Superficial Varicose and Deep Vein Concerns - ACOI Chicago Hospitalist Meeting

Davin Haraway DO, FACOI, FACWCS, RPhS
Diplomate – American Board of Venous and Lymphatic Medicine
Vein issues encountered by Hospitalists

- Cellulitis
- Infected venous stasis ulcerations
- Leg ulcers – consultations for evaluation and treatment
- Lymphedema Lymphangitis
- Superficial thrombophlebitis
- Post phlebetic syndrome
- Pulmonary Embolus
- Acute DVT at or above Common femoral vein
- Acute DVT below the Common femoral vein
- Upper extremity DVT
- Phlegmesia Cerula Dolens
- Venous malformations
- Stasis dermatitis vs Cellulitis vs hemosiderosis, vs calciphylaxis
Who do you hand off when the patient is discharged and what do you recommend?

- Sleep study
- Education on lymphedema treatment
- Referral to OP wound care/HBO
- Referral to phlebologist
- Who is going to manage their DVT/Anticoagulants/Stents
- Who is going to manage their wound care
- Do you have local support and know providers in the community to help the PCP?
Impact on venous disease outside the hospital
Davin Haraway DO,FACOI,FACCWS,RPhS
Diplomate Certified – American Board of Venous and Lymphatic Medicine
Outline

• Incidence and prevalence
• Anatomy
• Function and Physiology diagnosis
• Classification
• Conservative tx
• Intervention treatment
• Special considerations
What I see may have varicose veins?

All of the above
What I do
The Spectrum of Chronic Venous Disease

- telangiectasias
- superficial phlebitis
- varicose veins
- lipodermatosclerosis
- venous ulceration
Prevalence of Chronic Venous Disease

• 1 in 22 or 4.5% or 12.2 million people in the USA are affected by varicose veins

• Incidence increases with age and is more common in women with over 40% of women in their 50’s suffering from some sort of venous disorder

• Across all ages and gender, 60% of Americans suffer from venous disease and its sequelae

National Heart Lung and Blood Institute (NHLBI) http://www.nhlbi.nih.gov/
Total patients that would potentially just need GSV treatment by endothermal ablation range from 5,124 to 13,866 if symptomatic. A portion of these patients will need Phlebectomy and/or Ultrasound guided sclerotherapy in addition to endothermal ablation – all of which are reimbursable procedures by most Insurance carriers when symptomatic.

This assumes that all patients were healthy on a population basis. Age 35-64 years total 143,512 from Tulsa City Statistics 77% of this age group are 35-54 years old and 23% age 55 – 64 years old.
Medicare age patients with potential GSV reflux could be treated with endothermal ablation if symptomatic would range from 5,220 to 14,126. Medicare has coverage criteria for symptomatic patients.

Patients from age group 35 to over 65 total 10,344 – 27,922 in the Tulsa County
Prevalence and Etiology of Venous Insufficiency

Venous reflux disease is 2x more prevalent than coronary heart disease (CHD) and 5x more prevalent than peripheral arterial disease (PAD)

![Chart showing prevalence and etiology of various conditions including Venous Reflux Disease, Coronary Heart Disease, Peripheral Arterial Disease, Congestive Heart Failure, Stroke, Cardiac Arrhythmias, Heart Valve Disease, with comparison of annual U.S. incidence and U.S. prevalence.](chart.png)
Who should be screened?

- Patients with typical symptoms of venous disease with a constellation of symptoms of big varicosities that are symptomatic or a history of Venous Ulcerations or no skin changes at all could include:
  - Aching
  - Fatigue, heavines in legs
  - Pain: throbbing, burning, stabbing
  - Cramping
  - Swelling (peripheral edema)
  - Itching
  - Restless legs
  - Numbness
  - Leg ulcerations – usually on the lower leg could be medial or lateral with typically acceptable arterial circulation – What’s their ABI – not to miss Arterial disease
Are Vein procedures covered by my insurance?

- By and large if a patient has symptomatic venous disease with duplex findings of Reflux and enlarged size veins (variable to the insurance) of the GSV and or SSV and tributaries then Insurance will typically cover their treatment – Of course some insurances are more strict.

- Insurance LCD Also dictate mostly that the axial Veins of the GSV/SSV must be treated first before tributary disease can be covered.

- Foam and Liquid Sclerotherapy of veins less than 3mm and no reflux typically are not covered and are treated if symptomatic or at the desire of the patient for a more esthetic outcome on a Cash basis. (most SYMPTOMS are improved with treatment of the above however the end result is driven by the patient for cosmetic appearance).
Frequently Asked Questions

• What does my poor circulation have to do with my swelling?
  • We educate our patients on distinguishing between arterial, venous, and lymphatic circulation and go over their particular factors for their swelling verbally and with a diagram.
  • I have “stasis eczema” what is that
  • I use hydrocortisone on my legs for years why is that?
  • Draw Diagram How I educate patients on their Edema.
Do I have to wear Compression stockings?

• Compression stockings are a very important part of treating chronic venous disease – Depending on where the patient is on the spectrum of their particular disease process they may be recommended to always wear their compression hose (especially if Deep Reflux) or they may not need to wear their hose after their treatment if ALL reflux has been treated and no further symptoms. HOWEVER........

• All insurance Carriers start with a period of conservative treatment which includes Compression hose – NOT TED hose typically 20-30mmHg compression

• VENOUS DISEASE IS A CONDITION OF CHRONICITY AND MUST BE FOLLOWED – NEW AREAS OF REFLUX WILL LIKELY DEVELOP IN THE FUTURE AND MAY BE DEALT WITH EXPECTANTLY – to not notify a patient of this would disappoint the patient.
Anatomy
It all begins in the feet

• Vein anatomy that is...

• In general venous flow should be from superficial to deep in the lower extremity – the exception is the foot where there are general lack of valves in the deep veins of the foot that have outward flow thru perforators into the superficial venous arch that constitute the origins of the GSV and SSV.

• And otherwise directly communicate with the Posterior tibial and peroneal/anterior tibial veins.
Superficial Vein:

- The Great Saphenous Vein originates from the dorsal venous arch of the foot.
- After passing anterior to the medial malleolus, it runs up the medial side of the leg.
- At the knee, it runs over the posterior border of the medial epicondyle of the femur bone.
- The great saphenous vein then courses medially to lie on the anterior surface of the thigh before entering the saphenous opening.
- It joins with the femoral vein in the region of the femoral triangle at the saphenofemoral junction.
Superficial Venous System

- The superficial veins of the sole form a network that connects to the superficial dorsal veins of the foot and the deep plantar veins. The **dorsal venous arch**, into which empty the **dorsal metatarsal veins**, is continuous with the **greater saphenous vein** medially and the **lesser saphenous vein** laterally.
- The greater saphenous vein, in close proximity to the **saphenous nerve**, ascends **anterior to the medial malleolus**, crosses, and then ascends **medial to the knee**. It ascends in the **superficial compartment** and empties into the **common femoral vein** after entering the **fossa ovalis**.
- Before its entry into the **common femoral vein**, it receives **medial and lateral accessory saphenous veins**, as well as small tributaries from the **inguinal region**, **pubic region**, and **anterior abdominal wall**. The posterior arch vein drains the area around the medial malleolus, and as it ascends up the posterior medial aspect of the calf, it receives **medial perforating veins**, termed **Cockett’s perforators**, **before joining the greater saphenous vein at or below the knee**.
- The lesser saphenous vein arises from the **dorsal venous arch** at the lateral aspect of the foot and ascends **posterior to the lateral malleolus**, and it empties into the **popliteal vein** after penetrating the fascia. The **exact entry of the lesser saphenous vein into the popliteal vein** is variable.
THE VEINS OF LOWER LIMB

- The veins of the lower limb are subdivided into the superficial and deep. The double deep veins accompany the arteries.
- The superficial veins run below the skin and outside the proper fascia. The superficial veins give rise to the great and small saphenous veins. They arise from the dorsal and plantar venous networks of foot.
- **The great saphenous vein**, arises from the medial portion of the dorsal venous network of foot and ascend along the medial aspect of the leg and thigh. In the upper third of thigh, the vein runs along its anterior surface to reach the saphenous opening.
- On passing the saphenous opening, the vein joins the femoral vein. On the way to destination point, the vein receives numerous tributaries that anastomose with each other and with the tributaries of small saphenous vein and deep veins of lower limb.
Suprainguinal Venous Anatomy
What do you tell your patients about this?
Physiology
Etiology

- **AMBULATORY VENOUS HYPERTENSION**
  - Occurs when the vein valves become dysfunctional and impairs venous blood return.
    - Superficial venous reflux (long saphenous or short saphenous vein reflux)
    - Deep vein reflux (Primary or secondary to deep venous thrombosis)
    - Deep venous occlusion.
    - Perforating vein reflux.
    - Abnormal calf pump (Neurological/musculoskeletal)

- **Congenital absence of deep veins (e.g. Klippel-Trenaunay syndrome)**
- **Venous trauma (ligation of proximal vein).**
Ambulatory Venous Hypertension

Chronic venous disease

Distribution and extent of reflux
Amount of reflux
Risk factors
Genetic predisposition
Rate of progression
Severity of obstruction
Distribution and extent of obstruction
Foot and calf muscle pump efficiency
Venous wall and soft tissue cellular biology
Local responses
Lymphatic compensation
Ability to remove excess fluid

Signs and symptoms of CVI
Conservative management including compressive therapy
Satisfactory response
Unsatisfactory response or advanced clinical disease
Continue treatment
Non-Invasive Testing
Duplex and/or APG
Non-acute Obstruction
Reflux
Venography
Consider Venous Stenting
Venography
Consider Valve Reconstruction
Superficial
Consider Ablation or Stripping
Muscle Pump Dysfunction
Consider Exercise Program
Deep
Perforator
Consider SEPS
After Pappas (2002); TGF-β-1: transforming growth factor β-1; MMPs: metalloproteinases 1 & 2; PMN: polymorphonuclear; RBC: red blood cell; TIMP: tissue inhibitor metalloproteinase (deficient).

TGF: transforming growth factor; VEGF: vascular endothelial growth factor; MMP: metalloproteinases; TIMP: tissue inhibitor metalloproteinases. *: leukocyte adhesion area. SMCs: smooth muscle cells.
Ambulatory venous pressure

- The ankle venous pressure during walking.
- (AVP) is the "gold standard" test of the efficiency of the calf musculovenous pump
- by placing a small needle into one of the veins on the back of the foot and connecting the needle to a blood pressure measurement machine. The test has three parts
- standing venous pressure is around 90 mmHg, During exercise this should fall to around 30 mmHg.
Venous Valvular Function

- Valve leaflets allow unidirectional flow, upward or inward
- Dilation of vein wall prevents opposition of valve leaflets, resulting in reflux
- Valvular fibrosis, destruction, or agenesis results in reflux
A DVT Doppler study is NOT the same as Doppler study for Venous Reflux!
Doppler exam: Normal flow

Augmented flow in popliteal vein

Illustration by Linda S. Nye
Doppler: Reflux

Venous reflux

<table>
<thead>
<tr>
<th>Augmentation or Valsalva</th>
<th>Significant venous reflux of &gt; 2 sec duration</th>
</tr>
</thead>
</table>

Illustration by Linda S. Nye
REFLUX: its contribution to varicose veins
Pathophysiology: 2 components

REFLUX
• Dilatation of vein wall leads to valve insufficiency
• Monocytes may destroy vein valves
• Retrograde flow results in distal venous hypertension

OBSTRUCTION
• Thrombosis and subsequent fibrosis obstruct venous outflow
• Damage to vein valves may also cause reflux
• Both contribute to venous hypertension

The presence of both is far worse than either one alone
Venous Disease is a Hereditary Disorder

134 families were examined. The risk of developing varicose veins was:

- 89% if both parents had varicose veins
- 47% if one parent had varicose veins
- 20% if neither parent had varicose veins

The beginnings of venous disease may be found as early as childhood

<table>
<thead>
<tr>
<th>Age Group</th>
<th>Diagnosable Vein Disease</th>
<th>Actual Varicose Veins</th>
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<tbody>
<tr>
<td>10-12 y/o</td>
<td>2.5%</td>
<td>0</td>
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<tr>
<td>14-16 y/o</td>
<td>12.3%</td>
<td>1.7%</td>
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<tr>
<td>18-20 y/o</td>
<td>19.8%</td>
<td>3.3%</td>
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Inactivity aggravates venous disease

- 2,854 patients with varicose veins, working in a factory
- 64.5% had jobs standing in one place
- 29.2% had jobs requiring prolonged periods of sitting
- 6.3% had jobs allowing frequent walking during their shift

Santler, R Hautarzt 1956; 10:460
Each pregnancy worsens the condition

- 405 women with varicose veins
- 13% had one pregnancy
- 30% had two pregnancies
- 57% had three pregnancies

Brand FN, et al The epidemiology of varicose veins: the Framingham Study
Figure A5. Various patterns of the different flS anatomic types. (a) In the type "S", the superior epigastric vein is usually not visible. (b) In the "S" type, the visible epigastric vein is usually not visible. The "S" type gives rise to two different varicose patterns: (c) Only the superficial collateral is involved, whereas the main intramuscular superficial vein is competent and (d) Both the deep and superficial veins are involved.
Figure 25.1  Correlation of the venous reflux progression with age (A) and signs and symptoms (B) according to a retrospective study on 2,275 echo Doppler examination cases. All differences are significant (p<0.05).
CEAP Classification

• “C” = Clinical
  - C0 - no visible venous disease
  - C1 - telangiectasias or reticular veins
  - C2 - varicose veins
  - C3 - edema
  - C4 - skin changes without ulceration
    - C4a – pigmentation or eczema
    - C4b – LDS or atrophie blanche
  - C5 - skin changes with healed ulceration
  - C6 - skin changes with active ulceration

• “E” = Etiology (primary vs. secondary)
• “A” = Anatomy (defines location of disease within superficial, deep and perforating venous systems)
• “P” = Pathophysiology (reflux, obstruction, or both)
<table>
<thead>
<tr>
<th>CEAP classification and description</th>
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<tbody>
<tr>
<td>1. Clinical classification</td>
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<tr>
<td>C0: No visible or palpable signs of venous disease</td>
</tr>
<tr>
<td>C1: Telangiectases or reticular veins</td>
</tr>
<tr>
<td>C2: Varicose veins</td>
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<tr>
<td>C3: Edema</td>
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<tr>
<td>C4: Pigmentation and/or eczema</td>
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<tr>
<td>C5: Lipodermatosclerosis and/or atrophy</td>
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<td>C6: Healed venous ulcer</td>
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<td>C7: Active venous ulcer</td>
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<tr>
<td>C8: Symptoms, including ache, pain, tightness, skin irritation, heavity, muscle cramps, as well as other complaints attributable to venous dysfunction</td>
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<td>C9: Asymptomatic</td>
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<tr>
<th>2. Etiologic classification</th>
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<tbody>
<tr>
<td>E1: Congenital</td>
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<tr>
<td>E2: Primary</td>
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<tr>
<td>E3: Secondary (postthrombotic)</td>
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<tr>
<td>E4: No venous etiology identified</td>
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<tr>
<th>3. Anatomic classification</th>
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<tbody>
<tr>
<td>A1: Superficial veins</td>
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<tr>
<td>A2: Perforator veins</td>
</tr>
<tr>
<td>A3: Deep veins</td>
</tr>
<tr>
<td>A4: No venous location identified</td>
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<tr>
<th>4. Pathophysiologic classification</th>
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<tbody>
<tr>
<td>P1: Reflux</td>
</tr>
<tr>
<td>P2: Obstruction</td>
</tr>
<tr>
<td>P3: Reflux and obstruction</td>
</tr>
<tr>
<td>P4: No venous pathophysiology identifiable</td>
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<thead>
<tr>
<th>Anatomic classification and venous segment description</th>
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<tbody>
<tr>
<td><strong>Superficial veins</strong></td>
</tr>
<tr>
<td>1. Telangiectases/reticular veins</td>
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<tr>
<td>2. GSV above knee</td>
</tr>
<tr>
<td>3. GSV below knee</td>
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<td>4. Short saphenous vein</td>
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<td>5. Non-saphenous veins</td>
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<th><strong>Deep veins</strong></th>
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<tr>
<td>6. Inferior vena cava</td>
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<tr>
<td>7. Common iliac vein</td>
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<tr>
<td>8. Internal iliac vein</td>
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<tr>
<td>9. External iliac vein</td>
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<tr>
<td>10. Pelvic: gonadal, broad ligament veins, other</td>
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<tr>
<td>11. Common femoral vein</td>
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<tr>
<td>12. Deep femoral vein</td>
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<tr>
<td>13. Femoral vein</td>
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<tr>
<td>14. Popliteal vein</td>
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<tr>
<td>15. Crural veins: anterior tibial, posterior tibial, peroneal veins (all paired)</td>
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<tr>
<td>16. Muscular veins: gastrocnemius, soleal, other</td>
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<th>Perforating veins</th>
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<tr>
<td>17. Thigh perforator veins</td>
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<td>18. Calf perforator veins</td>
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History

- History of problem: onset, pregnancies, prior DVT, immobilization
- Associated symptoms and relationship to heat, menses, exercise and compression
- Current medications
- Family history
- Previous treatment and result
- Goals of patient
Physical Examination

- Examine patient in the standing position, from the groin to the ankle
- Inspect and palpate for varicose and telangiectatic veins
- Check the medial and lateral malleolar areas for skin changes suggestive of chronic venous insufficiency (e.g., corona phlebectatica)
- Inspect the abdomen for enlarged superficial veins if ilio-femoral thrombosis is suspected
Telangiectasias

- Also known as “spider veins” due to their appearance
- Very common, especially in women
- Increase in frequency with age
- 85% of patients are symptomatic*
- May indicate more extensive venous disease

Lateral Subdermic Plexus

- Very common, especially in women
- Superficial veins with direct perforators to deep system
- Remnant of embryonic deep venous system
Reticular Veins

- Enlarged, greenish-blue appearing veins
- Frequently associated with clusters of telangiectasias
- May be symptomatic, especially in dependent areas of leg
Varicose Veins –
Great Saphenous Distribution

• Most common finding in patients with varicose veins
• Varicosities most commonly along the medial thigh and calf but cannot assume location indicates origin
• At least 20% of patients are at risk of ulceration
Great Saphenous Insufficiency

- Skin changes are seen along the medial aspect of the ankle
- The presence of skin changes is a predictor of future ulceration

Varicose Veins –
Small Saphenous Distribution

• Less frequent than Great Saphenous involvement
• Varicosities may be seen on the posterior calf and lateral ankle
• Skin changes are seen along the lateral ankle
Skin changes suggestive of chronic venous insufficiency

- Corona Phlebectatica (C1)
- Pigmentation (C4a)
- Atrophie blanche (C4b)
- Healed ulcer (C5)
Frequently Asked Questions

• What does my poor circulation have to do with my swelling?
  • We educate our patients on distinguishing between arterial, venous, and lymphatic circulation and go over their particular factors for their swelling verbally and with a diagram.
  • I have “stasis eczema” what is that
  • I use hydrocortisone on my legs for years why is that?
  • Draw Diagram How I educate patients on their Edema.
PT Code: 93923  ICD-9 Code: [ ] 440 [ ] 443.8 [ ] 443.9 [ ]

**Doppler Waveforms**

**RIGHT**
- **FEMORAL**
- **POPLITEAL**
- **POSTERIOR TIBIAL**
- **DORSALIS PEDIS**

**LEFT**
- **FEMORAL**
- **POPLITEAL**
- **POSTERIOR TIBIAL**
- **DORSALIS PEDIS**

**Indexes**

<table>
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<td>L. THIGH</td>
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<td>CALF</td>
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<tr>
<td>ANKLE-PT</td>
<td>0.94</td>
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<tr>
<td>ANKLE-DP</td>
<td>0.91</td>
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</table>

(*) Indexes use highest brachial pressure.
CHF (left and/or Right)
Kidney disease
Liver Disease
Lymphedema
Lipo edema
Hypothyroidism
Varicose Veins

Exacerbating factors –
obesity/pulmonary hypertension

(too many bags of frito’s- True story!!!)
Inverted tree

• Pure Superficial Venous Edema is usually from the “knee down”
• If there is edema extends into the thigh and above think additional systemic factors and/or deep venous involvement
UNDERSTANDING VENOUS REFUX DISEASE
Healthy leg veins contain valves that open and close to assist the return of blood back to the heart. Venous reflux disease develops when the valves that keep blood flowing out of the legs and back to the heart become damaged or diseased. As a result, vein valves will not close properly, leading to symptoms of:
- Varicose veins
- Pain
- Swelling
- Leg heaviness and fatigue
- Skin changes
- Ulcers

THE VENOUS SYSTEM ANATOMY
The venous system is made up of a network of veins, including:
- Superficial veins: veins located close to the surface of the skin.
- Deep veins: larger veins located deep in the leg.
- Perforation veins: veins that connect the superficial veins to the deep veins.

THE VENEFIT™ procedure treats venous reflux disease in the superficial venous system, often the underlying cause of painful varicose veins.

VENOUS REFUX DISEASE IS PROGRESSIVE — SYMPTOMS CAN WORSEN OVER TIME IF LEFT UNTREATED
A Serious Progressive Disorder

EXPERIENCE THE VENEFIT™ PROCEDURE
The Venefit™ procedure is performed on an outpatient basis. Using ultrasound, your physician will position the Clotrulix™ catheter into the diseased vein through a small opening in the skin. The tiny catheter, powered by radio-frequency (RF) energy delivered near the vein wall, delivers RF energy to the vein wall. Once the diseased vein is closed, blood will re-route itself to other healthy veins.

Veganes imbalance
Vessel enlargement
Nerve damage
Nerve damage

Following the procedure, a simple bandage is placed over the insertion site, and additional compression may be provided to aid healing. Your doctor may encourage you to walk, and to refrain from extended standing and strenuous activities for a period of time.

The average patient typically resumes normal activities within a few days.

ARE YOU SUFFERING FROM VENOUS REFUX DISEASE?
Many factors contribute to the presence of venous reflux disease, including:
- Age
- Gender
- Family history
- Standing profession
- Obesity
- Multiple pregnancies
- Heavy lifting
- History of varicose veins

Using ultrasound to scan your leg(s), your physician will determine if venous reflux is present.

PROCEDURAL HIGHLIGHTS*
- Relief of symptom within 2 days
- Outpatient procedure
- Can be performed under local anesthesia
- The average patient typically resumes normal activities within a few days
- Proven results with positive patient outcomes and experience

VISUAL RESULTS*

INDICATIONS: The Clotrulix™ catheter is intended for endoluminal closure of small veins in patients with superficial venous reflux.

CONTRAINDICATIONS: Patients with chronic blood clots in the veins are not candidates for the Venefit™ Procedure.

POTENTIAL COMPLICATIONS: As with all medical procedures, potential side effects and complications exist including vein perforation (when the catheter punctures the vein wall), thrombosis, pulmonary embolism (when a blood clot travels to the lungs), pleural effusion (accumulation of fluid in the lungs), infection, nerve damage, overextension (an abnormal connection between an artery and a vein), hematoma (bruising), and skin burn. If any complications arise, consult your physician for information on the risks and benefits of the procedure.

* Individual results may vary.
Edema

• All edema is lymphedema
• CHF Left and/or Right, cirrhosis, Renal failure = Diuretics + compression
• Venous insufficiency = compression/Ablation/procedure if indicated – not diuretics
• Lymphedema = manual decongestive therapy + compression – Not diuretics
• Don’t forget Thyroid
• Lipo Edema – characteristic Cut off sign at ankle
• Can have all three – PhleboLipoLymphedema
• Be sure to check ABI if compressing – Caution if ABI <0.8
The Lymphatic System

- Lymphatic system functions:
  - Transport clean fluids back to the blood
  - Drains excess fluids from tissues
  - Removes “debris” from cells of body
  - Transports fats from digestive system
<table>
<thead>
<tr>
<th>Stage 0 (latent):</th>
<th>Stage 1 (reversible):</th>
<th>Stage 2 (irreversible):</th>
<th>Stage 3 (lymphostatic elephantiasis):</th>
</tr>
</thead>
<tbody>
<tr>
<td>Swelling is not evident despite impaired lymph transport and may exist for months or years before oedema becomes evident.</td>
<td>Early onset of the condition. Accumulation of tissue fluid that subsides with limb elevation. Oedema may be pitting at this stage (no fibrosis)</td>
<td>Limb elevation alone rarely reduces swelling, and pitting is manifest with protein rich oedema fluid. Late stage 2: There may or may not be pitting as tissue fibrosis is more evident.</td>
<td>Accumulation of protein rich edema fluid. Tissue is hard (fibrotic) and pitting is absent. Skin changes, such as thickening, hyperpigmentation, increased skin folds, fat deposits and warty overgrowths develop.</td>
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Frequently asked questions

• Aren’t Vein Practices just cosmetic and spa centers?

• No - Phlebology practice encompasses a wide range of Services. As with other practices may deal with Deep system problems (such as May/Thurner syndrome thrombolytic therapy and stenting/coiling vascular malformation) to only superficial disease – GSV/SSV/Perforator and or tributary disease to the reticular and telangiectasia OR both.

• Certainly patients may initially present with the intent of a Cosmetic outcome but the evaluation always begins with a History/Physical AND a Duplex Ultrasound specifically for REFLUX (different protocol for DVT).

• Surprising Number of patients when carefully questioned about their leg discomfort have Symptoms that can be attributed to Venous Disease.
Guidance for the treatment of deep vein thrombosis and pulmonary embolism


• Michael B. Streiff1 • Giancarlo Agnelli2 • Jean M. Connors3 • Mark Crowther4 • Sabine Eichinger5 • Renato Lopes6 • Robert D. McBane7 • Stephan Moll8 • Jack Ansell9

• Published online: 16 January 2016
Ó The Author(s) 2016. This article is published with open access at Springerlink.com
Who should be screened?

• Patients with typical symptoms of venous disease with a constellation of symptoms of big varicosities that are symptomatic or a history of Venous Ulcerations or no skin changes at all could include:
  • Aching
  • Fatigue, heaviness in legs
  • Pain: throbbing, burning, stabbing
  • Cramping
  • Swelling (peripheral edema)
  • Itching
  • Restless legs
  • Numbness
  • Leg ulcerations – usually on the lower leg could be medial or lateral with typically acceptable arterial circulation – What’s their ABI – not to miss Arterial disease
Evaluation and treatment of Venous Ulcerations

• We will get into this
• Summarize – Compression mainstay of treatment of venous ulcerations.
• Eschar Study showed no help of healing with ligation/stripping HOWEVER reoccurrence rate decreased with ligation/stripping with Evidence of Eschar Study – Several flaws in methodology (delay of 4-7 weeks before surgery was performed)

• Society for Vascular Surgery and American venous Forum recommendations
• Grade 1B compression therapy for healing venous ulcers
• Grade 1B if patient is candidate for Ablation of symptomatic varicose veins without ulcers over chronic compression
• Grade 1A ablation of incompetent superficial veins in addition to compression therapy for reduced reoccurrence rates for Venous Ulcerations
Long term results of compression therapy alone versus compression plus surgery in chronic venous ulceration (ESCHAR): randomised controlled trial

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ABSTRACT
Objective To determine whether recurrence of leg ulcers may be prevented by surgical correction of superficial venous reflux in addition to compression.

Design Randomised controlled trial.
Setting Specialist nurse led leg ulcer clinics in three UK hospitals.

INTRODUCTION
In recent years the importance of the effect of venous leg ulceration on healthcare expenses and quality of life has been recognised. In Europe, leg ulcers have been reported to affect 1% of the adult population, increasing dramatically in those aged more than 80. The precise pathophysiological mechanism...
Venous Ulceration

• Over 50% of patients have only superficial venous disease; superficial venous disease may be primary factor in 50-85% of patients*
• <10% have only deep venous disease
• Results from ambulatory venous hypertension, which leads to WBC activation, ↓TCpO2, local release of proteolytic enzymes

Venous Ulceration

Impending ulceration
Lipodermatosclerosis (C4a)

Venous ulceration (C6)
Unique issues

• Hemorrhage of varicosity
  • Can be scary for the patient
    • Usually story is in the shower and look down and “blood just pours”
    • Education to get their leg UP and put compression on it – not lean over and apply compression (just increasing the hydrostatic pressure to the vein)
  • This is usually an insurance automatic for an intervention especially if recurrent for some type of intervention whether it be ablation, phlebectomy, sclerotherapy or some combination of treatments – of course with venous reflux exam to determine the source of reflux.

• Active or healed venous ulcerations – usually an insurance automatic for intervention – must have an outlined plan for compression however before I will treat an active ulceration

• Recurrent Phlebitis – this will typically accelerate authorization
Who should be screened?

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Do I have to wear Compression stockings?

- Compression stockings are a very important part of treating chronic venous disease – Depending on where the patient is on the spectrum of their particular disease process they may be recommended to always wear their compression hose (especially if Deep Reflux) or they may not need to wear their hose after their treatment if ALL reflux has been treated and no further symptoms. HOWEVER........

- All insurance Carriers start with a period of conservative treatment which includes Compression hose – NOT TED hose typically 20-30mmHg compression

- VENOUS DISEASE IS A CONDITION OF CHRONICITY AND MUST BE FOLLOWED – NEW AREAS OF REFLUX WILL LIKELY DEVELOP IN THE FUTURE AND MAY BE DEALT WITH EXPECTANTLY – to not notify a patient of this would disappoint the patient.
CONSERVATIVE TREATMENT OF VENOUS DISORDERS
Compression Therapy

- Provides a gradient of pressure, highest at the ankle, decreasing as it moves up the leg
- Reduces reflux of blood
- Improves venous outflow
- Increases velocity of blood flow to reduce the risk of blood clots

Photo courtesy of Juzo
Compression Therapy

• Reduces symptoms of aching, fatigue, pain, and swelling
• Increases fibrinolytic activity
• Increases TCpO2
• Mainstay of treatment for venous ulcers
  • NOTE: Graduated compression therapy and wound care will heal venous stasis ulcers. Elimination of the reflux will reduce the recurrence.

• Note – Tubigrip is variable in patients with ulcerations depending on their anatomy and ankle pressure
Elastic compression stockings

- Must be graduated
- Calf high generally sufficient
- Replace q 6 months to assure proper pressure
- Available in a variety of strengths, styles, colors, and fabrics
Graduated compression is not the same as T.E.D. hose

- T.E.D.s are meant for non-ambulatory, supine patients
- T.E.D.s are indicated to decrease the incidence of thrombosis
- T.E.D.s do not provide sufficient pressure for ambulatory patients
<table>
<thead>
<tr>
<th>Compression Strength</th>
<th>Indications</th>
</tr>
</thead>
<tbody>
<tr>
<td>8-15mm</td>
<td>Leg fatigue, mild swelling, stylish</td>
</tr>
<tr>
<td>15-20mm</td>
<td>Mild aching, swelling, stylish</td>
</tr>
<tr>
<td>20-30mm</td>
<td>Aching, pain, swelling, mild varicose veins</td>
</tr>
<tr>
<td>30-40mm *</td>
<td>Aching, pain, swelling, varicose veins, post-ulcer</td>
</tr>
<tr>
<td>40-50, 50-60mm *</td>
<td>Recurrent ulceration, lymphedema</td>
</tr>
</tbody>
</table>

* Requires a prescription
Gradient compression hose

• At least Calf high 20-30mmhg
  • Thigh high or panty hose may be more comfortable depending on the situation
  • Open or closed toe depending on concurrent condition such as diabetes, neuropathy or concurrent arterial disease.

• The clock starts ticking when they began their compression so if you document it I can use that date for their insurance approval.

• Where can they get them?
  • DME store – LKM, Freeland Brown, Fidelity Lymphedema, can purchase over the counter (typically sized by the calf and thigh circumference and outseam)

• Yes some fit better than others but if symptoms helped and continue despite their stockings and reflux found – good chance they will respond to treatment.
If they have an Ulcer

- This is a good patient to send to both the wound center AND Tulsa Vein as the wound centers are great at getting them in compression, evaluating the wound for proper dressings/home health, debridement, biopsy, and education.

- Waiting on “complete healing” before a referral for ablation need not occur as this may just delay getting the patient the needed treatment - even if heals will likely need referral to keep it closed.

- We can work on the Cause of the Ulceration – REFLUX -and try to get the ulcer healed faster and maintain skin integrity with intervention such as endovenous ablation and foam sclerotherapy or other techniques.

- Likely their Ultrasound will prognosticate if they have single, double or triple component disease – meaning superficial or Deep or perforator disease or all three.
Endothermal and Non thermal Ablation

• Venefit Procedure  Endothermal Radiofrequency Ablation—Coviden/Medtronic
• Endothermal Laser Ablation
• Mechanical Occlusion Chemical Assisted ablation (Clarivein)
• Venaseal – (cyanoacrolate)
• Varithena – Microchemical Foam ablation
• Steam ablation (Europe)
• Foam ablation (polidocanol or sotradecol)
• Stripping
• Local Microphlebectomy
Catheter withdrawn from marker to marker

Until entire length of vein is treated

5. Introducing the ClosureFAST catheter
Things I did not talk about due to time- not that they are unimportant

- Type of compression bandaging - Inelastic vs elastic ie unna boot vs multilayer compression – both work
- Types of dressings – follow “best wound care protocols”
- Types of Cellular tissue products – I’ve used most all and have spoken on most - some have advantages over others but wont work at all if poor wound bed preparation – Again thank you Osiris Therapeutics for sponsoring this lecture with a grant.
- Lymphedema treatment as a component of venous ulcer healing – Lymphedema pumps and ambulatory lymphedema pump -Acti-touch
- Medications for Venous disease – trental for venous ulcers and other meds such as horse chestnut, pycnogenol, ketoprophen, resveratrol, tumeric, vasculera, diosmen complex etc.
- Ambulatory Venous Hypertension MUST be addressed to have optimal outcome and sustained healing
Guidance for the treatment of deep vein thrombosis and pulmonary embolism

• Summary Guidelines
Contraindications to outpatient treatment of venous thromboembolism

- Active or high risk of bleeding
- Recent surgery (within 7 days)
- Cardiopulmonary instability
- Severe symptomatic venous obstruction
- High risk pulmonary embolism*
- Thrombocytopenia (platelets<50,000/μL)
- Other medical or surgical condition requiring inpatient management

- Medical non-compliance
- Geographical or telephone inaccessibility
- Poor hepatic function (International Normalized Ratio (INR)>1.5)
- Unstable renal function (e.g. rising serum creatinine)
- Poor home health care support environment

- * High risk PE is characterized by systolic blood pressure<90 mmHg or a systolic blood pressure drop of>40 mmHg for>15 min not due to an arrhythmia, hypovolemia or sepsis
Wells clinical DVT model
Clinical characteristic

Active cancer (patient receiving treatment for cancer within 6 months or currently receiving palliative treatment) 1
Paralysis, paresis, or recent plaster cast immobilization of the lower extremities 1
Recently bedridden for 3 days or more, or major surgery within the previous 12 weeks requiring general or regional anesthesia 1
Localized tenderness along the distribution of the deep venous system 1
Entire leg swollen 1
Calf swelling at least 3 cm larger than the asymptomatic side (measured 10 cm below the tibial tuberosity) 1
Pitting edema confined to the symptomatic leg 1
Collateral superficial veins (non-varicose) 1
Previously documented deep vein thrombosis 1
Alternative diagnosis at least as likely as deep vein thrombosis -2

Score

A score of 0 indicates that a low pretest probability of deep vein thrombosis. A score of 1 or 2 points indicates a moderate risk of DVT and a score of 3 or higher indicates a high risk of deep vein thrombosis [152]
Wells clinical pulmonary embolism model

- Clinical characteristic Score
  - Active cancer (patient receiving treatment for cancer within 6 months or currently receiving palliative treatment) 1
  - Surgery or bedridden for 3 days or more during the past 4 weeks 1.5
  - History of deep venous thrombosis or pulmonary embolism 1.5
  - Hemoptysis 1
  - Heart rate [100 beats/min 1.5
  - Pulmonary embolism judged to be the most likely diagnosis 3
  - Clinical signs and symptoms compatible with deep venous thrombosis 3

- A score of <2 indicates a low probability of pulmonary embolism. A score of 2–6 indicates an intermediate probability of PE. A score of more than 6 indicates a high probability of pulmonary embolism.

Simplified PESI (pulmonary embolism severity index) score Predictors

- Age >80 years
  - History of cancer
  - History of heart failure
  - Pulse>110 beats/min
  - Systolic blood pressure<100 mmHg
- Arterial oxygen saturation<90 %
- Low risk=total point score 0
- Management of outpatients vs Inpatients
How is the diagnosis of deep vein thrombosis and pulmonary embolism established?

- We suggest the use of validated pre-test probability models in conjunction with D dimer testing and selective use of objective diagnostic imaging to increase the cost-efficiency and accuracy of VTE diagnosis.
Which patients require hospitalization versus initial outpatient therapy for the management of VTE?

• We suggest that most patients with DVT and many patients with PE can be managed as outpatients. PE patients should be risk stratified to determine appropriate management. A variety of laboratory tests and imaging modalities as well as clinical risk prediction models are available to identify PE patients who are suitable for outpatient management. Further research is needed to identify the optimal approach to risk stratification of PE patients.
What are the therapeutic options for the acute treatment of venous thromboembolism?

- With the variety of treatment options available, we recommend that the acute therapy of VTE should be customized to suit the unique clinical circumstances of the individual patient. We suggest that unfractionated heparin may be preferable for inpatients with planned invasive procedures, recent major bleeding episode or impaired renal function as well as underweight and morbidly obese patients although several members of panel felt there were insufficient data to support this suggestion. LMWHs are convenient options for inpatient and outpatient therapy. DOACs are optimized for outpatient therapy of VTE.

- We suggest that systemic and catheter-directed pharmacomechanical thrombolytic therapy are effective options for treatment of acute extensive proximal DVT and massive PE that can rapidly reduce thrombus burden. Given the greater risks of bleeding associated with these approaches, we recommend that a careful assessment of the risks and benefits of therapy should be performed in each patient prior to the initiation of thrombolytic therapy.
Which patients are candidates for a DOAC?

- **Dabigatran**
  - When used after a 5–10 day initial course of parenteral anticoagulation, dabigatran is as effective as warfarin in the acute and short term treatment of VTE. We suggest dabigatran as an alternative to vitamin K antagonists for the short term therapy of VTE. In some studies, dabigatran has been associated with an increased risk of acute coronary syndrome and gastrointestinal bleeding compared with vitamin K antagonists.

- **Rivaroxaban**
  - Rivaroxaban is as effective as LMWH/VKA in the treatment of DVT and PE. We suggest rivaroxaban as an alternative to LMWH/VKA for the acute and short term treatment of VTE in appropriate patients. No increase in acute coronary syndrome or gastrointestinal bleeding has been seen with rivaroxaban, however GI bleeding may be more common in patients age 75 and older.

- **Apixaban**
  - Apixaban is as effective as LMWH/VKA in the treatment of DVT and PE and associated with less major bleeding and major or clinically relevant non-major bleeding. We suggest apixaban as an alternative to LMWH/VKA in the acute and short term treatment of VTE in appropriately selected patients. No increase in acute coronary syndrome or gastrointestinal bleeding has been seen with apixaban.

- **Edoxaban**
  - After an initial 5–10 days of LMWH or UFH, edoxaban is as effective as LMWH/VKA in the treatment of acute DVT and PE but associated with less major or clinically relevant non-major bleeding. We suggest edoxaban as an alternative to VKA for the short-term treatment of VTE in appropriately selected candidates.
What is the role of vena cava filters if the patient is not a candidate for anticoagulation?

- We suggest that vena cava filters should be considered in any patient with acute VTE (within 4 weeks) who cannot be treated with anticoagulation. We suggest that retrievable filters are strongly preferred as most patients have only temporary contraindications to anticoagulation. Filters should be retrieved once anticoagulation can be reinitiated preferably within 6 months of placement. Patients with filters should be closely monitored in a structured program to facilitate retrieval and minimize the number of patients lost to follow up.

- Following anticoagulation-associated gastrointestinal bleeding, we suggest that anticoagulation can be re-initiated as early as 7 days after cessation of bleeding and treatment of causal lesions. Following anticoagulation associated ICH, we suggest resumption of anticoagulation no sooner than 10 weeks post-bleed. Further investigation of this topic is warranted.
How is upper extremity VTE treated?

• Identification and elimination of trigger factors when feasible is important to reduce the incidence of recurrent upper extremity DVT. For CVC-associated DVT, we suggest that anticoagulation without CVC removal is the treatment of choice. If symptoms fail to resolve, CVC removal can be considered. We suggest that anticoagulation should be continued for at least 3 months or the duration of the CVC whichever is longer. At least 3 months of anticoagulation is appropriate for pacemaker wire-associated VTE.

• The committee was divided as to the optimal approach to treatment of TOS/PSS-associated upper extremity DVT. The benefits of rib resection/scalenectomy following thrombolysis and anticoagulation remain to be rigorously demonstrated. Therefore, providers should consider therapy for TOS/PSS on a case-by-case basis until higher quality data are available. We suggest that TOS/PSS-associated upper extremity DVT warrants anticoagulation for at least

• 3 months. Treatment of upper extremity DVT associated with extrinsic compression due to cancer or infection should include treatment of the underlying disease in addition to anticoagulation.
When is ambulation/exercise safe after DVT/PE?

• We suggest that ambulation is safe in patients with acute DVT±PE after initiation of anticoagulation
Is the use of graduated compression stockings safe after acute DVT/PE?

• We suggest that GCS do not increase the risk of recurrent thromboembolism inpatients with acute VTE. We suggest that GCS do not have any beneficial effect on leg discomfort associated with acute DVT.
What is the recommended duration of therapy for a patient with distal DVT?

- We suggest treatment of distal DVT with anticoagulation versus observation. We suggest a duration of therapy of 3 months. In patients with contraindications to anticoagulation, we favour repeat duplex surveillance in 1 week rather than vena cava filter insertion.
What is the recommended duration of therapy for a patient with a surgically provoked VTE?

• We suggest that 3 months of anticoagulation is adequate for surgical risk factor-associated VTE unless risk factors for recurrence persist
What is the recommended duration of therapy for a pregnancy or estrogen-associated VTE?

• We suggest that patients with pregnancy-associated VTE should be treated for the duration of the pregnancy and the post-partum period (up to 12 weeks post-partum) or as long as dictated by the VTE, whichever is longer. Patients with pregnancy-associated VTE are at high risk for recurrent VTE with subsequent pregnancies, therefore we suggest that thromboprophylaxis for the duration of the pregnancy and post-partum period should be strongly considered.

• Patients with hormone-associated VTE appear to be at lower risk for recurrent VTE particularly if their D dimer is negative at the end of therapy and 1 month after discontinuing anticoagulation. Therefore, we suggest that long term anticoagulation beyond 3–6 months may not be associated with a favorable risk:benefit balance if hormonal therapy has been discontinued. If hormonal therapy is medically necessary, we suggest that anticoagulation should be continued as these patients are at high risk for recurrent VTE.
What is the recommended duration of therapy for a medical illness-associated VTE?

• We suggest that patients with medical-illness associated VTE should be treated for at least 3 months or as long as the medical risk factors for VTE remain present.
What is the recommended duration of therapy for a travel-associated VTE?

• Travel ([4 h duration]) is a modest and transient risk factor for VTE. Therefore, VTE should only be ascribed to air travel if it presents within 4 weeks of travel and is not associated with other concomitant triggers. In the absence of other precipitants, we suggest that travel-associated VTE should be treated for at least 3 months. We suggest that travel thromboprophylaxis be considered for future travel in these patients.
What is the recommended duration of therapy for a malignancy-associated VTE?

- Active cancer is a potent risk factor for VTE that varies with the type and extent of cancer and its treatment. Therefore, we suggest anticoagulation be continued as long as the underlying cancer is active or under treatment.
What is the recommended duration of therapy for a patient with unprovoked VTE?

• Patients with unprovoked VTE are at high risk for recurrence so we suggest long term anticoagulation. As there is limited information on the risks and benefits of anticoagulation beyond 2 years, we suggest that providers reassess patients on long term anticoagulation on an annual basis.
What are the therapeutic options for long term treatment of DVT/PE?

- Vitamin K antagonists
  - Adjusted dose vitamin K antagonists (INR 2–3) reduce the relative risk of recurrent VTE by 88%, but they are associated with 2.6 fold increase in major bleeding compared with placebo. Consequently, it is important to assess the risks and benefits of long term anticoagulation on a case-by-case basis. Since low intensity (INR 1.5–2) anticoagulation is associated with a similar risk of major bleeding, we prefer standard intensity anticoagulation for long term therapy of VTE.

- LMWH/Fondaparinux
  - Evidence indicates that LMWH is as effective as VKA in the reduction of recurrent VTE but associated with a reduced risk of major bleeding. Limited experience with fondaparinux in the long term treatment of VTE suggests that it is as effective as LMWH in the prevention of recurrent VTE. Fondaparinux may cause more bleeding than VKA in patients without cancer. We suggest that LMWH and fondaparinux are acceptable alternatives to VKA for treatment of VTE.

- Direct Oral Anticoagulants Dabigatran
  - In long term therapy of VTE, dabigatran was as effective as warfarin and superior to placebo in prevention of recurrent thromboembolism. There was a trend toward reduced major bleeding with dabigatran compared with warfarin but major bleeding was 3-fold higher with dabigatran than placebo. We suggest that these data establish dabigatran as a viable option to vitamin K antagonists for long term therapy of VTE.

- Rivaroxaban
  - In long term treatment of VTE (after 3–6 months of therapy), rivaroxaban was more effective than placebo but associated with 5 fold increase in major or clinically relevant non-major bleeding. We suggest that these data support the use of rivaroxaban in the long term treatment of VTE in candidates suitable for anticoagulation.

- Apixaban
  - In long term treatment of VTE (after 6 months of therapy), apixaban was more effective than placebo and associated with a similar risk of major or clinically relevant non-major bleeding. We suggest that these data support the use of apixaban for the long term treatment of VTE in patients who are appropriate candidates. The option of a reduced dose for long term secondary prevention maybe attractive for some patients.

- Aspirin
  - After an initial 6–18 months of anticoagulation, aspirin 100 mg daily was associated with a 34% reduction in the relative risk of recurrent VTE (from 7.5 to 5.1%) compared with placebo. Major bleeding was similar in both groups. Therefore, we suggest that aspirin should be considered an option for patients at risk for recurrent VTE who are not considered appropriate candidates for long term anticoagulation or who chose to discontinue anticoagulation.
What is the best treatment of patients who have recurrent VTE in spite of anticoagulation?

• In patients with recurrent VTE despite anticoagulation, we suggest that it is important for providers to assess adherence to therapy and identify clinical conditions associated with anticoagulation failure including cancer, antiphospholipid syndrome, heparin-induced thrombocytopenia and vascular compression syndromes (May-Thurner syndrome, thoracic outlet syndrome). We suggest that higher-intensity anticoagulation (VKA INR 2.5-3.5 or 3–4 or based upon chromogenic factor X activity or escalated dose [125 % dose] LMWH), alternative forms of parenteral anticoagulation and therapies directed at restoring adequate blood flow are effective strategies to consider
How can you assess the risk of recurrent VTE and anticoagulant-associated bleeding?

• We suggest that patients with unprovoked VTE should be considered intrinsically thrombophilic and long term anticoagulation should be considered. When assessing the risk of recurrent VTE in patients with provoked VTE, it is important to determine whether provoking factors persist. If such factors are still present, we suggest that continued anticoagulation should be considered if bleeding risk is not excessive.

• We suggest that D dimer testing represents a promising global measure of pro-thrombotic potential that can be used to risk stratify patients for their future risk of VTE. However, we believe it is important to recognize that different D dimer assays may have different performance characteristics in regards to VTE risk assessment. In addition, the impact of age on the D dimer results and VTE risk prediction remains incompletely characterized. The value of other global laboratory measures (endogenous thrombin potential) and imaging studies remains to be established. Multimodality risk assessment models appear to be effective approach to risk stratification of patients with unprovoked VTE. Further validation of these risk assessment tools is underway.

• Anticoagulation-associated bleeding

• While a bleeding risk assessment is important to the decision on the duration of anticoagulation, we suggest that it is premature to use formal bleeding risk assessment models to identify patients who should discontinue anticoagulation. Development of better risk prediction models remains a priority.
Thank You!

• It is always an Honor to be invited to speak to colleagues in medicine!