“Trendelenburg is history”
A modern understanding of venous disease

Dr Sriram Narayanan

Senior Consultant Vascular and Endovascular Surgeon, The Harley Street Heart & Cancer Centre

Adj Asst Prof of Surgery, National University of Singapore

Chairman, Asian Venous Forum 2016

Member, American College of Phlebology

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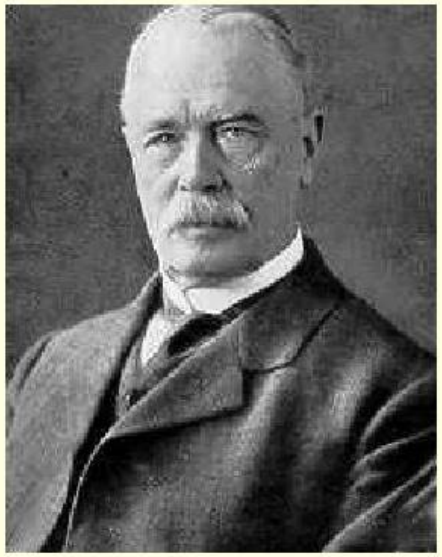
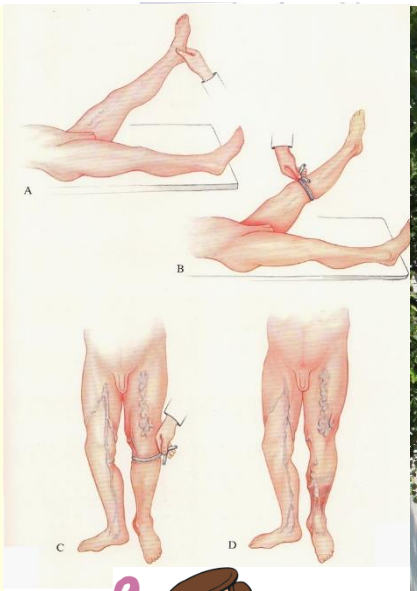
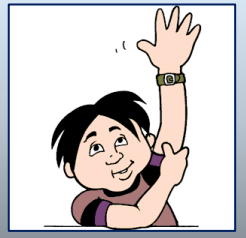
The father of venous surgery ???

Friedrich Trendelenburg (1844-1924)





5 warm up statements



Spider veins and varicose veins first need an US scan before treatment

5 warm up statements

1. Spider veins and varicose veins first need an US scan before treatment

Varicose veins are caused by valves not maintaining blood flow towards the heart

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Modern treatment of varicose veins is by laser

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Spider veins are just an aesthetic problem

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4. Spider veins are just an aesthetic problem

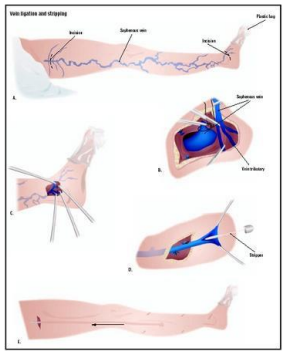
Pelvic congestion syndrome is rare and treated by gynaecologists

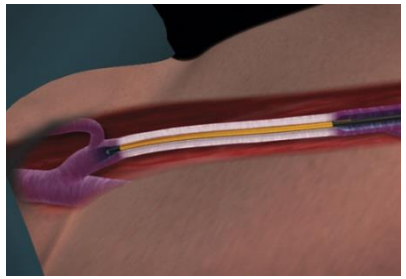
5 warm up statements

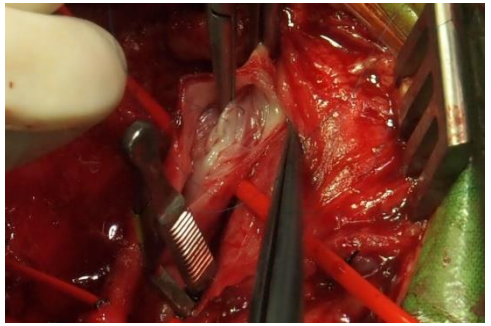
1. Spider veins and varicose veins first need an US scan before treatment
2. Varicose veins are caused by valves not maintaining blood flow towards the heart
3. Modern treatment of varicose veins is by laser
4. Spider veins are just an aesthetic problem
5. Pelvic congestion syndrome is rare and treated by gynaecologists

Why do we perform any venous intervention

- Superficial vein surgery
- Deep venous valve reconstruction
- Venous bypass
- Venous stenting
- Compression therapies







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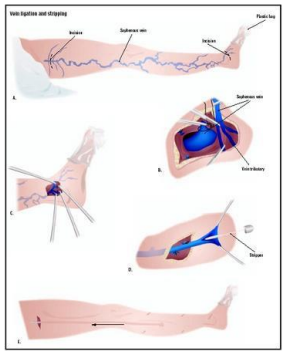

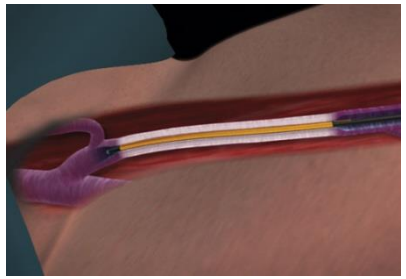

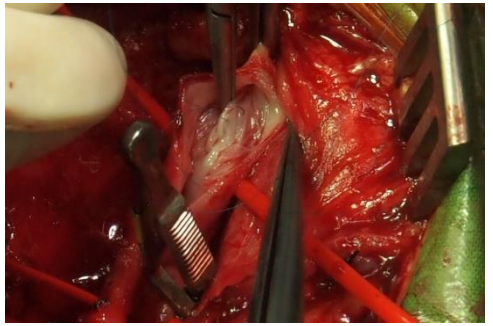

Common Aim – to reduce the ambulatory venous pressure at the ankle

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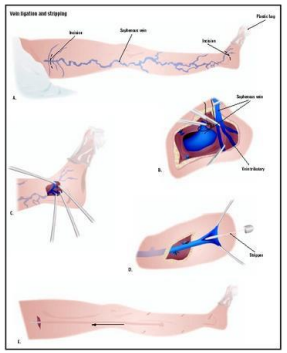

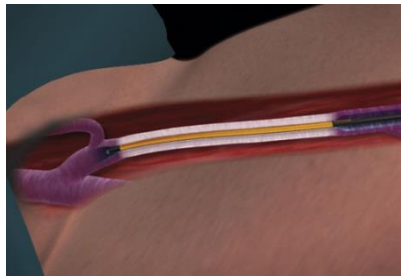

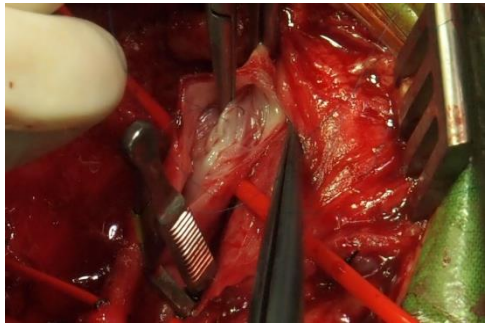

Common Aim – to reduce the ambulatory venous pressure at the ankle

And yet we never measure it ???

A paradigm shift in understanding Chronic venous disease

Eur J Vasc Endovasc Surg (2015) 49, 678–737

Editor's Choice — Management of Chronic Venous Disease

Clinical Practice Guidelines of the European Society for Vascular Surgery (ESVS)

Writing Committee [a] **C. Wittens, A.H. Davies, N. Bækgaard, R. Broholm, A. Cavezzi, S. Chastanet, M. de Wolf, C. Eggen, A. Giannoukas, M. Gohel, S. Kakkos, J. Lawson, T. Noppeney, S. Onida, P. Pittaluga, S. Thomis, I. Toonder, M. Vuylsteke,**
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Keywords: Chronic venous disease, Venous disease, Varicose veins, CEAP, VCSS, Villalta, AVVQ, Vein Qol Sym, Duplex ultrasound, Plethysmography, Phlebography, MRV, CTV, Wound dressings, Compression, Ambulatory compression, Sclerotherapy, Thermal ablation, Non-thermal ablation, Laser, Radiofrequency ablation, Stripping, High ligation, Phlebectomy, Stenting, Endophlebectomy, AV fistula, Recurrent varicose veins, Venous malformation

TABLE OF CONTENTS

3.1.2. Physical examination

Scientific evidence. Patients with CVD are examined in a physiological upright standing position. Both legs should be examined completely. When signs of severe CVD or secondary (e.g. post-thrombotic) varices are present, the abdominal region should be inspected for the possible presence of venous collaterals. Venous collaterals on the lower abdomen, flanks, and pubic region are pathognomonic of iliac or ilio-caval outflow obstruction.

Corona phlebectatica paraplantaris should be noted as this may indicate advanced venous stasis.[143]

In recurrent disease, it is important to bear in mind the patient's pre-operative state and assess any amelioration or worsening in signs such as skin changes or ulceration.

During physical examination, it is important to consider alternative pathology such as signs of arterial insufficiency, orthopaedic, rheumatological, or neurological pathology (muscle pump function). The main circumferences of both legs should be measured when indicated (e.g. phlebolymphedema, suspicion of vascular malformations).

Traditional clinical tests such as Trendelenburg, Perthes, and others have proven unreliable and have no place in the mapping of venous incompetence in general, and of varicose veins in particular.[144,145]

3.2. Diagnostic tools

3.2.1. Definition of reflux

Scientific evidence. In a study using DUS, reflux times in the various venous segments of the lower extremity were system, the deep femoral vein, and the calf veins, longer than 1 s in the common femoral, femoral vein, and popliteal vein, and longer than 0.35 s in perforating veins.[146]

An additional finding of this study is that an erect position is the only reliable way to detect reflux.[146]

Previous international consensus held 0.5 s as a cut off value in all leg vein segments, but this appears to vary with the type of venous segment. The present consensus recommends 1 s as the cut off duration for reflux in femoral and popliteal vein, whereas above 0.5 s is considered reflux in saphenous veins, lower leg veins, and perforators.[146]

The GSV, AASV, PASV, thigh extension, and SSV all situated in their saphenous compartment, are the main superficial conduits to be imaged for morphology and tested for possible reflux, and its segmental distribution.[147] Main thigh or lower leg perforators, mostly on the medial aspect of the limb, should be examined with diameters measured at fascia level. Perforators should also be tested for their inward and/or outward flow during distal calf compression (systole) and release (diastole).[147] Saphenous diameter should be measured at specific locations: the GSV 3 cm below the saphenofemoral junction, at mid-thigh, at the knee, and lower leg; the AASV 3 cm below the SFJ and at mid-thigh when still lying in its saphenous compartment; and the SSV 3 cm below the SPJ.[148] The terminal and pre-terminal valves of GSV must be tested for their function, as Cappelli demonstrated that GSV

Recommendation 7	Class	Level	References
Physical examination of patients should always be performed, looking for varicose veins, oedema, and skin changes.	I	C	140,142
Recommendation 8			
The traditional diagnostic tests Trendelenburg, Perthes, and other such tests are not recommended in the work up of patients with chronic venous disease.	III	B	144,145

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A paradigm shift in understanding Chronic venous disease

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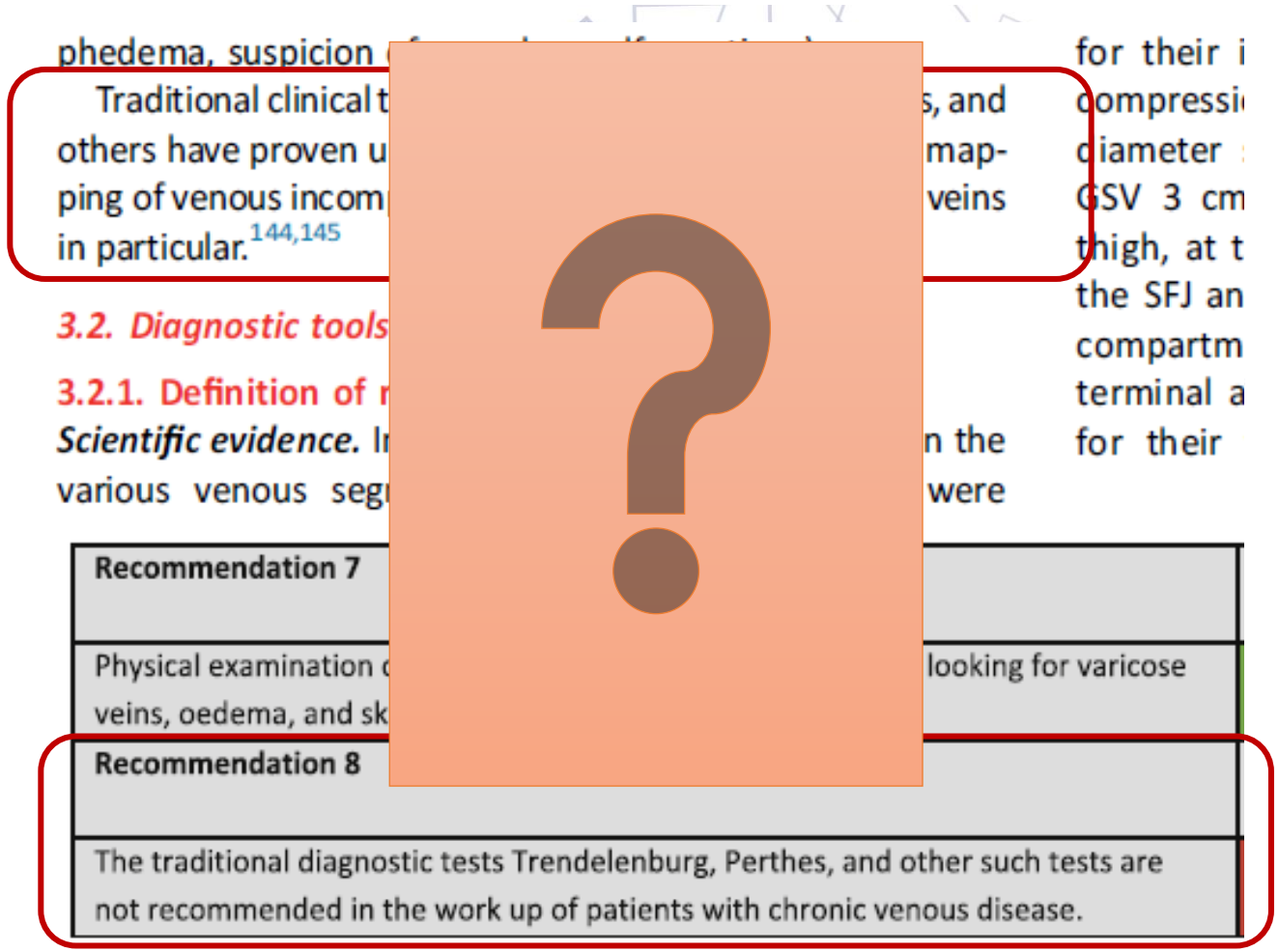
What has changed in our understanding of chronic venous disease



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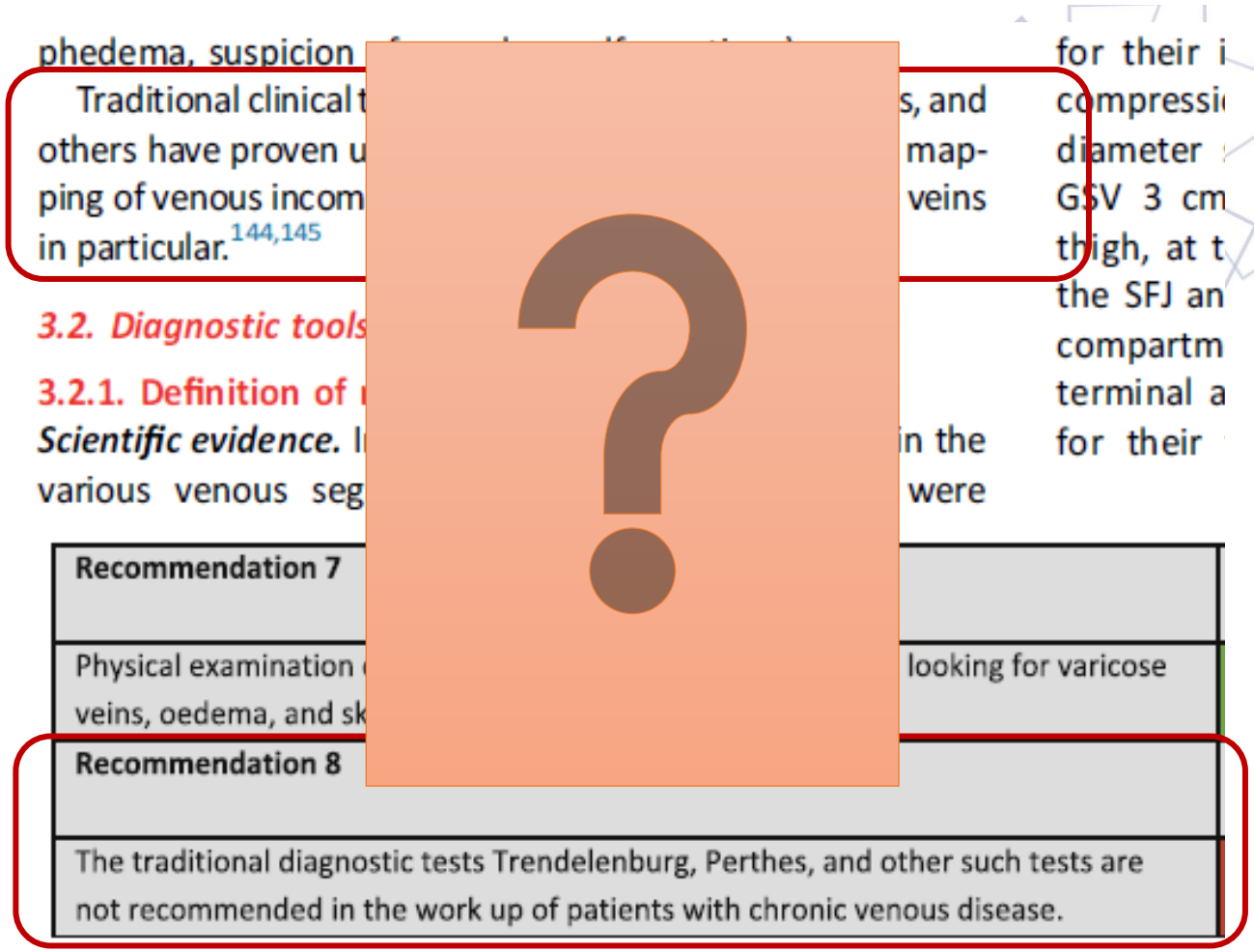

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- **The development and function of venous valves**
- **The mechanism of venous return**
- **The haemodynamics of development of CVI**

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What do valves really do ???

EDITORIAL

Venous valve incompetence: the first culprit in the pathophysiology of primary chronic venous insufficiency

by J. Bergan, USA

John BERGAN, MD

CHRONIC VENOUS INSUFFICIENCY (CVI) IS MANIFESTED IN THE lower extremities. Its most obvious sign is protuberant, saccular varicose veins. These fail to fulfill their assigned function of transporting blood from the lower extremities to the heart. Instead, they allow the weight of the column of blood from the right atrium as it is transmitted through the valveless vena cava and iliac veins to be expressed in the thigh and leg. There, the venous hyper-

- **Is incompetence the same as reflux?**
- **Is incompetence a manifestation of high outflow pressure?**
- **Do valves aid forward flow or prevent back pressure?**

Valve segmentation controls transmission of upstream pressure

J ENDOVASC SURG
1998;5:42–51

◆ EXPERIMENTAL STUDY ◆

Tube Collapse and Valve Closure in Ambulatory Venous Pressure Regulation: Studies With a Mechanical Model

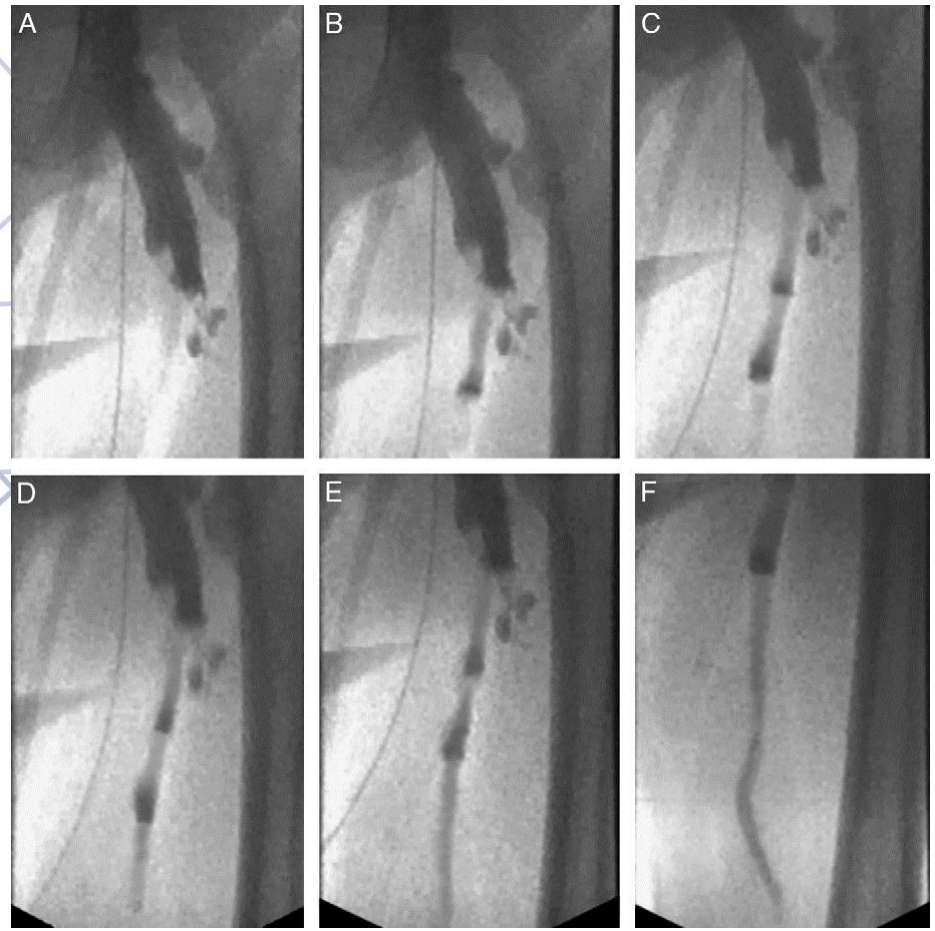
Seshadri Raju, MD; Austin B. Green, MS*; Ruth K. Fredericks, MD; Peter N. Neglen, MD, PhD; C. Alexander Hudson, MD; and Keith Koenig, PhD*

Department of Surgery, the University of Mississippi Medical Center; and the *Department of Aerospace Engineering, Mississippi State University, Jackson, Mississippi, USA

Purpose: To determine the role of valve closure and column segmentation in ambulatory venous pressure regulation.
Methods: Using a mechanical model consisting of a graduated adjustable valve and a collapsible tube, we studied the differential effects of valve closure and tube collapse on venous pressure regulation. By utilizing materials with differing wall properties for the infravalvular tube, the influence of wall property changes on tube function and pressure regulation was explored.
Results: Valve closure, per se, does not cause venous pressure reduction. Collapse of the tube below the valve is the primary pressure regulatory mechanism. The nonlinear volume-pressure relationship that exists in infravalvular tubes confers significant buffering properties to the collapsible tube, which tends to retain a near-constant pressure for a wide range of ejection fractions, residual tube volumes, and valve leaks. Changes in tube wall property affect this buffering action, at both the low and high ends of the physiological venous pressure range.
Conclusions: The valve and the infravalvular venous segment should be considered together in venous pressure regulation. Tube collapse of the segment below the valve is the primary pressure regulatory mechanism. An understanding of the hydrodynamic principles involved in pressure regulation derived from this model will provide the basis for construction of more complex models to explore clinical physiology and dysfunction.

J Endovasc Surg 1998;5:42–51

Key words: experimental study, venous valves, venous pressure, venous physiology, venous pump



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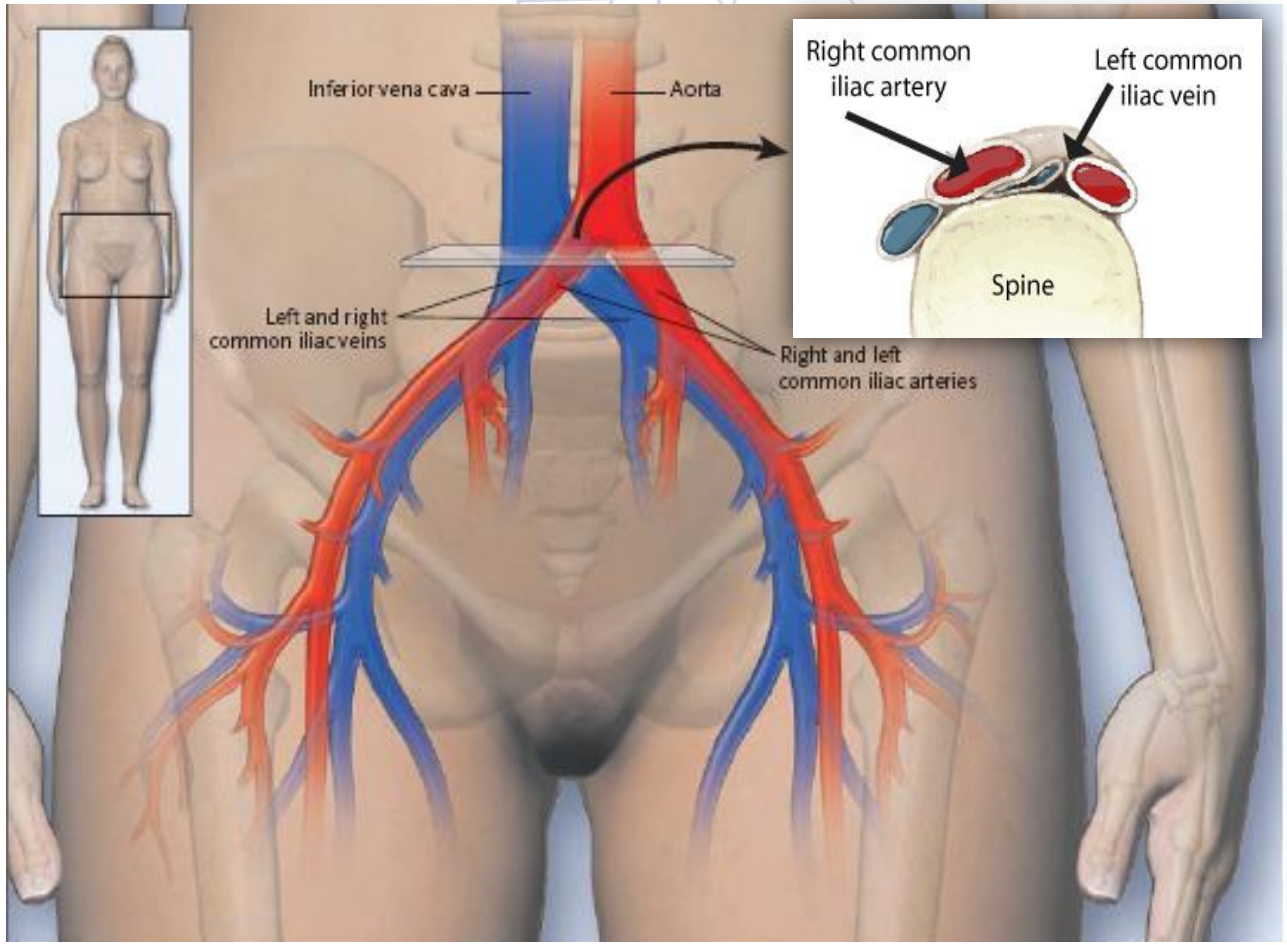
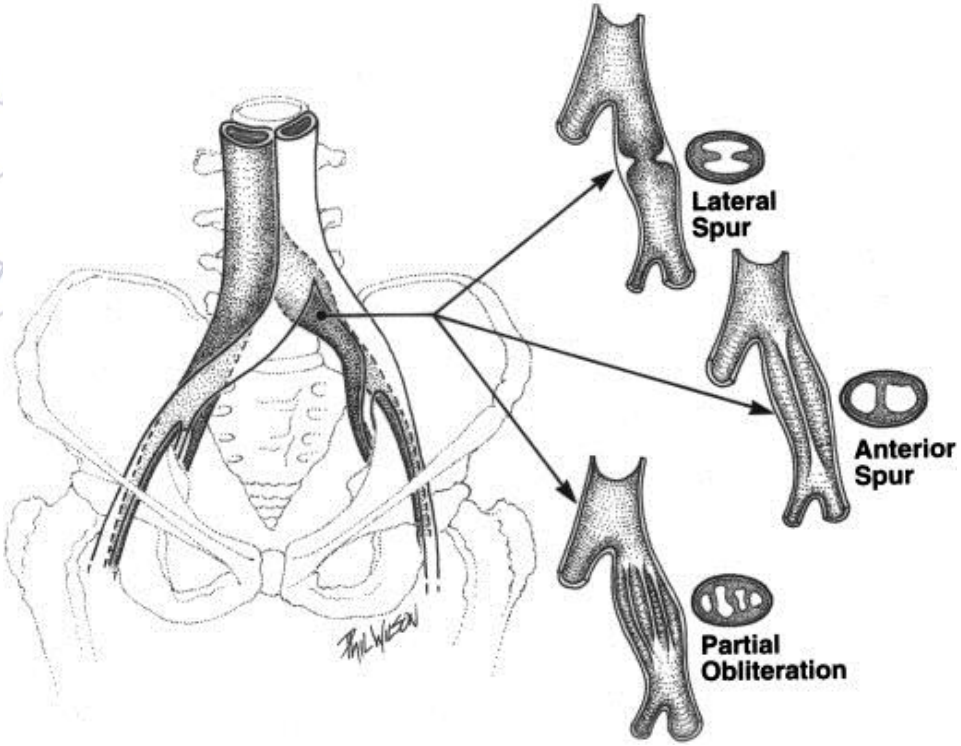
The problem of an upright posture

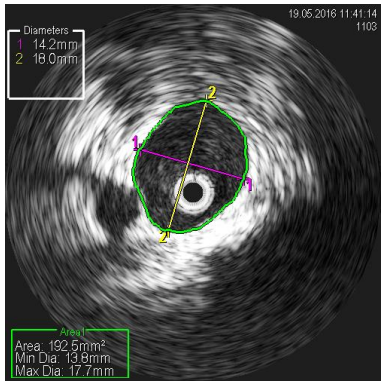
May-Thurner syndrome / Non Thrombotic Iliac Vein Lesion



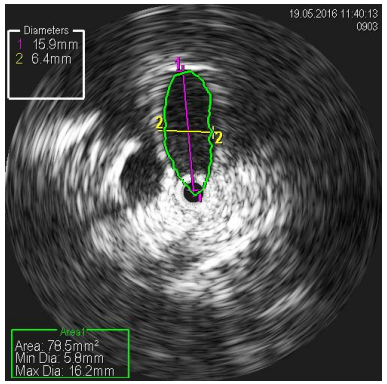
May Thurner syndrome / NIVL

- True incidence unknown
  - 22-32% cadavers
  - 18-40% in patients with left LL DVT
- May be as high as 70-90% on IVUS

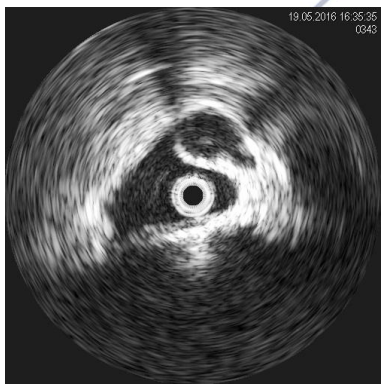




Normal CIV



NIVL



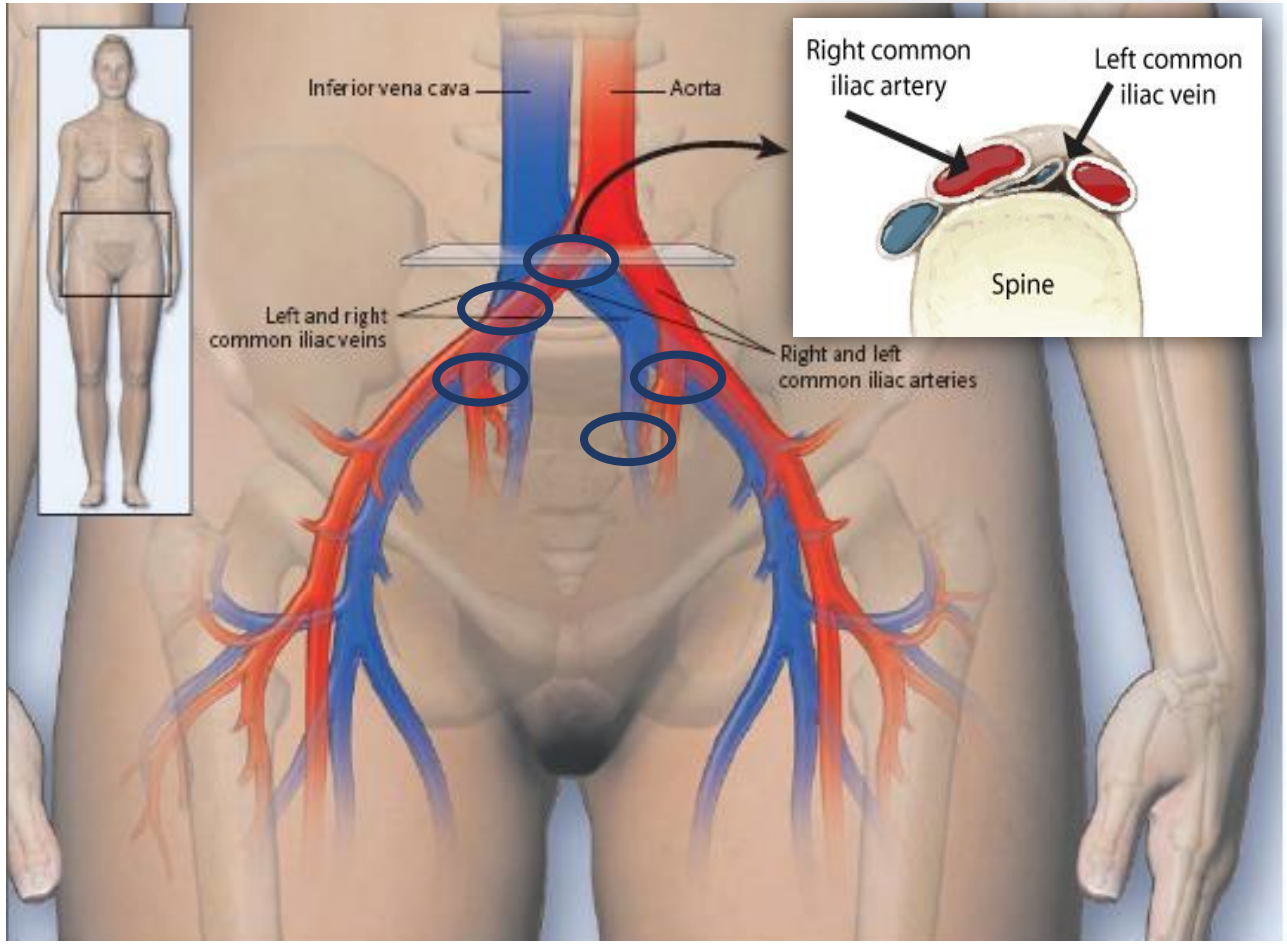
CIV compression from calcified artery

Multiple compression sites due to NIVL

May-Thurner syndrome / Non Thrombotic Iliac Vein Lesion

The 80 % story

- VVs 80 % left side
- DVT 80% left side
- Venous ulcer 80% left side
- Ovarian vein incompetence 80% left side
- **NIVL – 80% LEFT SIDE**



NIVL and the arrival of venous stenting

High prevalence of nonthrombotic iliac vein lesions in chronic venous disease: A permissive role in pathogenicity

Seshadri Raju, MD, and **Peter Neglen, MD, PhD,** *Flowood, Miss*

Purpose: Nonthrombotic iliac vein lesions (NIVL), such as webs and spurs described by May and Thurner, are commonly found in the asymptomatic general population. However, the clinical syndrome, variously known as May-Thurner syndrome, Cockett syndrome, or iliac vein compression syndrome, is thought to be a relatively rare contributor of chronic venous disease (CVD), predominantly affecting the left lower extremity of young women. The present study describes the much broader disease profile that has emerged with the use of intravascular ultrasound (IVUS) scanning for diagnosis and analyzes stent placement outcome in two specific NIVL subsets that may offer clues to their pathogenic role.
Methods: Among 4026 patients with CVD symptoms spanning the range of CEAP clinical classes, IVUS examinations were selectively done in severely symptomatic patients for indications as described. Iliac vein obstructive lesions were found in 938 limbs of 879 patients; 53% of the limbs had NIVL, 40% were post-thrombotic, and 7% were a combination. Stents were placed in 332 limbs in 319 patients in two NIVL subsets. The subsets, one with and one without associated distal limb reflux, were compared. Reflux was left untreated in the first subset.
Results: The median age was 54 years (range, 18 to 90 years). The female–male ratio was 4:1 and the left–right ratio was 3:1. NIVL lesions in the iliac vein occurred at the iliac artery crossing (proximal lesion) and also at the hypogastric artery crossing (distal lesion), a new IVUS finding. Venography was only 66% sensitive, with 34% of venograms appearing "normal." IVUS had a diagnostic sensitivity of >90%. The cumulative results observed at 2.5 years after stent placement in the NIVL subsets with reflux and without reflux, respectively, were complete relief of pain 82% and 77%, complete relief of swelling 47% and 53%, complete stasis ulcer healing 67% and 76%, and overall clinical relief outcome 75% and 79%. These results are nearly identical between the two subsets even though distal reflux remained uncorrected in the NIVL plus reflux subset.
Conclusions: NIVL has high prevalence and a broad demographic spectrum in patients with CVD. Similar lesions in the asymptomatic general population may be permissive of future development of CVD. Stent placement alone, without correction of associated reflux, often provides relief. (J Vasc Surg 2006;44:136-44.)

Unexpected major role for venous stenting in deep reflux disease

Seshadri Raju, MD,[a] **Rikki Darcey, BS,**[b] and **Peter Neglén, MD, PhD,**[b] *Jackson and Flowood, Miss*

Background: Treatment of chronic venous insufficiency (CVI) has largely focused on reflux. Minimally-invasive techniques to address superficial and perforator reflux have evolved, but correction of deep reflux continues to be challenging. The advent of intravascular ultrasound (IVUS) scan and minimally invasive venous stent technology have renewed interest in the obstructive component in CVI pathophysiology. The aim of this study is to assess stent-related and clinical outcomes following treatment by iliac venous stenting alone in limbs with a combination of iliac vein obstruction and deep venous reflux.
Methods: A total of 528 limbs in 504 patients, ranging in age from 15 to 87, underwent IVUS-guided iliac vein stent placement to correct obstruction over an 11-year period. The etiology of obstruction was nonthrombotic in 196 (37%), post-thrombotic in 285 (54%) limbs, and combined in 47 (9%). Clinical severity class of CEAP was C_3 in 44%, $C_{4,5}$ in 27%, and C_6 in 25% of stented limbs. Deep venous reflux was present in all limbs, associated with superficial and/or perforator reflux in 69%. Reflux was severe in 309/528 (59%) limbs (reflux multisegment score ≥3) and 224/528 (42%) limbs had axial reflux. Venography and other functional tests had poor diagnostic sensitivity to detect obstruction, which was ultimately diagnosed by IVUS. The IVUS-guided iliac vein stenting was the only procedure performed and the associated reflux was left uncorrected.
Results: There was no mortality; morbidity was minor. Cumulative secondary stent patency was 88% at 5 years; no stent occlusions occurred in nonthrombotic limbs. Cumulative rates of limbs with healed active ulcers, freedom of ulcer recurrence in legs with healed ulcers (C_5), and freedom from leg dermatitis at 5 years were 54%, 88%, and 81%, respectively. Cumulative rate of substantial improvement of pain and swelling at 5 years was 78% and 55%, respectively. Quality of life improved significantly. Reflux parameters did not deteriorate after stenting.
Conclusion: Iliac venous stenting alone is sufficient to control symptoms in the majority of patients with combined outflow obstruction and deep reflux. Partial correction of the pathophysiology in limbs with multisystem or multilevel disease can provide substantial symptom relief. Percutaneous stent technology in concert with other minimally-invasive techniques to address superficial and/or perforator reflux offers such partial correction in limbs with advanced CVI and complex venous pathology. Open correction of obstruction or reflux is now required only infrequently as a "last resort". (J Vasc Surg 2010;51:401-9.)

Key research from Raju and Neglen

Journal of
Vascular Surgery
Venous and Lymphatic Disorders™

BASIC RESEARCH STUDIES

Hemodynamics of "critical" venous stenosis and stent treatment

Seshadri Raju, MD,[a,*] Orla Kirk, BS,[a] Micah Davis, BS,[a] and Jake Olivier, PhD,[b] *Jackson, Miss; and Sydney, New South Wales, Australia*

Background: The concept of "critical" stenosis at which there is a sharp reduction in forward flow is derived from arterial disease. The critical element in venous stenoses is upstream pressure, not downstream flow. Many venous symptoms and microvascular injury are related to venous hypertension. We studied the effect of venous stenosis on upstream pressure using a mechanical model and with clinical measurements after stenting of iliac vein segments (common and external).
Methods: The experimental model consisted of a Starling Resistor – Penrose tubing enclosed in a pressurized plastic chamber to simulate abdominal venous flow. Clinical measurements included time-averaged velocity, area, rate of flow, and quantified phasic flow volume in the common femoral vein before and after iliac vein stenting. Traditional air plethysmography and occlusion plethysmography were also performed.
Results: The mechanical model showed that upstream pressure varied based on (1) volume of venous inflow, (2) abdominal pressure, (3) outflow pressure, and (4) outflow stenosis. Upstream pressure changes were inverse to flow as kinetic energy was converted to pressure as required. A venous stenosis of as little as 10% raised upstream pressure in the model when the abdominal pressure was low, but high grades of stenosis had no contribution when abdominal pressure was high. Stenting of the Penrose moderated or nullified upstream pressure changes related to abdominal pressure. There was significant decompression of the common femoral vein, implying pressure reduction after stenting; median area reduction was 15% and 10% in erect and supine, respectively. Air plethysmography showed improvement in venous volume and in other parameters in confirmation of venous decompression. There was significant prolongation of phasic flow duration and quantitative phasic flow increased (median, 16%) after stenting in the erect position. There was no increase in arterial inflow.
Conclusions: The criticality of iliac vein stenosis is based on peripheral venous hypertension, which is controlled by more confounding factors than in arterial stenosis. The experimental model clarifies the interplay of the many variables. Clinical measurements indicate that iliac vein stenting results in decompression of the limb veins and, by inference, a reduction in venous pressure. Venous flow is improved less consistently and, in part, is related to an increase in duration of phasic flow. Limb arterial flow is not increased, and the venous flow changes are likely the result of rearrangement of the velocity and pressure components of venous flow. (J Vasc Surg: Venous and Lym Dis 2014;2:52-60.)

Clinical Relevance: Clinical features of iliac vein stenosis are related to peripheral venous hypertension. The interplay of the many factors that influence peripheral venous pressure and hence the "criticality" of iliac venous stenoses are clarified using an experimental venous model. The beneficial effects of iliac vein stenting are related to peripheral venous decompression as detailed in duplex flow and plethysmographic studies in stented limbs. These insights may be useful in assessing individual patients with iliac vein stenosis for stent correction.

THE HARLEY STREET HEART & CANCER CENTRE

Understanding venous return and valve function

David S. Warner, M.D., and Mark A. Warner, M.D., Editors

Anesthesiology 2008; 108:735–48

Venous Function and Central Venous Pressure

A Physiologic Story

*Simon Gelman, M.D., Ph.D.**

The veins contain approximately 70% of total blood volume and are 30 times more compliant than arteries; therefore, changes in blood volume within the veins are associated with relatively small changes in venous pressure. The terms *venous capacity*, *compliance*, and *stressed* and *unstressed volumes* are defined. Decreases in flow into a vein are associated with decreases in intravenous pressure and volume, and *vice versa*. Changes in resistance in the small arteries and arterioles may affect venous return in opposite directions; this is explained by a two-compartment model: compliant (mainly splanchnic veins) and noncompliant (nonsplanchnic veins). Effects of intrathoracic and intraabdominal pressures on venous return and central venous pressure as well as the value of central venous pressure as a diagnostic variable are discussed.

IN the era of genetic revolution and exciting discoveries in molecular mechanisms of diseases, the systems' physiologic relation within the venous system rather than on molecular and biochemical mechanisms of smooth muscle contraction and relaxation of the venous walls. Finally, the article will address the question of usefulness (or lack of it) of the CVP as a clinical guide for physiologic diagnoses and therapeutic interventions.

Function of the Venous System

The main functions of the venous system are to return blood to the heart from the periphery and to serve as a capacitance to maintain filling of the heart. Veins contain approximately 70% of total blood volume compared with 18% in arteries and only 3% in terminal arteries and

RESEARCH COUNCILS UK

Gateway to Research

Innovate UK

Factors responsible for venous valve development and disease

Lead Research Organisation: King's College London
Department Name: Cardiovascular
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- Overview
- Organisations
- People
- Publications
- Outcomes

Abstract

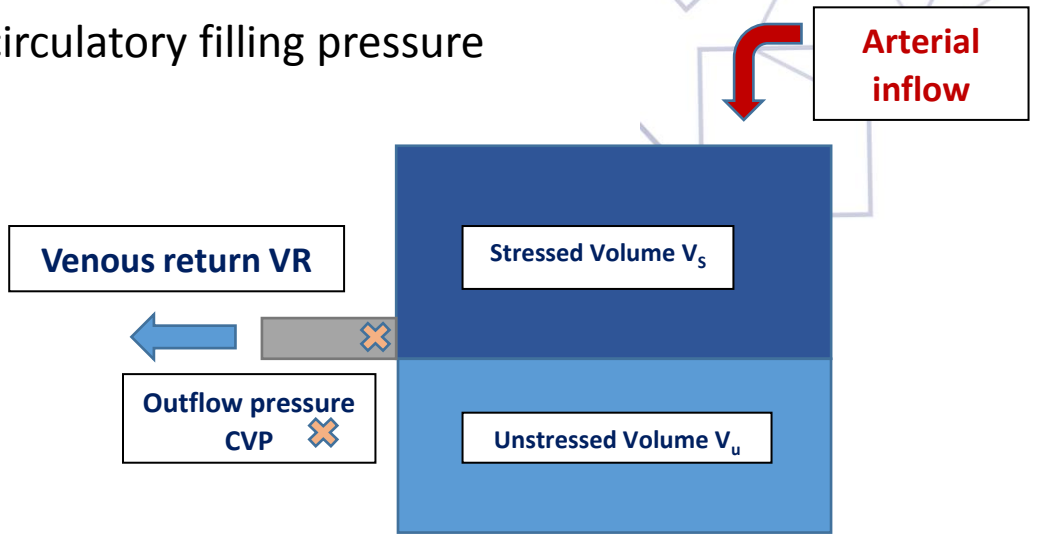
Funding details

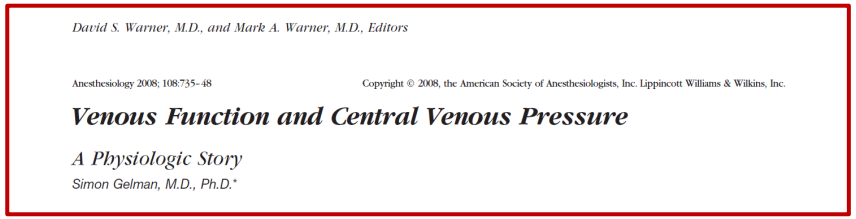
Varicose veins are a very common condition in which malfunction of the valves within the veins leads to high blood pressure in the leg veins. This damages the skin and can cause repeated leg ulceration. We know very little about how the valves in veins grow, and this makes it difficult to tell exactly what problems with the valves could cause varicose veins - it could be a problem with the vein wall or with the valves themselves. This project will tell us not only how the valves in veins develop, but also what genes control the process and their interaction with factors such as blood flow. We will study the structure of the venous valves in people who have a faulty gene that causes varicose veins. We hope to find out something about the causes of varicose veins. This understanding could allow us to develop better treatments such as replacements for faulty venous valves, or even prevent them from occurring in the first place.

The hemodynamics of venous return

$$VR = \frac{MCFP - CVP}{\text{Venous resistance}}$$

MCFP – mean circulatory filling pressure





David S. Warner, M.D., and Mark A. Warner, M.D., Editors

Anesthesiology 2008; 108:735–48 Copyright © 2008, the American Society of Anesthesiologists, Inc. Lippincott Williams & Wilkins, Inc.

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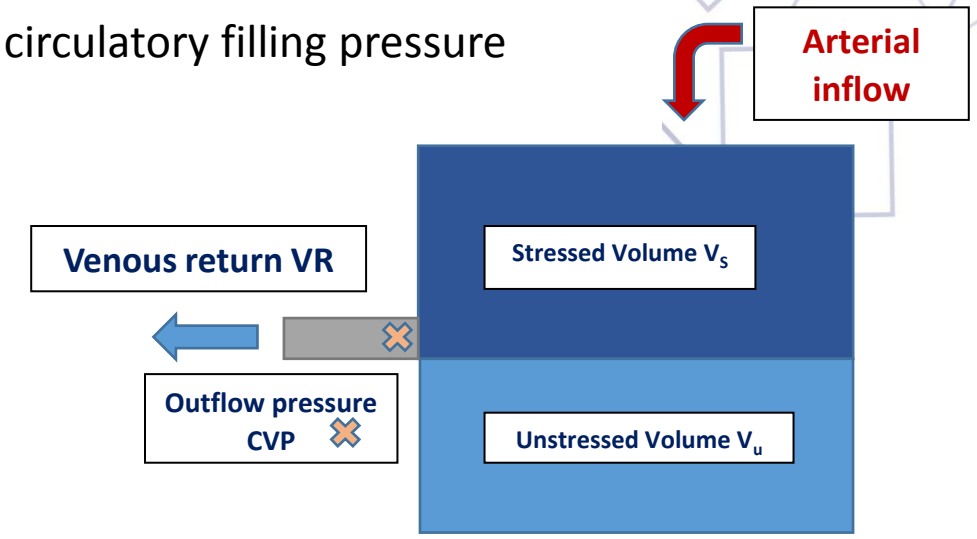
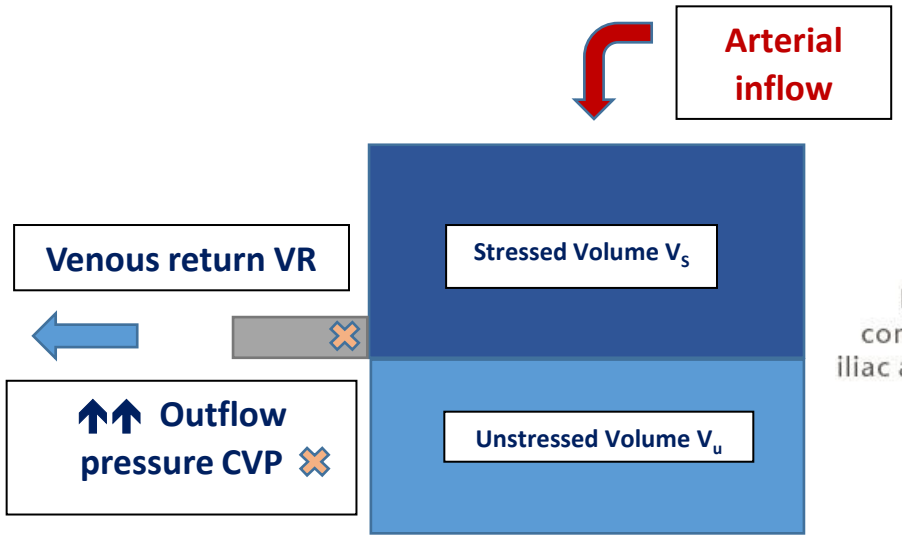


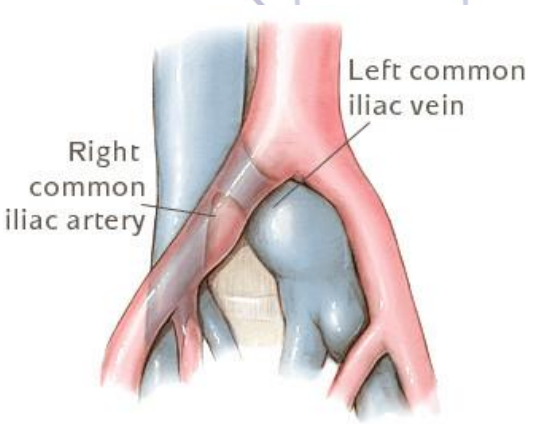
FOR THE LIMB

- MCFP – mainly Vs – deep system
- Vu is superficial system
- CVP_{limb} is outflow pressure
- Venous resistance – constant unless
  - obesity
  - fibrosis
  - obstruction (thrombus)

The hemodynamics of venous return with NIVL

$$VR = \frac{MCFP - CVP}{\text{Venous resistance}}$$





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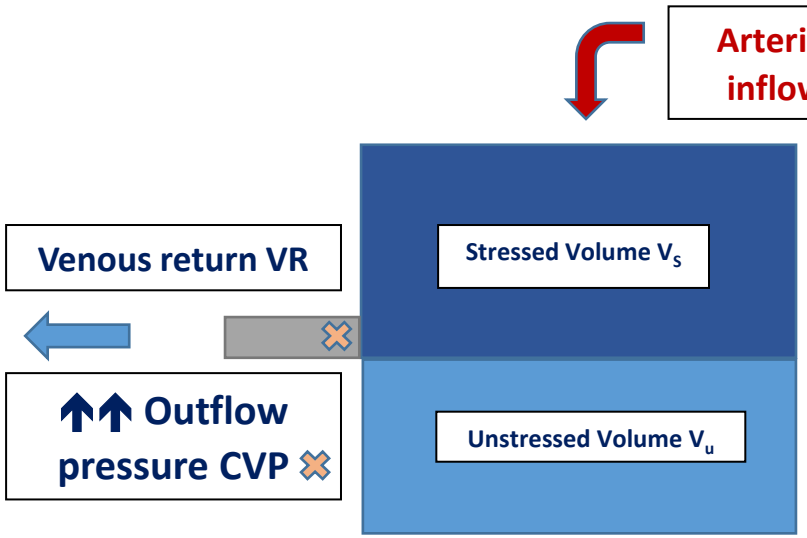
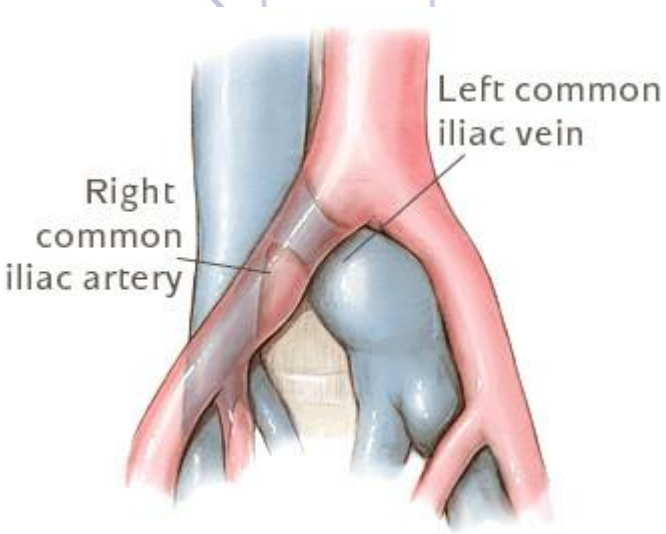
The hemodynamics of venous return – responding to an NIVL

$$VR = \frac{MCFP - CVP}{\text{Venous resistance}}$$



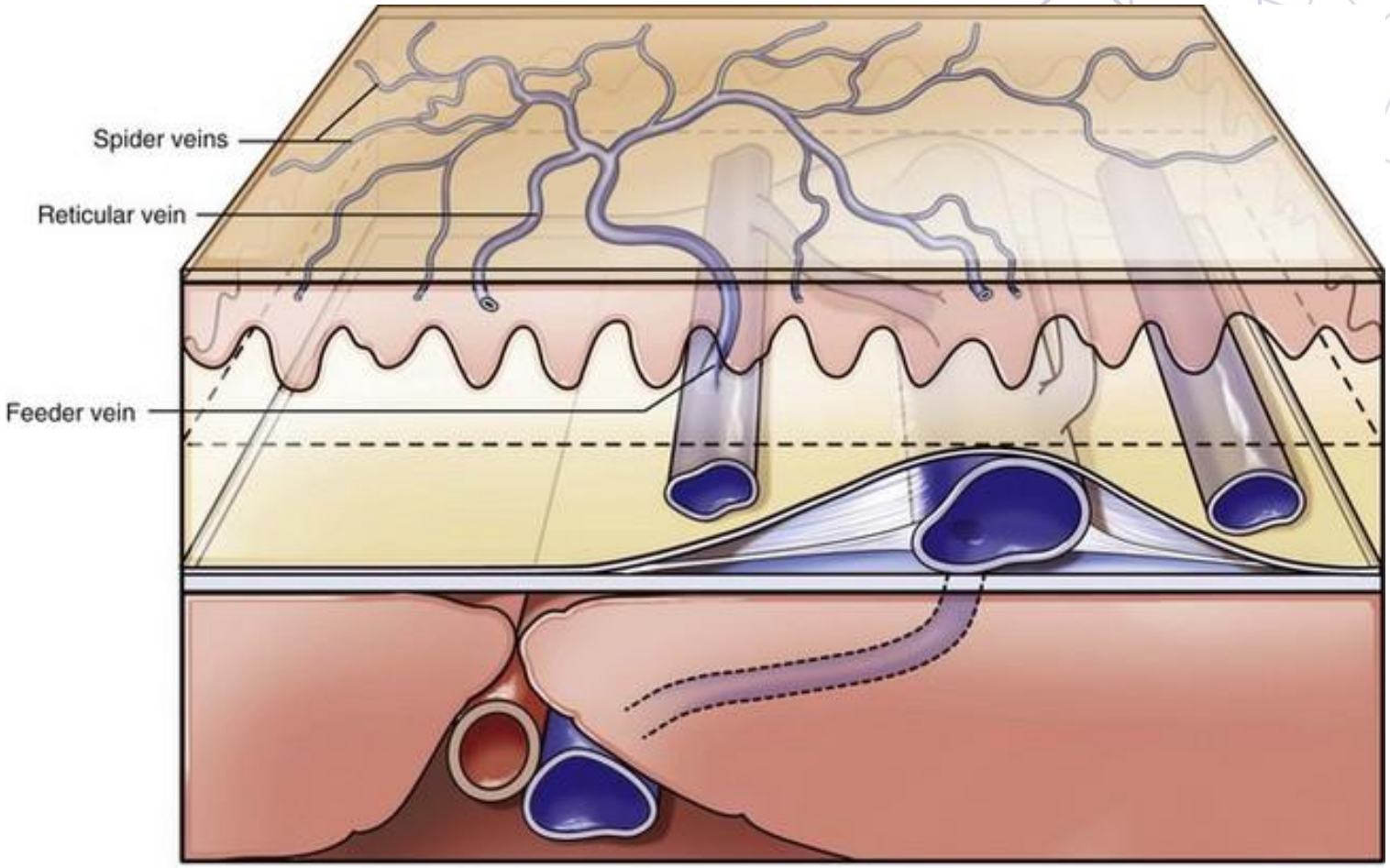


Compensating for the raised CVP_{limb}

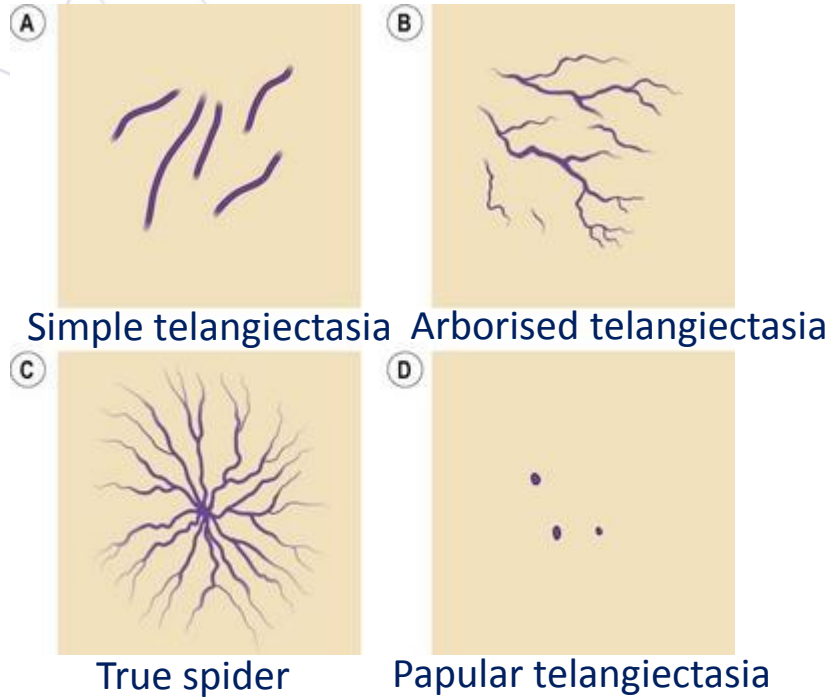
1. MCFP – pressure of blood in deep system has to rise to preserve VR
   CVI

2. System has to accept decreased VR and divert excess volume into V_u
   VARICOSE VEINS

The anatomy of a spider



< 1mm – telangiectasia - **RED**
1-3 mm – reticular veins – **BLUE GREEN**



Simple telangiectasia
Arborised telangiectasia
True spider
Papular telangiectasia

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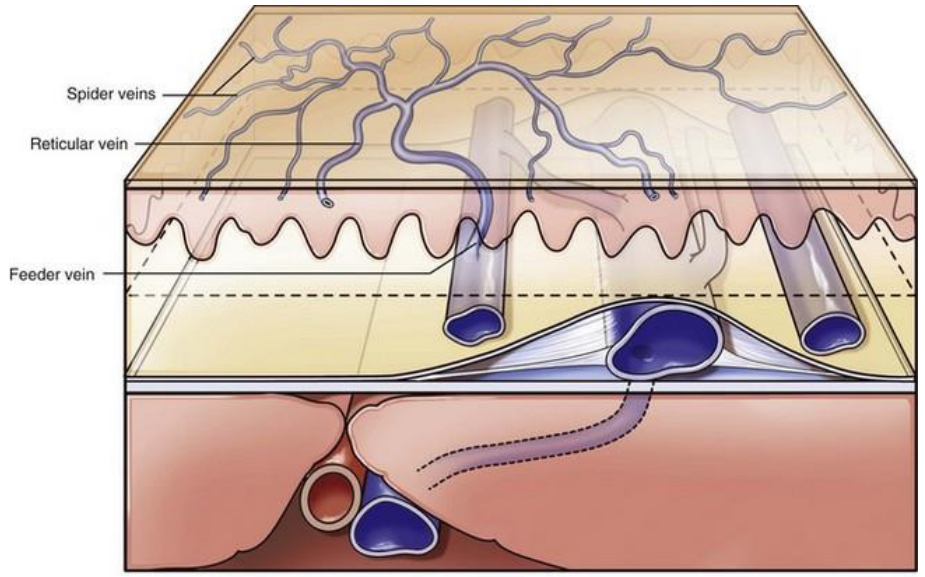
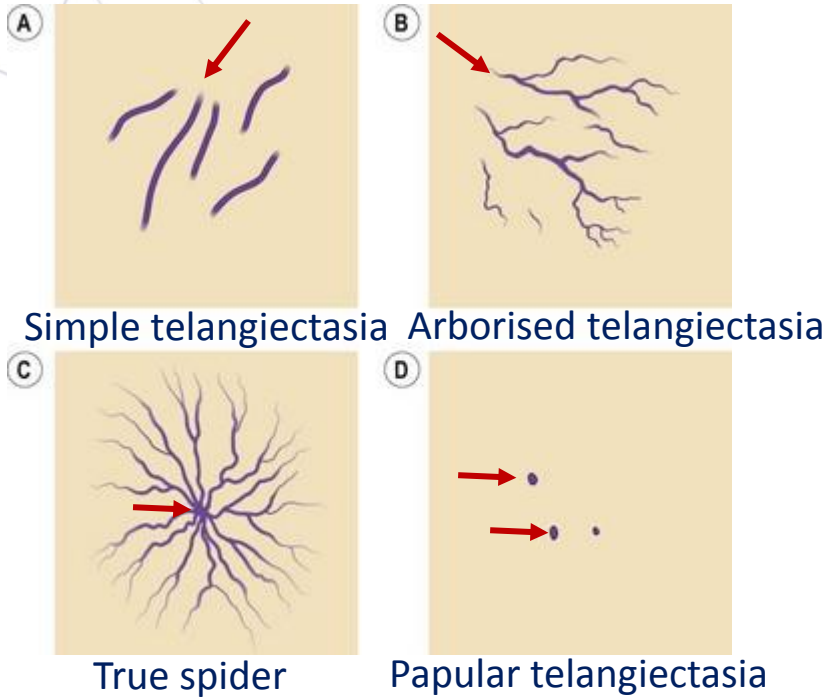
Microsclerotherapy of spider veins



< 1mm – telangiectasia – **Low volume low conc.**
1-3 mm – reticular veins – **Higher conc. but Foam if possible**



Simple telangiectasia
Arborised telangiectasia
True spider
Papular telangiectasia

Always rule out underlying venous hypertension – hemodynamic study

Treat truncal incompetence first if hemodynamics positive– 60%

Pure Aesthetic – oestrogen induced – 30-40%

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Pelvic congestion syndrome – is pelvic venous hypertension

Primary PCS – 10%

Increase in ovarian, uterine and pelvic vein volume due to

- Multiple pregnancies
- Estrogenic effect

Secondary PCS – 90%

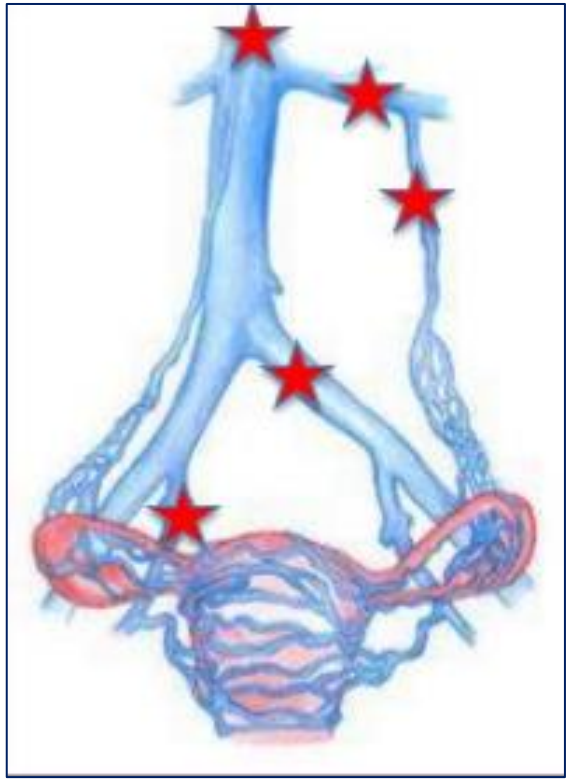
Venous outflow obstruction from

- NIVL
- Retro-aortic left renal vein
- Nutcracker phenomenon

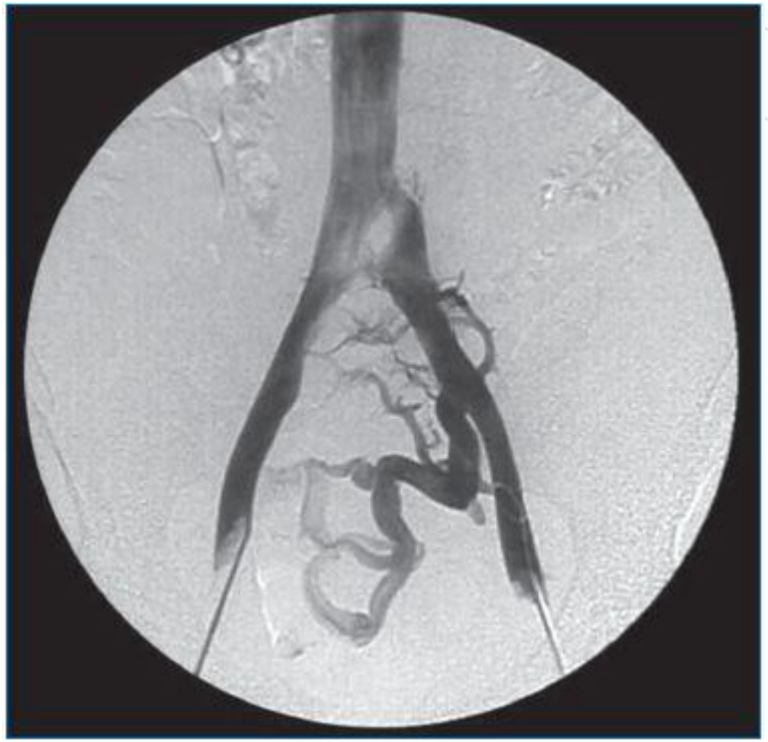
Affects 10-15% of women in their lifetime *
Pelvic pain, congestive dysmenorrhoea, dysfunctional bleeding, dyspareunia

** Jamieson D, Steege J. The prevalence of dysmenorrhea, dyspareunia, pelvic pain, and irritable bowel syndrome in primary care practices. Obstet Gynecol. 1996;87:55-58.*

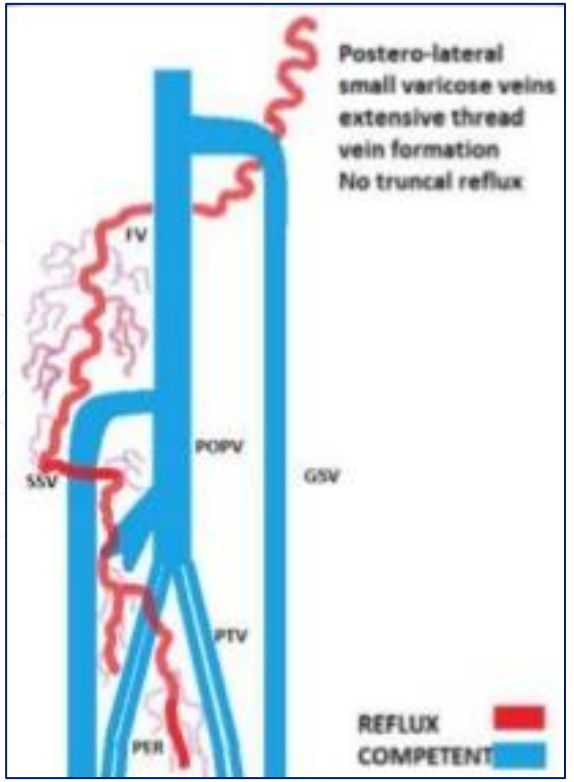
Pelvic congestion syndrome – is pelvic venous hypertension



Venous compression sites causing PCS

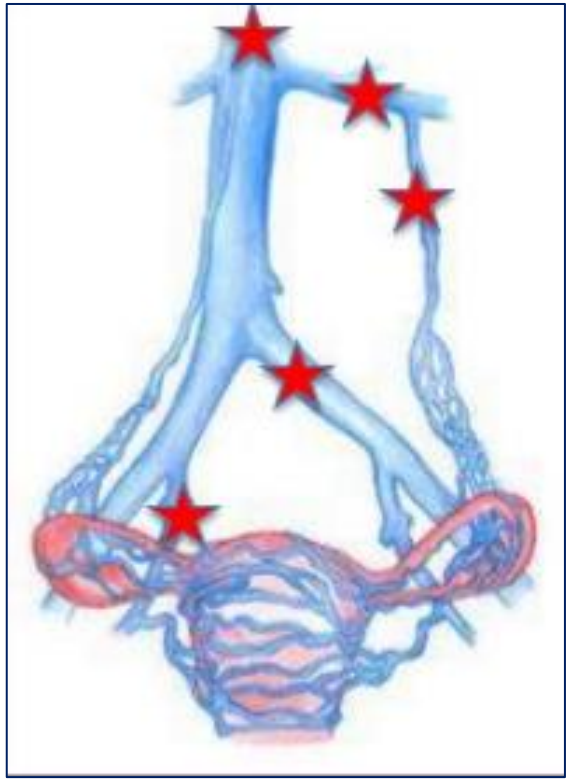


Left NIVL causing PCS



Abnormal reflux pattern in PCS

Pelvic congestion syndrome – is pelvic venous hypertension



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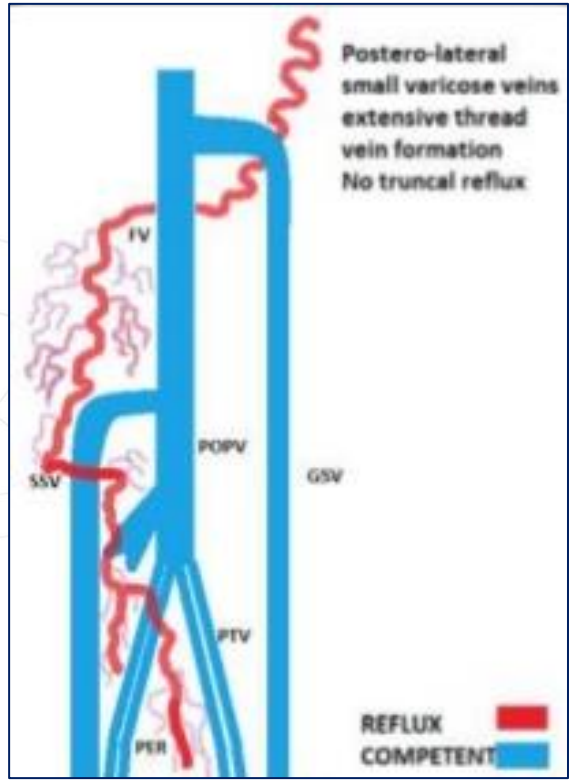

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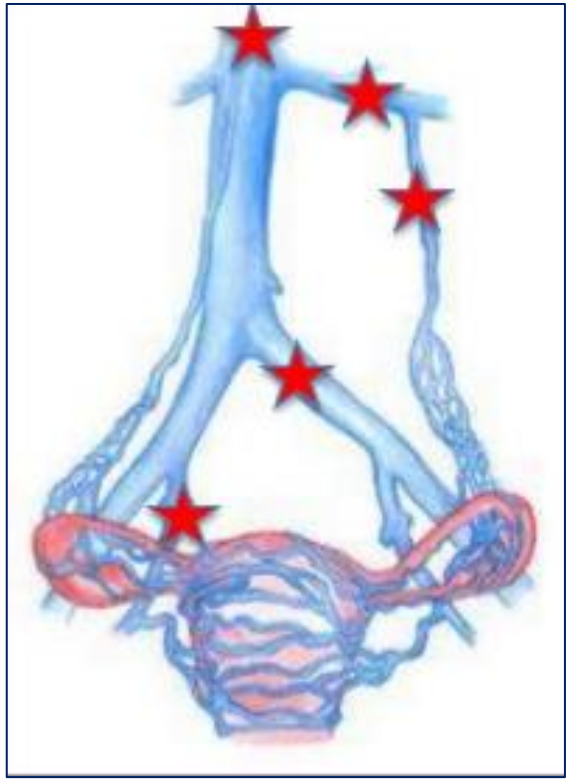

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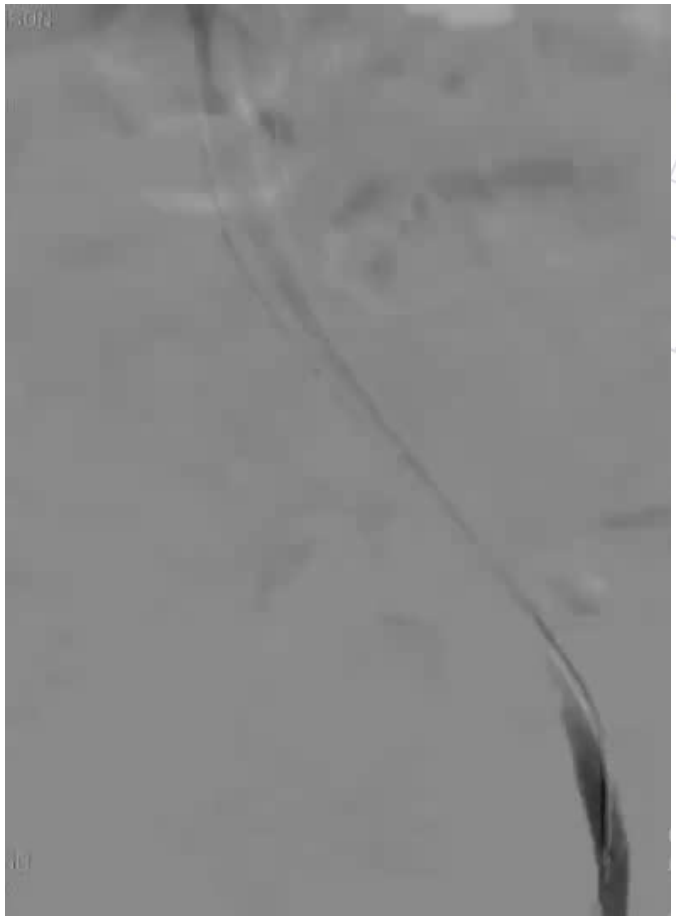
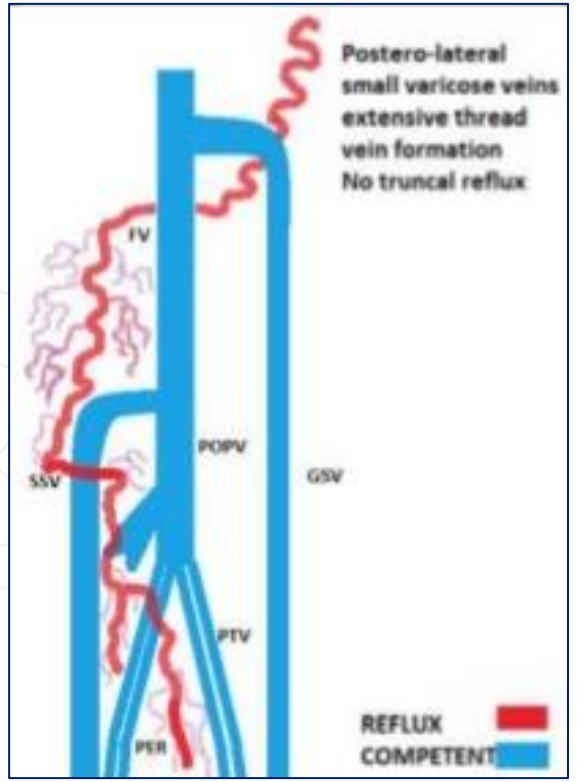
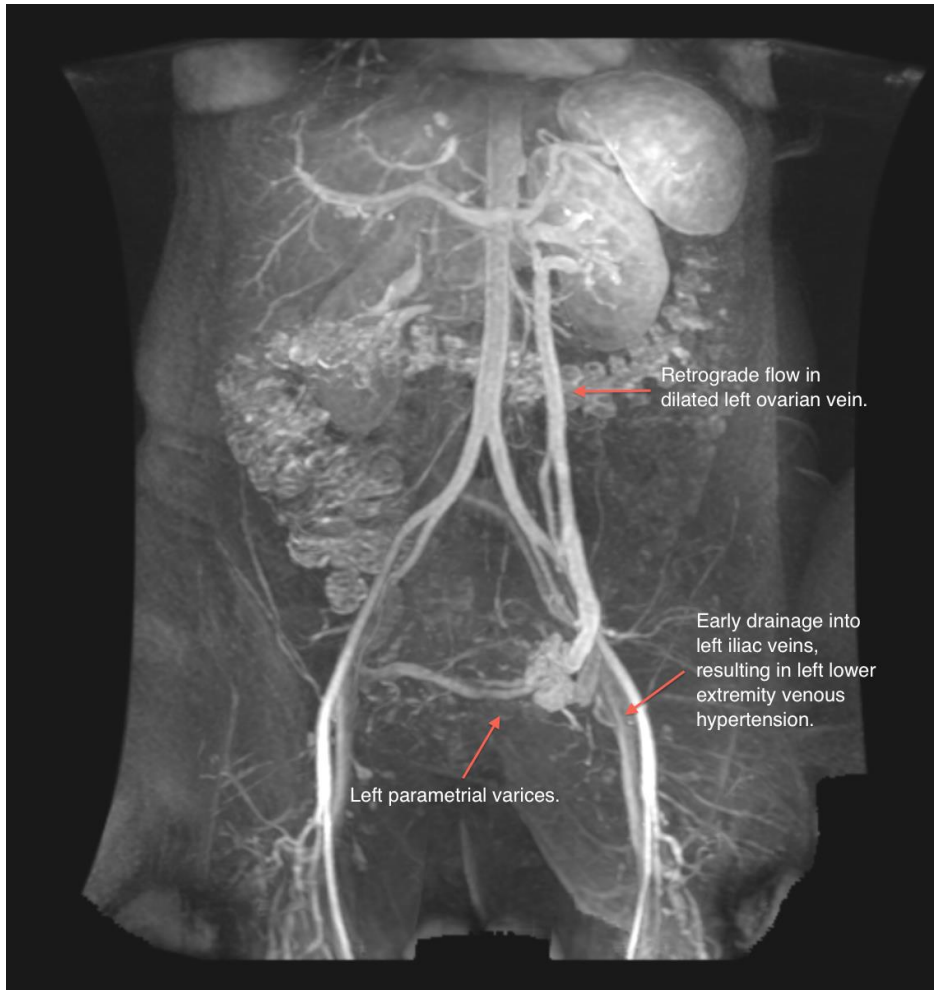


NIVL causing PCS – post stenting



Abnormal reflux pattern in PCS

Pelvic congestion syndrome – the full picture



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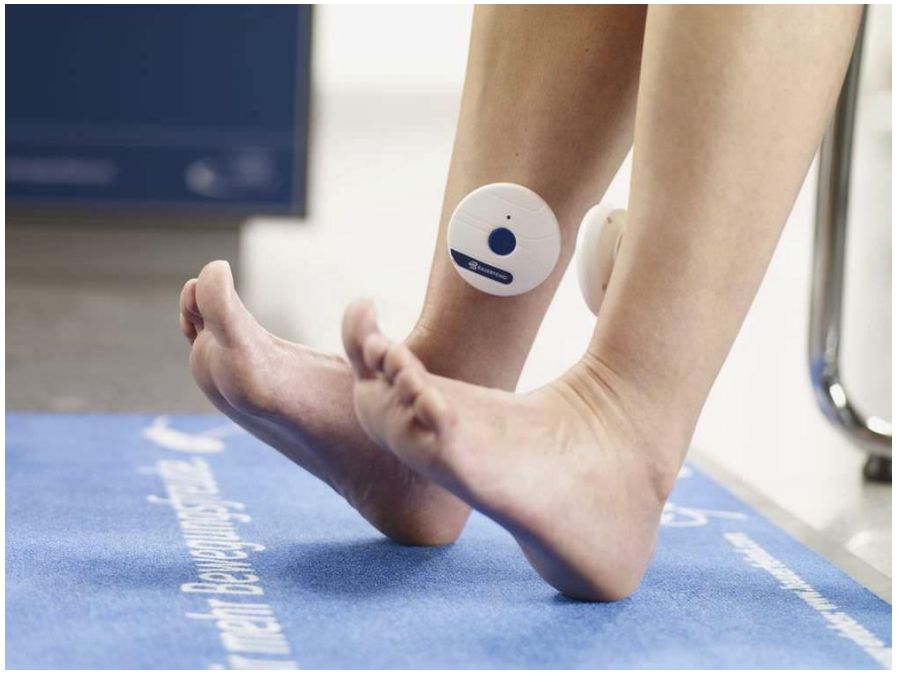
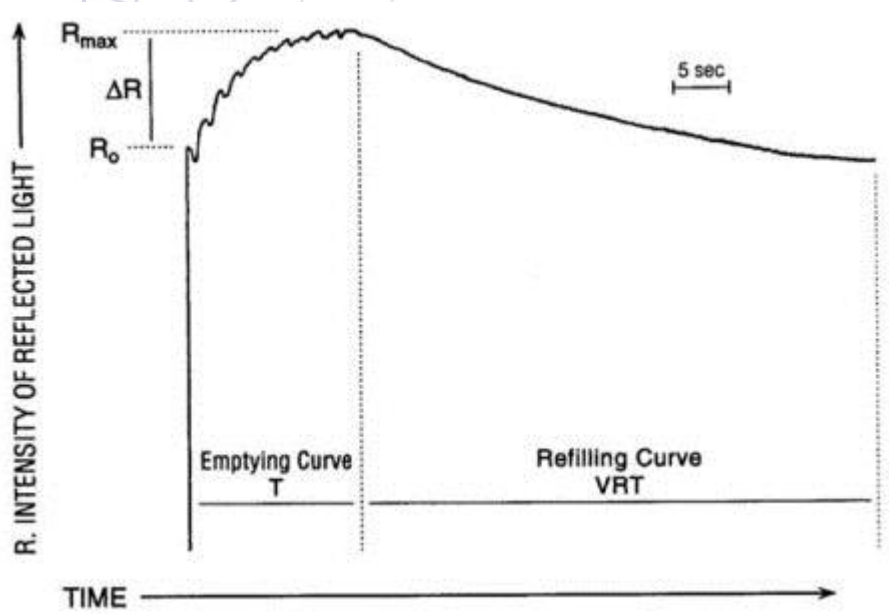
THE HARLEY STREET HEART & CANCER CENTRE

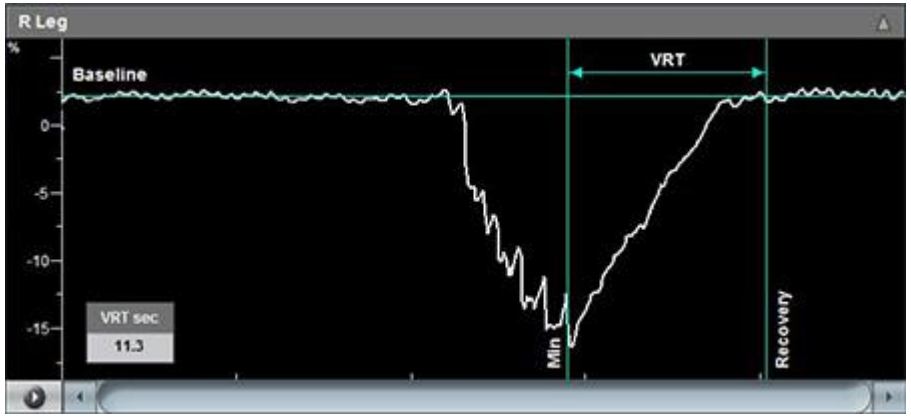
Venous hemodynamic assessment – Light reflex rheography



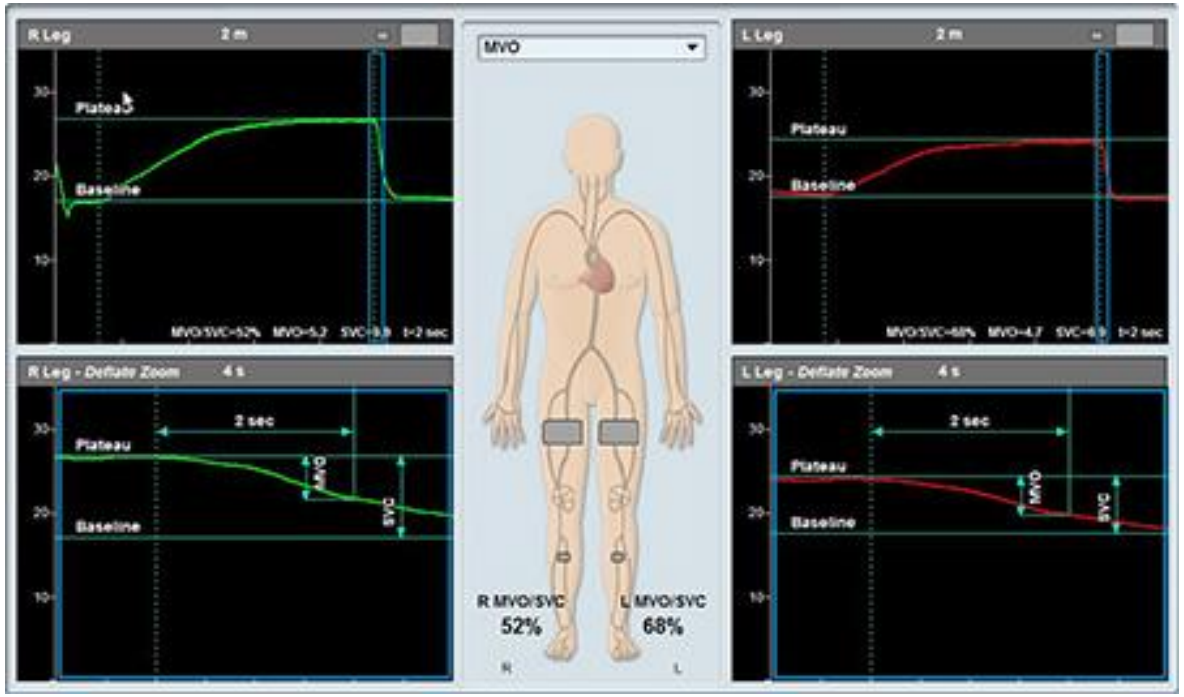


Simple baseline screening test
Assesses if true venous hypertension is present and calf pump function

Venous hemodynamic assessment – Plethysmography



Venous recovery time



MVO/SVC for outflow obstruction assessment

More advanced hemodynamic testing
Measures venous recovery time – hypertension from superficial or deep system
Measures maximum venous outlflow – degree of outflow obstruction
Measures segmental venous capacitance – degree of venous stasis

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The modern approach to venous disease

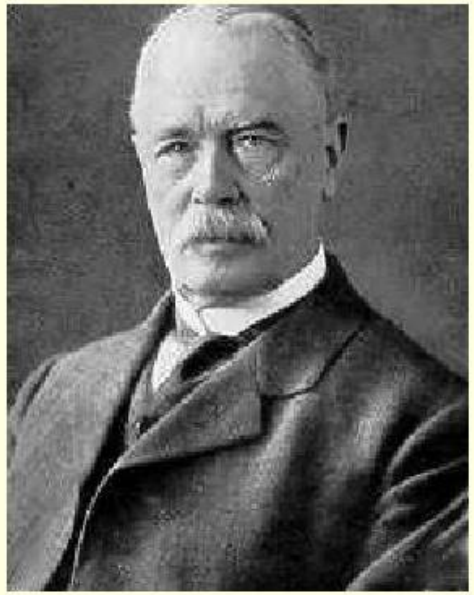
- All patients with suspected venous disease need a hemodynamic assessment
- Telangiectasiae with NORMAL LRR - SCLEROTHERAPY
- Telangiectasiae with ABNORMAL LRR, but no CVI – VENOUS DUPLEX
- Frank CVI or varicose veins – plethysmography to rule out outflow obstruction, then duplex to plan treatment
- All PCS, vulvar varicosities, abnormal varicosity pattern – plethysmography
- If positive for outflow obstruction – MR Venogram =/- TV duplex

Treatment options in venous disease

1. Pure telangiectasiae - Sclerotherapy
2. Superficial vein incompetence predominantly above knee – endothermal ablation i.e Radiofrequency ablation or Endovenous laser
3. Superficial vein incompetence predominantly below knee – Venous glue ablation
4. CVI with outflow obstruction – Balloon angioplasty of vein =/- iliac vein stent after IVUS
5. Severe PCS with no iliac vein hypertension – possible ovarian vein embolisation
6. Severe PCS with iliac vein hypertension – iliac vein stent with possible ovarian vein embolisation

Questions ???

Friedrich Trendelenburg (1844-1924)



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