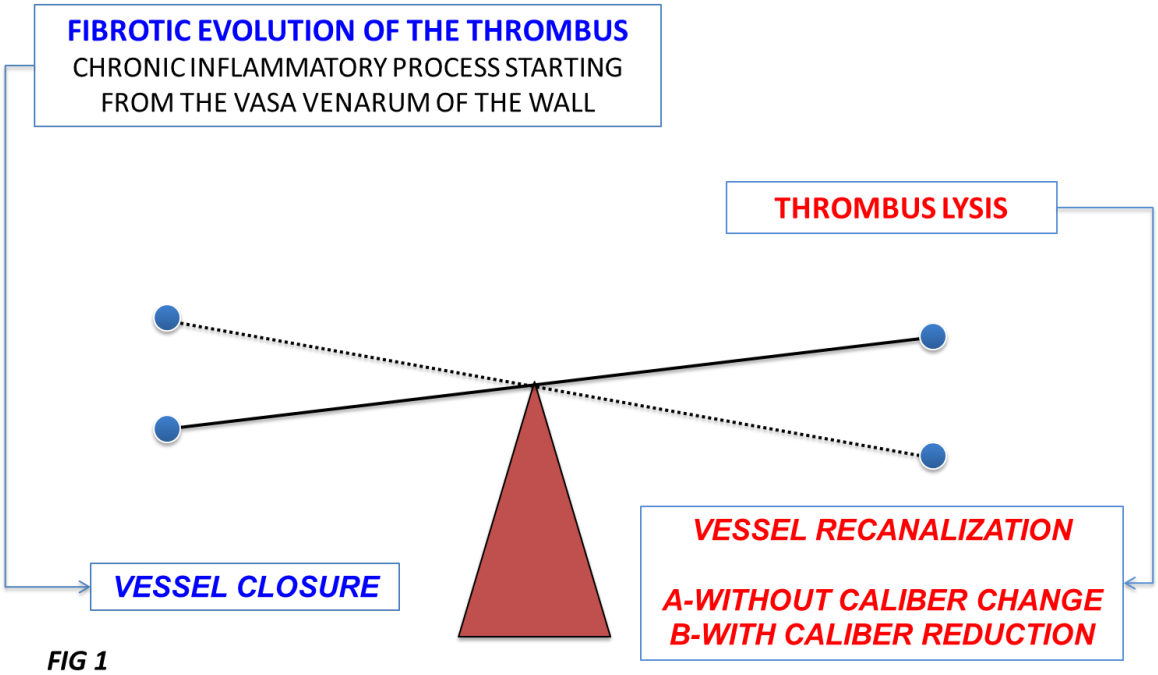


FIG 1

For the fibrotic process to prevail in this competition, it is essential that the endothelial injury be confined to this structure or at most involve the innermost part of the media without going further. Let us see why:

1°) The extended lesion of the media would lead to the destruction of those structures from which the intraparietal inflammatory process starts: the vasa venarum (particularly represented in the veins, extended up to the endothelium, given the low partial pressure of oxygen in the venous reflux blood); consequently the phlogosis, originating from the

adventitia, would take longer to invade the thrombus, which in this case would be larger. Since the inflammation extends to more structures, the result would be an acute inflammation with greater activity, also involving perivenous tissues and related structures, such as nerves.

Ref: (" Les vasa vasorum des artères" WILLIAMS J.K. et Al. Journal des Maladies Vasculaires 1996 21, supp. C, 266-269)

("Improvement of immediate and long-term results in sclerotherapy" Wenner L. : VASA. 1986, Vol 15, Num 2, pp 180-183)

2) The more extensive the lesion is, the greater and more acute will be the inflammatory reaction with a consequent delay in the change to the chronic form and therefore delay in the formation of the fibrotic process.

3°) As we have seen, the greater the acute inflammatory process, the greater and more rapid will be the activation of fibrinolysis of the thrombus.

Ref: ("PNEI and the myofascial system: the structure that connects" Marco Chiera and Al. EDRA publisher).

As proof of this, it is common experience that an inflammatory reaction particularly pronounced post-sclerotherapy, tends to an early recanalization of the treated vessel, depending of course, also on the calibre of the vessel, then the size of the thrombus, as well as its tortuosity. (Fig 2)

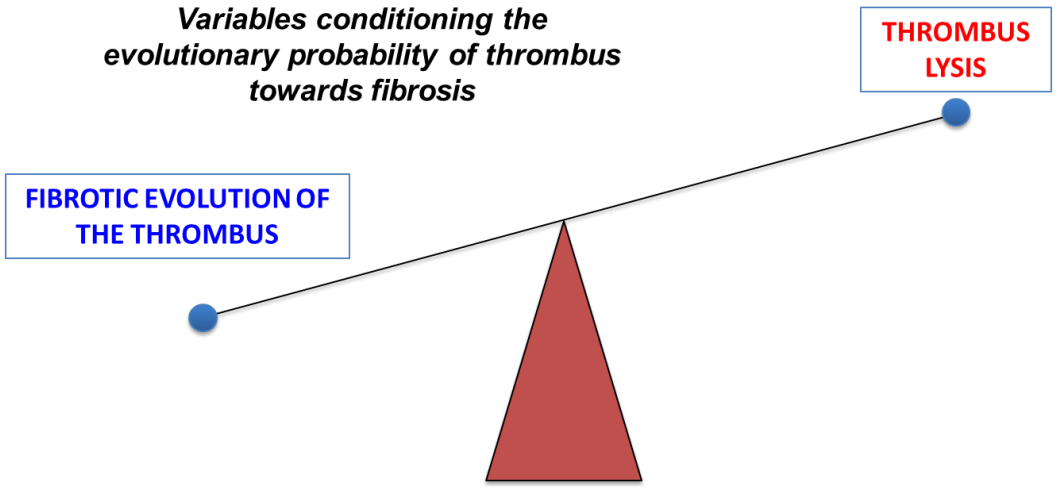


FIG 2

In this regard, it is essential, especially in very superficial vessels in the territory of R3 (N3), to apply eccentric compression after sclerotherapy, aimed at reducing the calibre of the vessel and thus the size of the thrombus secondarily induced.

The application of eccentric compression, regardless of the depth of the vessel and therefore of its possible reduction in calibre, has however the function of modulating the inflammation triggered by sclerosis, through an inhibitory reflex originating from a reduction of the afferential amyelin load of the C fibers, supported by both algogenic receptors and informational pathways of the tissue (somatic introceptive receptors). In fact, the myelinated Ib fibers stimulated by compression, block at the level of the posterior horns the afferential amyelinic load, through the mechanism of "Gate Control", resulting in a control of the sympathetic response with modulation of inflammation and algogenic threshold.

Ref: "Theories of pain: from specificity to gate control": .Moayedi M et. Al Neurophysiol. 2013 Jan;109(1):5-12. doi: 10.1152/jn.00457.2012. Epub 2012 Oct 3.PMID: 23034364

As evidence of this, it is common experience, not to find an inflammatory reaction under eccentric compression, but rather close to it.

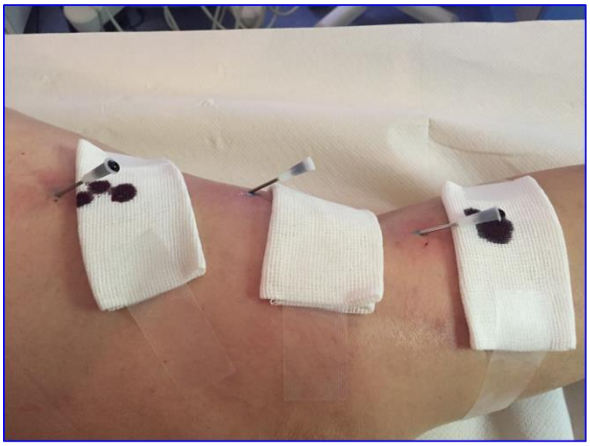
Therefore, after sclerotherapy, always apply an eccentric compression with possible overlapping of an elastic restraint.

In the case in which the thrombotic and therefore inflammatory component is particularly marked, once the latter has been reduced, the thrombotic material and/or its colliquation by fibrinolysis and/or the blood "trapped" between two thrombotic segments must be evacuated, using needles of different calibre or the tip of a scalpel (Fig 3)

REMOVAL OF "TRAPPED BLOOD"

ULTRASOUND-GUIDED NEEDLE PLACEMENT

WASHES WITH BASIC PROCAINE SOLUTION



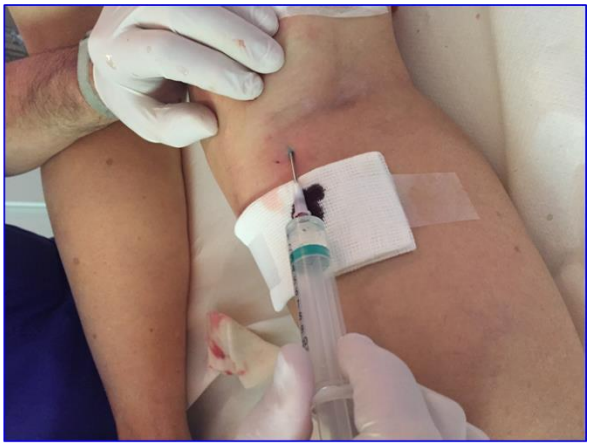


FIG 3

. In this way, all the inflammatory process is reduced and therefore also the melanocytic activation, which is the main responsible, together with hemosiderin, for post-sclerotherapy hyperpigmentation. **Ref:** *("Pigmentation and matting after C1 sclerotherapy" MORAGLIA L. Phlébologie 2018, 71, 1, pag 79)*

Any perivenous inflammatory reaction, always the expression of an overreaction, will tend to be less and less symptomatic, although still present, as it affects vessels in depth, for example in the saphenous compartment.

Ideal sclerosis should occur completely asymptomatically, which translates echographically into a nondeformable vessel, initially for thrombus, subsequently for fibrosis, with no change in perivascular echogenicity, the inflammatory process being confined to the vessel wall. An initial perivenous hypoechogenicity, followed by a hyperechogenic halo denotes a perivenous inflammatory engagement with subsequent fibrosis, involving the structures located close to the treated vein.

In this regard, particular attention should be paid to those vessels of the external region of the popliteal fossa , located under the superficial fascia, often in contact with the external popliteal sciatic nerve (EPS), as for example we find in some perforators of the popliteal fossa or in external collaterals of the small saphenous vein located very proximally. (Fig 4)

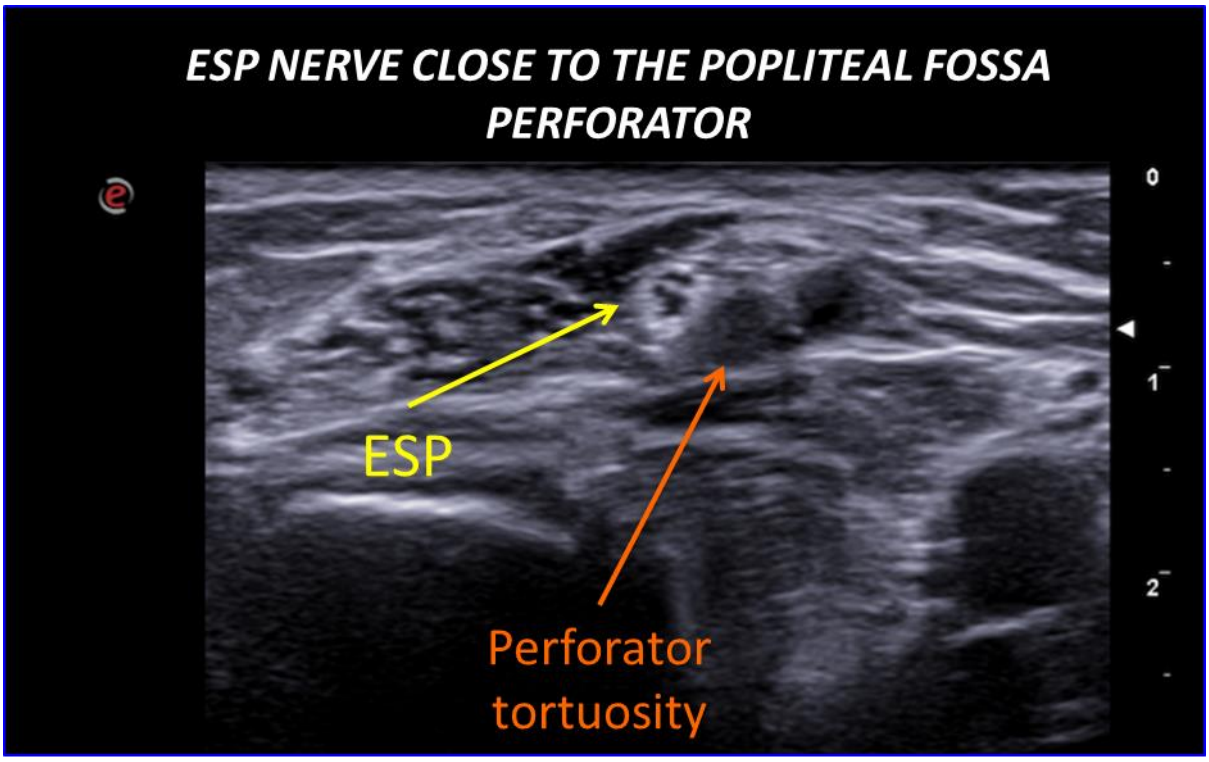


FIG 4

A correct sclerosis, therefore, without perivascular commitment sustained also by a possible accidental perivenous injection, does not involve any difficulty in case of subsequent surgical dissection. We would find, in that case, only an indurated vessel at section.

The use of ultrasound-guided sclerosis also reduces the risk of intra-arterial injections to a minimum. However, the rich vascularization around the perforators of the popliteal fossa and the post-surgical scar areas, especially in the inguinal region, are noteworthy. (Fig 5)

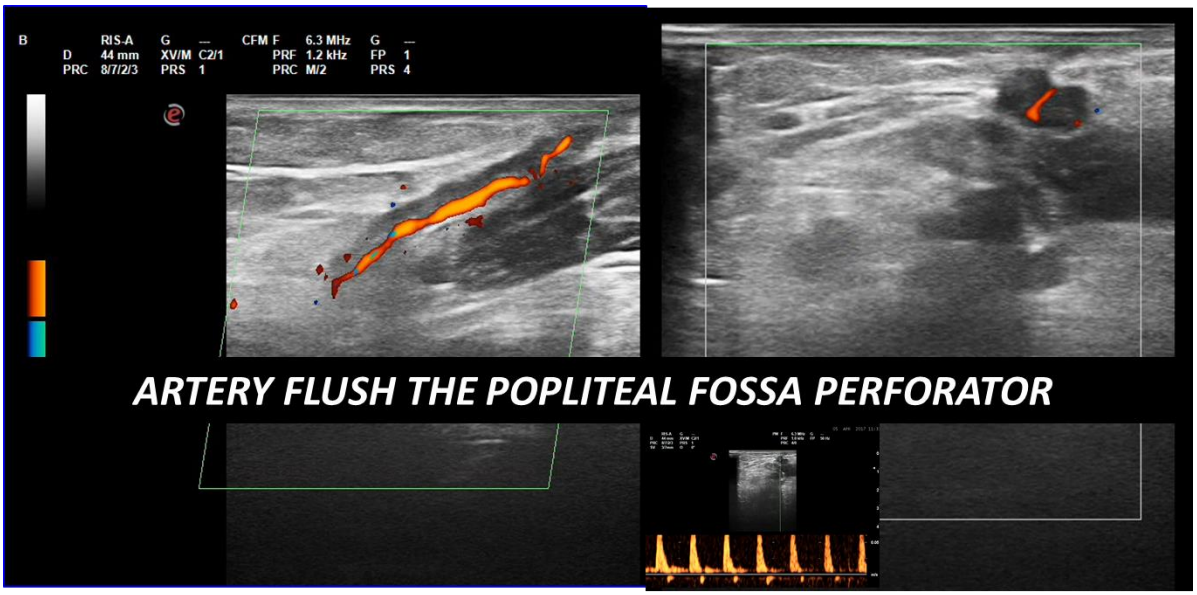


FIG 5

Therefore:

1) NON-AGGRESSIVE SCLEROSIS, USING THE LOWEST CONCENTRATION DEEMED EFFECTIVE.

THE PERCENTAGE AND TYPE OF SCLEROSANT CAN BE VARIED AT SUBSEQUENT SESSIONS

2) USE A SUITABLE VOLUME.

3) ALWAYS APPLY ECCENTRIC COMPRESSION

IT IS A MISTAKE TO THINK OF USING SCLEROTHERAPY AS A SCALPEL.

Sclerosis resistance induced by the use of various percentages in progression is a rare event and, in any case, very debatable.

SCLEROSING SUBSTANCES

Several sclerosing agents are available, the most commonly used being dehydrating substances and cleansers.

Dehydrating substances, which are much less potent than detergents, are of two types:

- Hypertonic, such as saline (20% sodium chloride).

- Non-hypertonic, such as salicylate and glycerol, which interact with the vessel wall by opening water channels, resulting in dehydration of endothelial cells.

Detergents act by altering the lipid and protein component of the membranes of endothelial cells and among these we find two groups:

Anionic group, including sodium tetracil sofate (Thrombovar, Fibroven)

Non-ionic group, including polydodecanol (Aetoxisclerol) are both quickely deactivated by the bloof proteins Ref: *("Terapia sclerosante ed elastocompressiva delle flebopatie" F. FERRARA. P Piccin Editore)*

Detergents, besides being used in liquid form as well as dehydrating substances, being surfactants, can be mixed with gas (air or O2/CO2) in various percentages, regarding both the sclerosant and the liquid/gas ratio, and be used in foam form.

Foam is much more powerful than the liquid form with the same percentage of sclerosant, since its molecules are distributed on a much larger Superficial, represented by the gas bubbles. It also has a very strong inertia to movement, being a visco-elastic body, resulting in a longer contact time with the endothelial Superficial.

It is always said that the foam once injected moves the blood as a whole and does not mix with it, unlike liquids. This statement is only partially true, depending mainly on the speed of the injection and the volume of foam injected. If the injection is done slowly, the foam stratifies on the superficial side of the lumen, progressing adhered to the surfaceof the wall, without mixing with the blood or displacing it. **All this can be verified by ultrasound and has a very important repercussion on the injection technique.**

The great advantage of the foam is that it can be identified ultrasound and therefore followed in its distribution in the various compartments.

We are not talking about a single foam but about various foams and, according to the type of detergent, its concentration and how they are produced, they can be wet or dry, stable or unstable, little viscous or very viscous. For a detailed description of their characteristics, please refer to specific texts. Ref: ("Foam sclerotherapy. State of the art" Henriet J.P. Editions Phlébologiques Francaises)

Any foam can be made with the Tessari method (2 syringes and a three-way valve) by changing the percentages and the liquid/gas ratio.Ref: ("Nouvelle technique d'obtension de la scléro-foam" Tessari L.: Phlébologie 2000; 53: 129)

Wet foam: liquid/gas ratio 1/4

Dry foam: liquid/gas ratio 1/8-9

WET FOAM can be made with any concentration of sclerosant, it has a higher liquid component, it has large and more inhomogeneous bubbles, therefore subject to coalescence, thus conditioning a lower stability, especially if more diffusible gases are used such as a mixture O2/CO2 than air, rich in nitrogen. The high liquid component, the large size of the bubbles, as well as their inhomogeneity, give it a low viscosity and therefore a rapid dislocation in the injected vessel.

DRY FOAM cannot be made with low concentrations of sclerosant, it has a lower liquid component, smaller bubbles, homogeneous and polygonal in shape, therefore subject to a much lower coalescence, thus conditioning a greater stability, especially if air is used, rich in nitrogen and therefore less diffusible than a mixture of more diffusible gases such as

O2/CO2. The low liquid component, the small size of the bubbles, as well as their shape and homogeneity, give it a high viscosity and therefore a slow dislocation in the injected vessel. *Ref: (" Quelle foams pour quelles indications ?" MONFREUX Phlébologie 2013, 66, 3, p : 11-18)*

As we will see later, these differences in the various foams will prove to be an important variable for sclerotherapy treatment placed in the CHIVA context.

POST-SCLEROSIS HEMODYNAMICS

As we have seen, the possible evolution of sclerotherapy of a vessel is twofold: on the one hand obliteration, on the other hand reduction of calibre.

The vessel obliteration, whatever it is, as all ablative methods, may affect a more or less stable increase of TMP Transmural Pressure in the vessels afferent to the treated vessels, thus realizing an acute district venous insufficiency. Ref: ("Pathogenesis of varicose veins - lessons from biomechanics" L. Pfisterer, T. Korff VASA 2014; 43: 81)

Depending on the extent of macrocirculatory compensation represented by supra- and transfascial networks we will have the more or less marked reduction of TMP.

In the case of a recanalization with reduced calibre the hydrostatic pressure will be the same being it independent of the section; what will vary will be the refluent volume, being reduced the calibre. This results in a reduction of the hydrodynamic energy of the reflux (E = P tot. x volume) and therefore of the lateral pressure and velocity.

Added to this is an increase in the elastic reaction force of the venous wall due to fibrosis, thus opposing the force of a reduced TMP. It follows that even in the case of a recanalization of a treated vessel, we can have a patient completely asymptomatic and without visible varices.

The problem is how long this situation can last, no one can predict it, also because the hydrostatic pressure remains constant and in the presence of a significant shunt, sclerosis can demonstrate its limits in the treatment of the escape point.

SCLEROSIS OF INCONTINENT CONFLUENCES (ESCAPE POINTS)

The progression of a sclerosing substance towards a confluence is conditioned by the Residual Pressure and by the pressure developed by the operator on the syringe piston during injection.

In case the outlet is in a vessel with a much higher flow velocity, as it can be a deep vessel, the sclerosing substance is immediately diluted and removed by the flow velocity, thus minimizing the contact time with the wall of the deep axis, thus preventing its injury; also to be taken into account is the progressive and rapid inactivation of the sclerosing substance by blood proteins. The result is a flush sclerosis of the treated branch of the deep vessel,

without extension of the thrombus beyond the outlet. The rate of post-sclerotherapy DVT is indeed very low, well below 1%.

Ref: ("Le traitement de l'insuffisance de la petit saphène par écho-sclérotherapie à la foam est-il à risque de thrombose veineuse profonde?" GILLET J.L. et al. Phlébologie 2015, 68, 2, p. 16-25)

It seems consistent, according to some works, that the rate of post-sclerotherapy DVT increases with the use of foam, given its intrinsic characteristics, conditioning a viscosity and therefore a slowed displacement compared to liquid substances.

Ref: ("The frequency and clinical significance of nontarget superficial and deep vein occlusion after physician compounded foam sclerotherapy of varicose tributaries" LOBASTOV K. et Al. Phlebology 2020 35 (6) 430-439)

In the case where the escape point originates from the saphenous vein, the probability that the sclerosis of the branch extends to it is high, since the saphenous vein at rest does not have a high flow velocity or, in any case, a much higher one than the treated branch. This is why sclerotherapy tactics aimed at preventing or otherwise limiting the passage of the sclerosant into the saphenous vein must be applied, as we will see later.

SCLEROSIS OF INCOMPETENT CONFLUENCES (ESCAPE POINTS)

The progression of a sclerosing substance towards a confluence is conditioned by the Residual Pressure and by the pressure developed by the operator on the syringe piston during injection.

In case the outlet is in a vessel with a much higher flow velocity, as it can be a deep vessel, the sclerosing substance is immediately diluted and removed by the flow velocity, thus minimizing the contact time with the wall of the deep axis, thus preventing its injury; also to be taken into account is the progressive and rapid inactivation of the sclerosing substance by blood proteins. The result is a flush sclerosis of the treated branch of the deep vessel, without extension of the thrombus beyond the outlet. The rate of post-sclerotherapy DVT is indeed very low, well below 1%.

Ref: ("Le traitement de l'insuffisance de la petit saphène par écho-sclérotherapie à la foam est-il à risque de thrombose veineuse profonde?" GILLET J.L. et al. Phlébologie 2015, 68, 2, p. 16-25)

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In the case where the escape point originates from the saphenous vein, the probability that the sclerosis of the branch extends to it is high, since the saphenous vein at rest does not have a high flow velocity or, in any case, a much higher one than the treated branch. This is why sclerotherapy tactics aimed at preventing or otherwise limiting the passage of the sclerosant into the saphenous vein must be applied, as we will see later.

THE ROLE OF SCLEROTHERAPY IN CHIVA

How can the application of an essentially destructive method, such as sclerotherapy, be reconciled with a conservative strategy such as CHIVA?

It depends on the context and purpose with which sclerotherapy is used.

WHEN TO DO SCLEROTHERAPY AND WHY

The fields of application are as follows

A) IN THE CHIVA STRATEGY

B) IN THE POST-CHIVA AESTHETIC FINISHING

C) IN THE CONTROL OF RECURRENCES/EVOLUTION OF VARICOSE DISEASE AFTER CHIVA INTERVENTION

D) IN THE TREATMENT OF POST-CHIVA MATTING

A) SCLEROTHERAPY IN THE CHIVA STRATEGY TACTICS

In CHIVA we have two key points to address: one of strategic order and the other of tactical order.

-- The strategic one is represented by the need to reconcile the reduction of TMP, with the maintenance of an optimal outflow velocity in the treated systems. This is to avoid pro-inflammatory stress shares conditioning the onset of relapses.

-- The tactical problem is the deconnection, at the level of an incontinent junction (escape point), of the refluent branch. It must be performed strictly flush with the vessel of origin, in order to avoid stumps, sources of recurrences.

The flow within a stump can be laminar or turbulent depending on the size, length, and shape of the stump.

In the case of a stump with laminar flow we will have a reduction in velocity by the flow continuity equation, and a subsequent increase in TMP by Bernouilli's law. Both conditions are pro-inflammatory.

In the case of a stump with turbulent flow, a vibrational discontinuity of the TMP is added to the above, which is also a source of inflammation.

Phlogosis is triggered by endothelial adhesion by leukocytes and platelets, which is followed by their activation. If the stump was "washed" by afferent branches of adequate capacity, the increase in speed and therefore of the stress share within it, would not allow the phenotyping on the endothelial glycocalyx of the various adhesion molecules, thus preventing the establishment of a pro-inflammatory condition and therefore evolutionary. Ref: ("Pathogenesis of varicose veins - lessons from biomechanics" L. Pfisterer, T. Korff VASA 2014; 43: 81)The inflammatory reaction of the wall always triggers angiogenetic processes with consequent possible formation of relapses starting from the lumen: this is made possible by the connection that these vessel structures may have with the functional component of the vasa venarum, whose outlet is located at the level of the valvular sinuses.

Ref: ("The venous valve agger and plasma noradrenaline-mediated venodilator feedback" Crotty TP. Phlebology. 2007;22(3):116-30.)

In some particular anatomical conditions, the surgical act, in order to reach the flush of the origin of the passage of the refluent compartment, requires an invasive gesture, such as the displacement of the sartorius muscle with opening of the vaso-adductor membrane to disconnect at flush of the femoral a refluent Hunter perforator.

In such conditions sclerotherapy can play a role and be of great help.

Therefore, it can be applied in the treatment of escape points that are difficult to attack surgically properly, such as:

-- THE SPJ when the strategy requires its disconnection

-- SOME PERFORANTS

-- SOME PELVIC SHUNTS, as we will see later (especially the parietal ones, some P points and the Clitoral point).

Another field of application of sclerotherapy, in the execution of the CHIVA strategy, is the treatment of peripheral vessels programmed for disconnection, even in the context of a two-steps CHIVA strategy (CHIVA 2) that provides for the execution of the peripheral time first, as may occur in type III or type V shunts.

To be the object of sclerosis, these vessels must be essentially small calibre and/or tortuous; tortuosity, by slowing the flow, increases the contact time of the sclerosant with the wall, thus optimizing the sclerotherapeutic response. Another fundamental criterion for the

choice of sclerotherapy instead of surgical disconnection, is the deep outlet of the vessel in the saphenous trunk, where the escape point is located, especially in adipose extremities affected by FEP (Fibrous Edematous Panniculopathy).

The aesthetic result, in these conditions will be much better than a surgical approach, since the latter requires an incision not small to go flush with the vanishing point, even if of small calibre.

B) SCLEROTHERAPY IN POST-SURGICAL AESTHETIC FINISHING

The varicose saphenous collaterals, once disconnected, tend to reduce their calibre due to the reduction of the TMP. This process is amplified with the application of an elastic containment that determines a further reduction of the TMP. With time, the parietal remodelling (reduction of V0 and increase of compliance) induces, in most cases, the disappearance of varicose branches. Sometimes this process is partial, and the varicose branch is still visible, representing an aesthetic problem. This occurs especially in the case of inveterate varices, characterized by a perivascular fibrous reaction, with formation of a "canyon" along the course of the varicose branch. In such cases the disconnected vessels can be treated with sclerotherapy, also using liquid substances with low concentration.

C) SCLEROTHERAPY IN THE CONTROL OF RECURRENCES/EVOLUTION OF VARICOSE DISEASE AFTER CHIVA SURGERY.

In the course of time, after CHIVA surgery, and in any case less frequently than with saphenous trunk ablative methods, refluent vessels may appear. Ref: ("CHIVA method for the treatment of chronic venous insufficiency" Bellmunt-Montoya S. et Al. Cochrane Database of Systematic Reviews 2012, Issue 2. Art. No.: CD009648. DOI:10.1002/14651858.CD009648 .)

In this case, the outlet of the varicose branch will be located at the level of the saphenous trunk. The picture of recurrence/evolution will therefore be orderly and straightforward in the surgical resumption, unlike in the recurrence of ablative treatments, which is often not associated with escape points (the so-called outflow recurrence, found in about 22% of operated patients

Ref: ("Varicose Vein Stripping vsHaemodynamic Correction (CHIVA): a long-term randomized trial" Carandina S. et Al.: European Journal of Vascular and Endovascular Surgery 2008;35(2):230-237).

In case the recurrence is characterized by small calibre and/or tortuous vessels, especially if their origin from the saphenous trunk is deep, especially in adipose extremities affected by PEF (Panniculopatia Edematosa Fibrotica), we can, as already mentioned above, resort to sclerotherapy treatment. Obviously, vessels of a certain calibre and with straight outlet will be of surgical competence.

HOW TO PROCEED WITH SCLEROTHERAPEUTIC TREATMENT

If we review all the conditions listed above in which it is possible to apply sclerotherapy, we can group them into 3 groups:

1) TREATMENT OF SOME ESCAPE POINTS

2) TREATMENT OF COLLATERALS DISCONNECTED FROM THE SAPHENOUS TRUNK

3) TREATMENT OF COLLATERALS CONNECTED WITH THE SAPHENOUS TRUNK

These 3 conditions require a different sclerotherapeutic treatment for each of them, aimed at not involving the saphenous trunk in the sclerosis reaction of the treated vessel; **in other words, the sclerosing substance must remain confined to the vessels under treatment and not pass into the saphenous vein.**

It must be kept in mind that the possible sclerotherapeutic involvement of the saphenous trunk determines a thrombosis on a chemical basis and not on a hemodynamic basis, resulting instead from an absent or insufficient drainage. Therefore, the tendency to recanalization will be reduced, since the fibrinolytic potential of the endothelium is damaged, whereas it is preserved when the thrombosis is caused by stasis.

The recanalization or not of the thrombus depends not only on the mechanism that generated it, but also on its extension. A segmental thrombosis is recanalized in the majority of cases. **This is why it is necessary to prevent the passage of the sclerosant into the saphenous vein or to limit the amount of sclerosant as much as possible.**

It must be kept in mind, however, that even a segmental obliteration of the saphenous trunk, which is probably transitory, conditions a hemodynamic instability of the system during the occlusion phase. The blockage or reduction of post-CHIVA reflux conditions an absence or reduction of sheare-stress in the saphenous vein (Dynamic Pressure) with consequent triggering of an inflammatory reaction on the whole axis, to which is added the inflammatory reaction of sclerosis close to the obstruction. The result is an involvement of the ostial valves of the collaterals, especially in the distal area close to the area of sclerosis, with possible appearance of valvular incontinence of the same, up to the structuring of a reflux determining a possible varicose recurrence, or the appearance of a matting, as we will see later.

Let us now consider the three points listed above

1) TREATMENT OF SOME ESCAPE POINTS

PERFORATOR

Once it has been decided, according to the strategy, to interrupt a perforator, the modality, surgical or sclerotherapeutic, will depend on some characteristics of the perforator itself, such as the length of the subfascial tract and its morphology. In the vast majority of cases, subfascial deconnection of perforators still leaves stumps. (Fig 6)

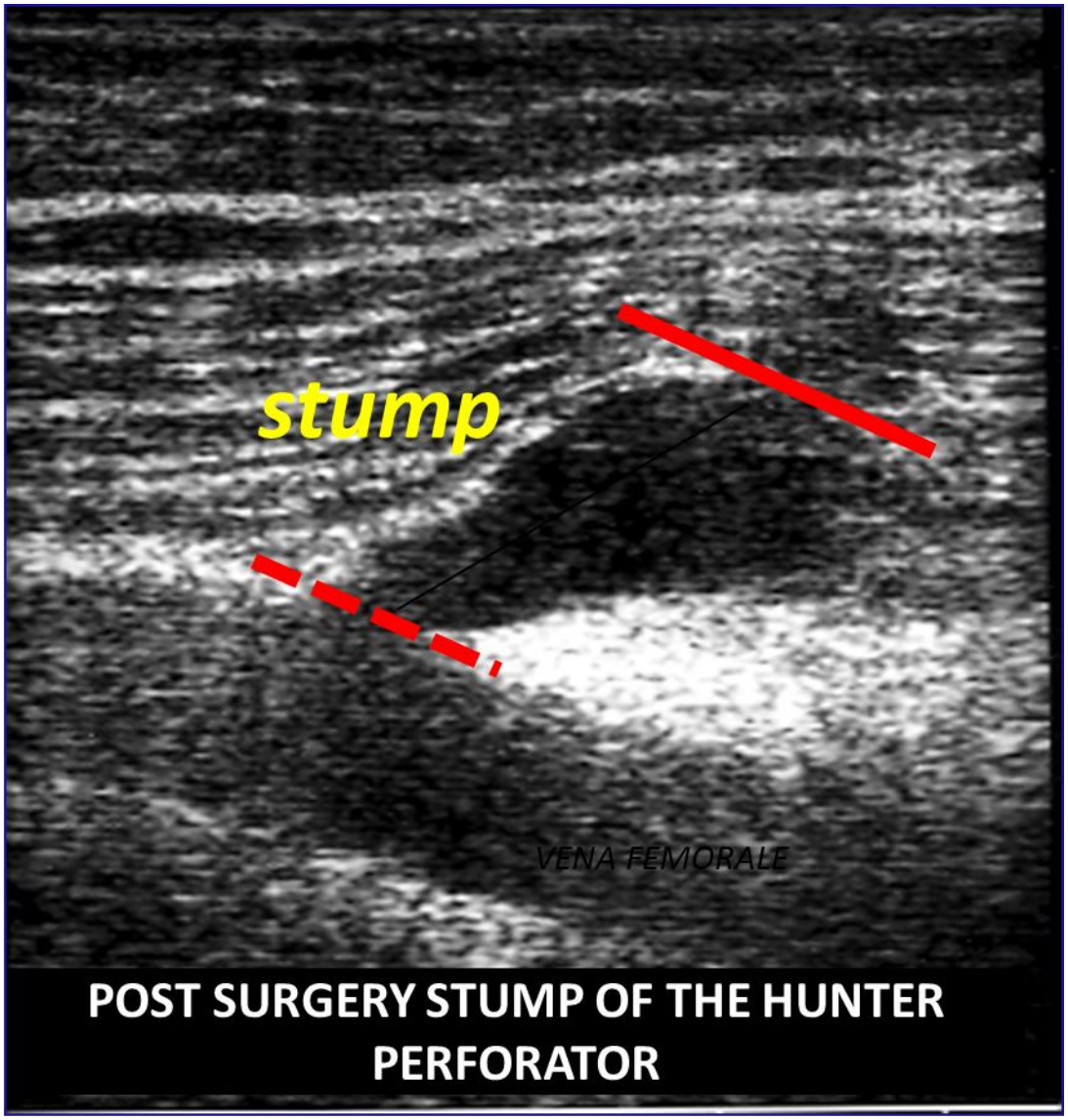


FIG 6

The transfascial network, of which the perforators are the main collectors, means that finding multitroncular perforators, especially in the subfascial tract, is the rule. Therefore, a flush surgical disconnection of multiple branches of the multitroncularity becomes practically impossible. It should be kept in mind that even at the surfacewe can find multiple outlets in different vessels. This variability therefore requires not only a hemodynamic study

of the perforator, but also an anatomical one. It follows that surgical interruption should be performed only in selected cases.

A perforator centred on one of the medial collectors of the soleus pump (branch connecting the soleus veins with the posterior tibial veins (Figure 7))

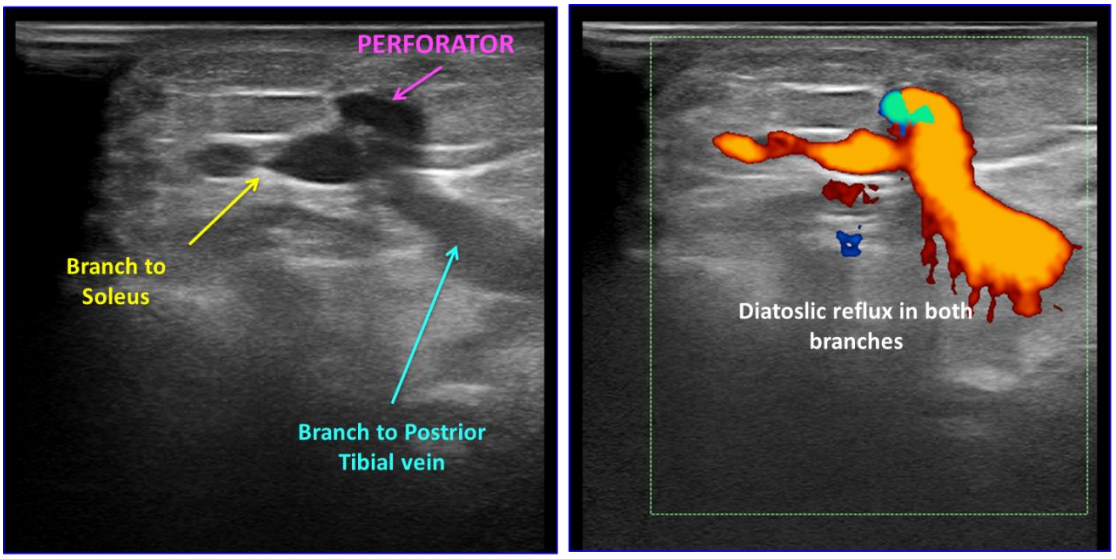


FIG 7 **CALF PUMP PERFORATOR DIASTOLIC REFLUX**

in a lean limb can be surgically disconnected with minimal gesture without leaving stumps, the collector being located close to the fascia. Another thing, as we have seen above, is a Hunteriana perforator, or a perforator of the popliteal cord or external thigh, all characterized by a particularly long subfascial course.

In such conditions the sclerotherapeutic indication is maximal, because:

the sclerosis extends along the entire length of the perforator, coming flush with the outlet in the deep axis without involving it.

the sclerosis involves the multritroncularity that may be present.

PERFORATOR CENTERED ON THE SAPHENOUS TRUNK

In case the perforator is centred on the saphenous axis, before performing the intraoperative sclerotherapy, it is necessary to isolate it from it surgically to avoid the passage of the sclerosing substance into the saphenous vein, after which we can proceed to the sclerosis of the perforator by incanulation.

To minimize the risk of DVT, the use of liquid sclerosants is essential, with immediate passive and active mobilization of the limb to increase the flow velocity at a deep level. Heparin prophylaxis and ultrasound monitoring at 7 days is routinely performed.

PERFORANT OFF-CENTER FROM THE SAPHENOUS TRUNK

In cases where the perforant is off-centre from the saphenous trunk and connected to it by a collateral branch, surgical disconnection or manual compression of the communicating vessel before the sclerotherapy procedure is essential, again to prevent passage of the substance into the saphenous vein.

Therefore, to summarize, the sclerotherapeutic treatment of a perforant depends on the length of the subfascial tract and how the multitroncularity is distributed in depth and on the Superficial.

In the case of large deep perforators centered on the saphenous trunk, the use of a small amount of glue with appropriate cure time, injected directly under ultrasound guidance into the perforant itself, may be considered. However, the procedure leaves stumps, as does a laser treatment of the perforant, since it cannot go flush with the deep outlet due to the risk of DVT.

SAPHENOUS-POPLITEAL JUNCTION

The CHIVA approach to the treatment of the saphenous-popliteal junction involves deconnecting the saphenous arch distally to a collateral of the convexity (Giacomini's v. and/or thigh extension of the external saphenous vein (TE-SSV), so that it ensures saphenous-popliteal "stump flushing."

This hemodynamic condition is not always achieved. In fact, it depends on the incontinence of the left collateral, therefore with downward retrograde flow, and on the entity of its calibre conditioning a good flow in the stump. (Fig 8)

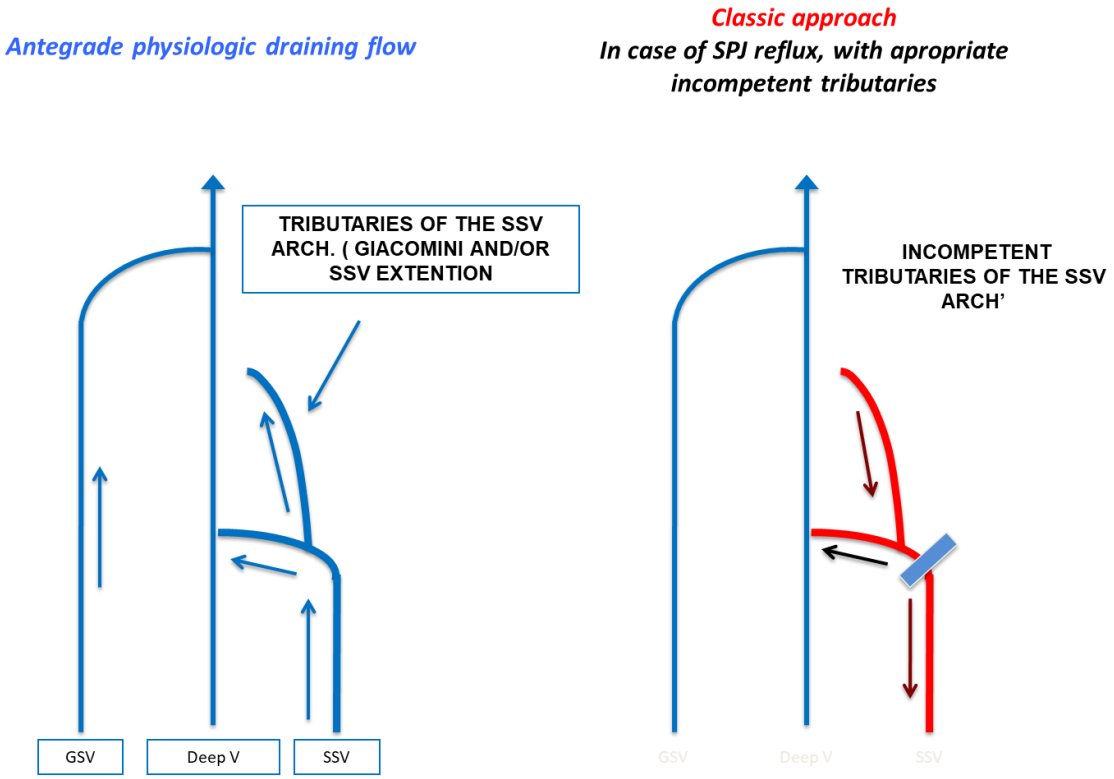


FIGURA 8

Fig 8

o

A common trunk with the gastrocnemus veins always ensures drainage of the saphenous-popliteal stump, regardless of the conditions described above, such as the presence of systolic reflux through the junction, an expression of a vicarious shunt OVS. **(Fig 9)**

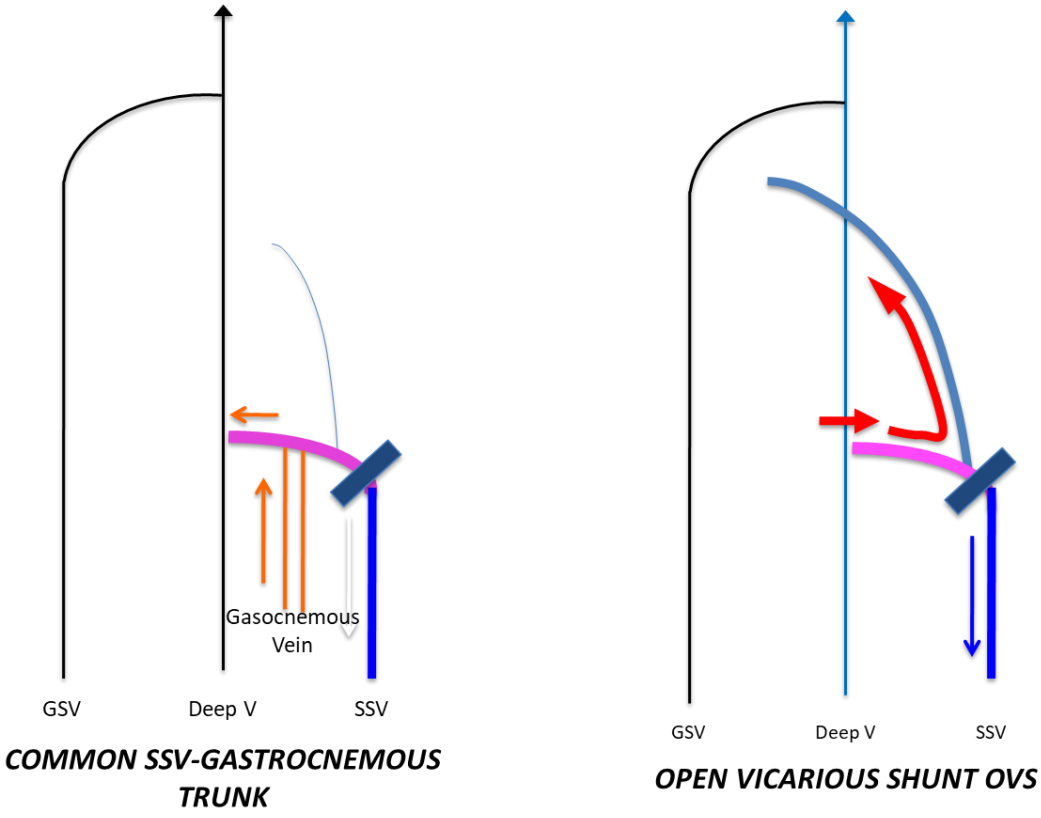


Fig 9 ***CONDITIONS FOR A GOOD STUMP WASH***

If the hemodynamic conditions, according to the classical approach, foresee the realization of an insufficiently drained stump, we could opt: either for a surgical interruption of the saphenous-popliteal junction or, much more simply, for an intraoperative sclerotherapeutic option.

The surgical solution, as to be performed flush with the popliteal, presupposes in most cases, also considering the variability of the outlet, a wide surgical access with wide dissection. As a consequence, there is a risk of popliteal DVT as well as neurological damage due to the presence of perinervous scar tissue, even in the absence of direct nerve injury.

The sclerotherapeutic solution is much easier and has a lower incidence of complications. The aim is to disconnect the external saphenous vein before it deepens in the popliteal cavity, performing an intraoperative sclerosis of the stump with liquid sclerosants, according to the criteria already exposed, with immediate post-sclerotherapy mobilization, both active and passive, and heparin prophylaxis with ultrasound control at 7 days.

The sclerosis will come to the flush of the popliteal and the associated surgical deconnection, will stabilize over time the sclerosis of the stump, until its fibrous retraction to the flush of the deep axis. In the case of stump recanalization, an ultrasound-guided sclerotherapy touch-up will be sufficient.

The progress of the stump should be monitored over time until its disappearance, usually within one year. (Fig 10)

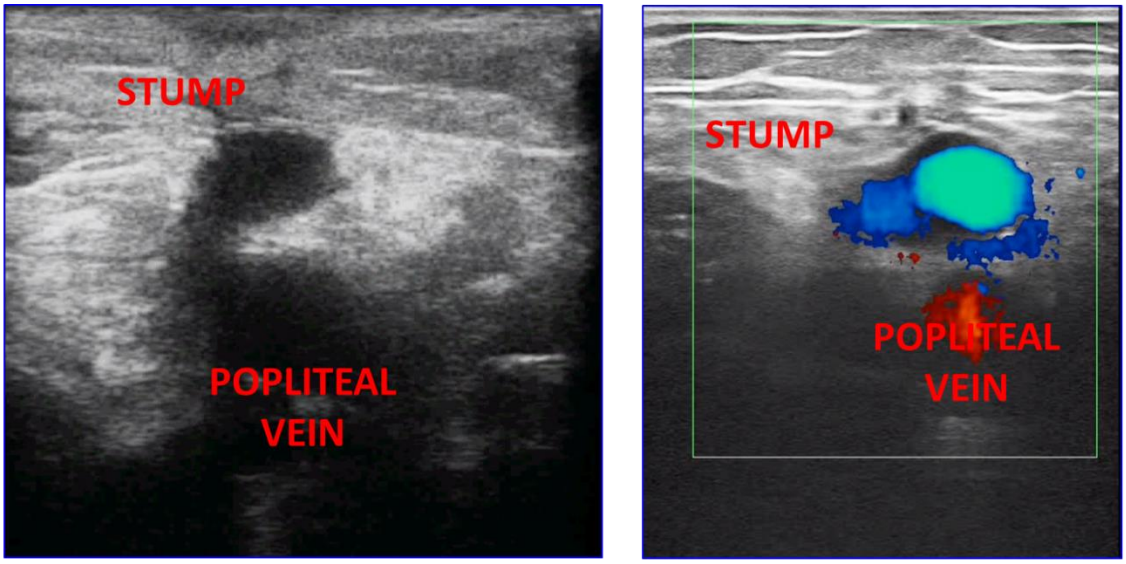


FIG 10 **FIBROUS RETRACTION OF THE STUMP TREATED WITH INTRAOPERATIVE SCLEROSIS AT 1 YEAR**

PELVIC SHUNTS

Pelvic shunts subject to sclerotherapeutic treatment are essentially represented by:

-- parietal shunts: superior and inferior gluteal veins

-- shunt originating from the deep dorsal vein of the clitoris (C Point)

-- shunt originating from the anterior labial vein (Intermediate Point)

-- shunt originating from the posterior labial vein at the level of the outlet in the internal pudenda in the canal of Alcock. (P Point)

The sclerotherapeutic treatment of the above-mentioned points can be done intraoperatively or even postponed with respect to the other surgical points, depending on the hemodynamic weight of the shunt in the context of the clinical and general cartographic picture. In any case, the interruption, either surgically or by compression, of the collateral outlet connecting the pelvic outlet with the saphenous trunk is fundamental.

Sclerotherapeutic treatment is usually ultrasound-guided, choosing the best position of the patient to inject close to the vanishing point. The Midpoint and P Point are generally treated in the gynaecologic position, injecting, under ultrasound guidance, superficially to the deep perineal fascia and never into the Alcok's canal because of the risk of injecting the internal pudendal artery or damaging the pudendal nerve.

Response to sclerosing therapy is good even in the case of a pelvic shunt with significant flow rate, because these vessels are tortuous and therefore particularly sensitive to sclerotherapy.

The high flow rate of a pelvic shunt, especially if continuous flow, may be the expression of a hypertensive condition of the intrapelvic network due to ovarian vein involvement (pelvic varicocele) or to a left iliac vein obstruction syndrome, whatever the cause; it may or may not be associated with a clinical picture referable to a pelvic congestion syndrome. In such conditions, before proceeding to the treatment of the shunt, it is necessary to solve the pathology at the basis of the hypertensive picture, also because the treatment of the shunt alone, assuming that it does not recur, which is very likely, could trigger or aggravate the clinical picture supported by pelvic congestion. The pelvic shunt assumes, in this case, the hemodynamic role of an open vicarious shunt perforator.

2) TREATMENT OF DECONNECTED COLLATERALS FROM THE SAPHENOUS TRUNK

These are deconnected vessels, therefore not subjected to shunt. This results in a low transmural pressure and flow velocity. If a certain tortuosity is added to this, the optimal conditions for a sclerotherapeutic treatment are achieved. It must be taken into account that in these conditions, the sensitivity to the action of sclerosing substances is high; this is why it is preferable to use liquid substances, applying a well-calibrated eccentric compression along the course of the treated vein, especially in case of "canyons".

The cartographic research of possible communications with the saphenous trunk is fundamental, in order to compress them manually during the injection, thus avoiding the extension of the sclerosing process from the branch to the saphenous axis.

3) TREATMENT OF COLLATERALS CONNECTED WITH THE SAPHENOUS TRUNK

The key point in the sclerotherapeutic treatment of a vessel connected to the saphenous trunk, is the realization of a flush sclerosis without stump, not involving the main axis in the sclerosis reaction. Therefore, it is necessary to prevent or limit as much as possible the passage of the sclerosant into the saphenous vein.

Compression of the outlet, the most logical option, prevents the passage of sclerosing fluid, but does not allow flush sclerosis, resulting in a stump that is a source of possible recurrence.

Therefore, it is necessary to calibrate the action of the sclerosis, bringing the sclerosant up to the saphenous vein outlet and not beyond.

Therefore, some conditions are needed:

1°) The sclerosing substance must be ultrasound visible, so a foam cleanser must be used.

2°) We must use a viscous foam, therefore slow in progression

3°) The injection must be slow and discontinuous in order to progress the foam millimeter by millimeter, until the outlet. The progression must take place when the vessel is completely filled with foam, so that the action of the sclerosis is extended over the entire endothelial surfaceof the vessel.

Let us now analyze some problems related to the method, essentially represented by the type of foam used and the injection method.

FOAM TYPE

As we have said, the foam must be viscous, therefore dry (see types of foam). This foam, with the Tessari method, can only be made using high concentrations, therefore excessive for a vessel that is the site of a secondary shunt, not of large calibre; it should also be kept in mind that this foam, possibly passing even in small quantities in the saphenous trunk, can affect an extensive sclerosis reaction.

For this reason, the use of an automatic mixer, Varixio® designed by Dr. E. Roche, can help us. It is able to form viscous foams, therefore tend to be dry, using low percentage detergents (for example Aetoxi 0.125% -0.5%), therefore suitable for the vessel to be treated and much less aggressive for the saphenous trunk. ("A new automated system for the preparation of sclerosant foam: A study of the physical characteristics produced, and the device settings required" Roche E. et Al. Phlebology 2020 Oct 35 (9) 724-733)

In my experience, cooling the foam with ice spray applied to the syringe further increases the viscosity and thus reduces the rate of progression through the vessel.

Moreover, the foam should not be particularly stable, as it must perform its action and rapidly degrade, especially in the case of an eventual passage into the saphenous vein; therefore, we prefer foam prepared with a mixture of O2/CO2 rather than with air.

VASCULAR FILLING OF THE FOAM AND INJECTION TECHNIQUE

In the case of a tortuous vessel, the slow and very discontinuous injection of foam leads to the total filling of the vessel and to its slow progression towards the outlet, until it stops at that level. (Fig 11)

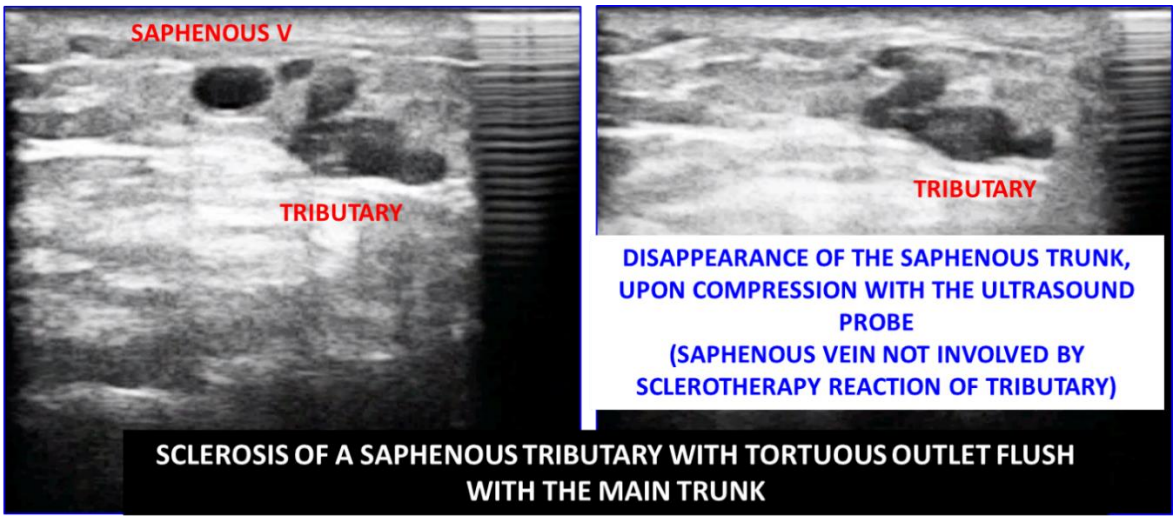


FIG11

In the case of a rectilinear vessel, the slow injection, even if discontinuous, conditions the stratification of foam on the superficial wall of the vessel without filling it, insinuating itself between the wall and the blood, with rapid progression towards the outlet; the result is an insufficient sclerosis reaction with passage of foam into the saphenous vein. Obviously, this phenomenon also depends on the calibre of the vessel. The smaller the vessel, the greater the tendency to fill, and the slower the progression of foam toward the outlet, according to the increase in resistance.

A more rapid and continuous injection will lead to the total filling of the vessel but will also condition a rapid and massive passage of the foam into the saphenous vein, which is obviously to be avoided.

For this reason, when we are faced with a straight vessel, we should proceed in such a way as to obtain the filling of the vessel and protect the saphenous vein trunk by slowing down the progression of the foam towards it.

If we combine the slow and discontinuous injection with a series of slow and alternating compressions with the ultrasound probe tilted to one side, always following the flow of the foam, we will notice a tendency to fill the vessel. In fact its closure, obtained with the bending of the probe, allows the realization of the filling.

Devices can be used, during evaluation, to be attached to the probe, always aimed at the same purpose. (Fig 12)

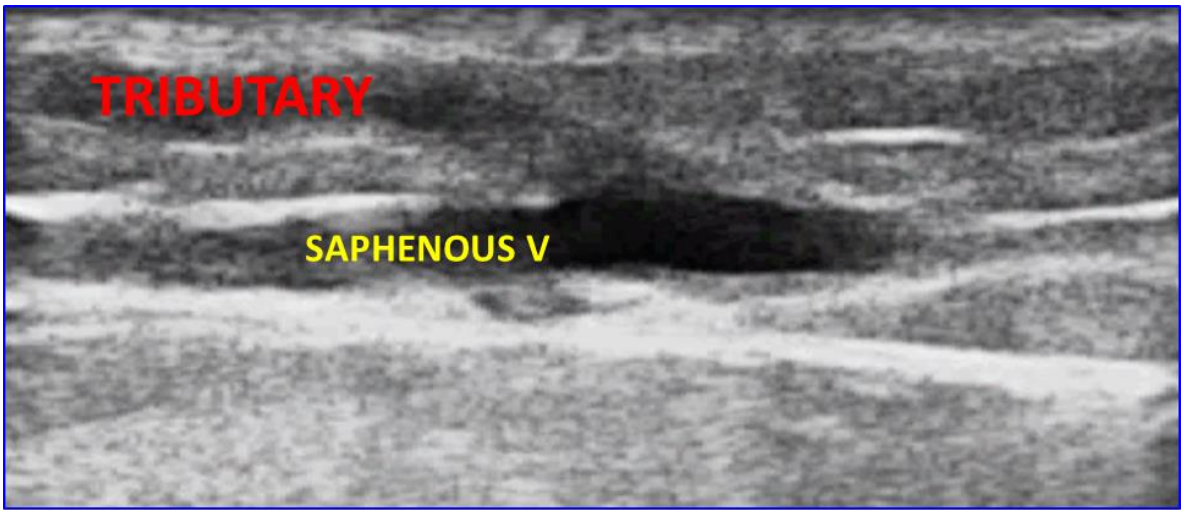
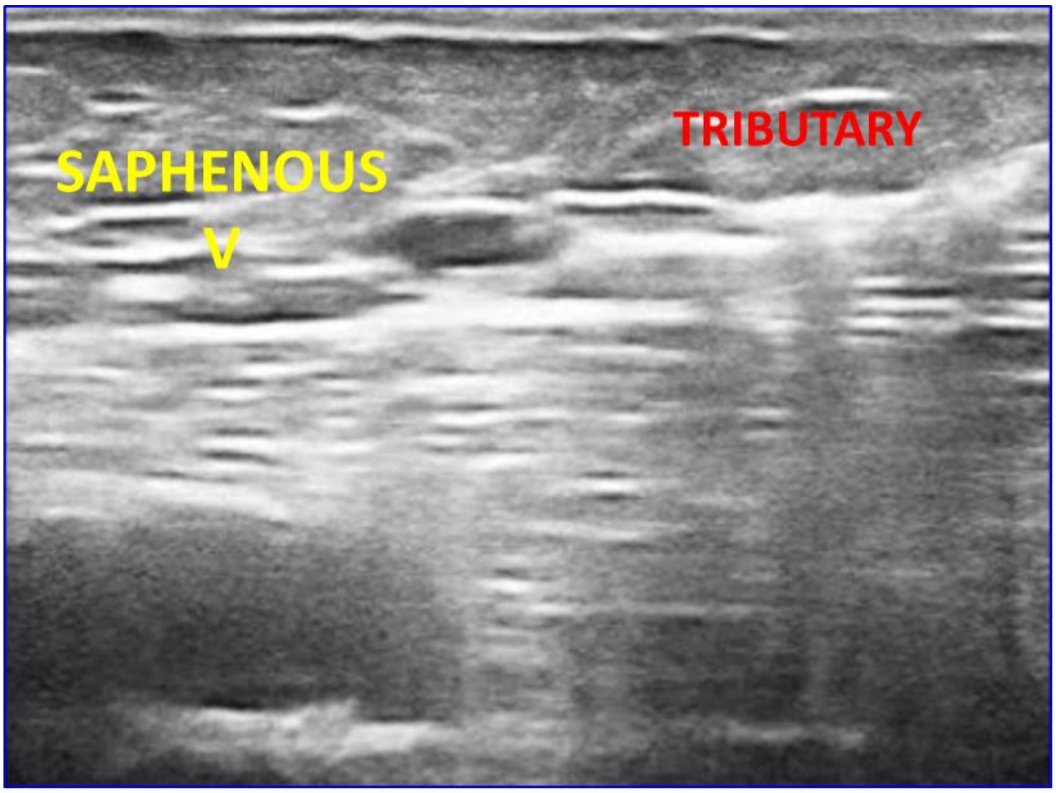


FIG 12 **SCLEROSIS OF A SAPHENOUS TRIBUTARY WITH STRAIGHT FLUSH OUTLET OF THE SAPHENOUS TRUNK**

Protection of the saphenous trunk and further slowing of foam progression can be accomplished by affixing a small sleeve, inflated to 90 mm/Hg downstream of the saphenous outlet, which is slowly and discontinuously deflated by a second operator while foam is injected as outlined above.

This guides the foam toward the outlet; once it is reached, the sleeve is re-inflated, completely stopping the foam and protecting the saphenous trunk, and it waits about two minutes before finally deflating it; this optimizes the contact time of the sclerosant with the endothelium and gives time for inactivation of the foam by the blood proteins. Upon release of the compression, we will see the foam pass into the saphenous vein, but it will be a completely unloaded foam, that is, inactive. (Fig 13)



BLOCKING MOUSSE PROGRESSION TO THE SAFENIC
FIG 13 **TRUNK WITH AN INFLATED CUFF AT 90 mm/Hg**

Eccentric compressions inside the saphenous eye can also be used, done in ultrasound, injecting cold physiological solution, deforming first the posterior wall, then the anterior one until the lumen is closed.

An intraluminal lavage with heparin solution, injected rapidly, can also be performed under ultrasound guidance in case of foam passage into the saphenous trunk.

The choice of the various injection techniques, the percentage of foam, as well as the type, will be evaluated depending on the case, since there is an anatomical variability of the outlets. Nothing is codifiable and therefore standardizable.

MATTING

Matting is an area characterized by the appearance of red telangiectasias constituted by the dilation of very distal venules of the subpapillary dermal plexus.

It can appear after any surgical or sclerotherapeutic procedure or after any traumatic process.

We can have a EARLY MATTING or a LATE MATTING.

EARLY MATTING

Venular dilatation is related to an immediate increase in TMP usually sustained by an increase in Residual Pressure due to obstruction or vasodilatation.

Increased PR by obstruction occurs whenever drainage of an area is compromised: NON-DRAINING CHIVES, FLEBECTOMY, SCLEROTERAPIC TREATMENT Ref: ("Pathogenesis of varicose veins - lessons from biomechanics" L. Pfisterer, T. Korff VASA 2014; 43: 81)

The increase in PR by vasodilation occurs due to an inflammatory process directly proportional to SURGICAL TRAUMA and SCLEROSIS REACTION

There are conditions in which the PR is already basically high and therefore the phenomenon of matting may be more frequent, such as: adipose extremities with or without associated PEF.

The treatment is waiting, giving time for the supra- and transfascial macrocirculatory reserve to activate. This process can be facilitated with subcutaneous decongestant treatments such as mesotherapy and carboxytherapy.

Never treat early matting in sclerosis, for obvious pathophysiological reasons.

LATE MATTING

It appears approximately one month or more after the treatment and is associated with a progression of incontinence of small saphenous collaterals involving branches located distal to the perforators, thus involving the 3rd-4th order of division starting from the first saphenous collateral (order 0). In this case, the increase in TMP, always at the basis of venule dilatation, is supported by hydrostatic pressure. It should be kept in mind that distally to the 3rd order of division there are completely avalvolated branches that extend to the microcirculation. ("Failure of microvenous valves in small superficial veins is a key to the skin changes of venous insufficiency Ref: " Vincent R. et Al.: J. Vasc. Surg. Vol. 54 N°19S 2011 p.: 62S-69S) (Fig 14)

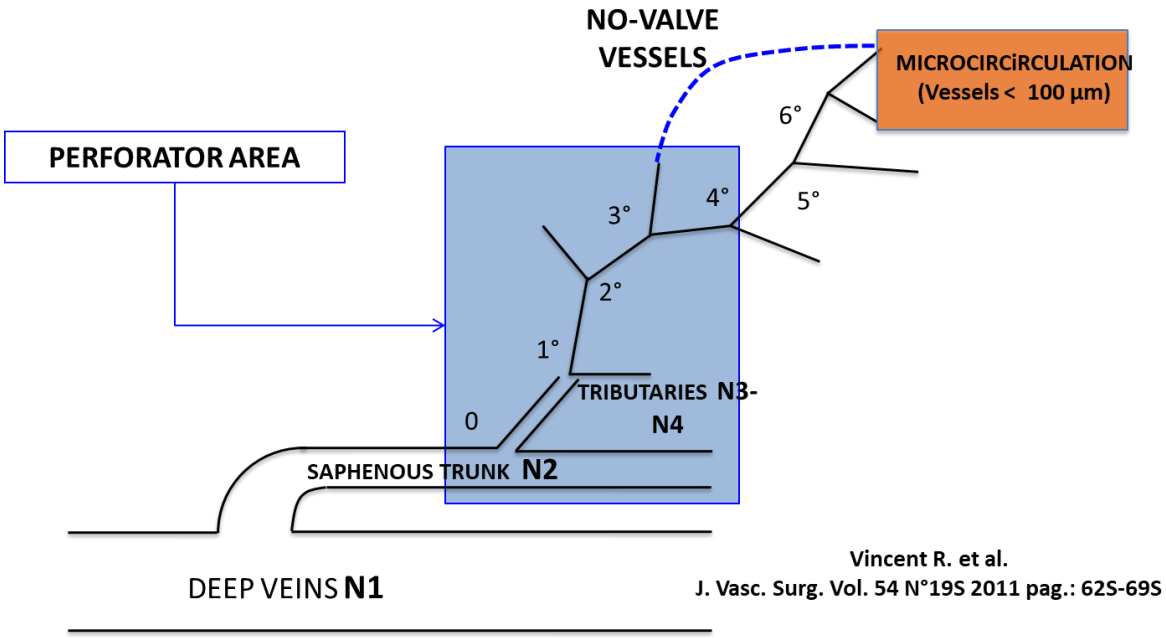


FIG 14 **DIVISION ORDER OF THE SUPERFICIAL VENOUS NETWORK**

The treatment consists first of all in identifying the tributaries anatomically related to the area of matting. Any reflux will be evident proximal to the perforators and not distal to them. We will have to search, using high-frequency probes, the identifiable vessels closest to the matting area and treat them in ultrasound using thin needles bent parallel to the skin, using mainly liquid sclerosants or wet foam in small quantities.