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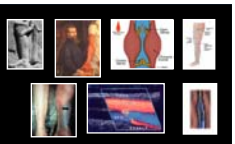

Vein Science:

Changing Perspectives on a very old disease

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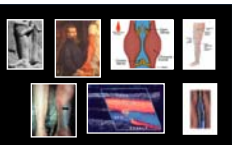
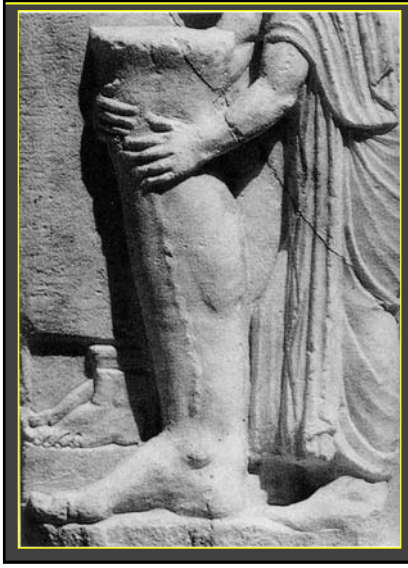


History



Athens 550 B.C
Votive offering from a patient

Vesalius 1543
Venous anatomy

Richard Lower 1640
Venous Return

Rudolf Virchow 1846
Triad: Stasis – Hypercoagulability - Injury

Ochsner and DeBakey 1939
Phlebothrombosis v Thrombophlebitis

Homan 1954
Associates DVT with sitting

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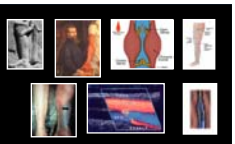
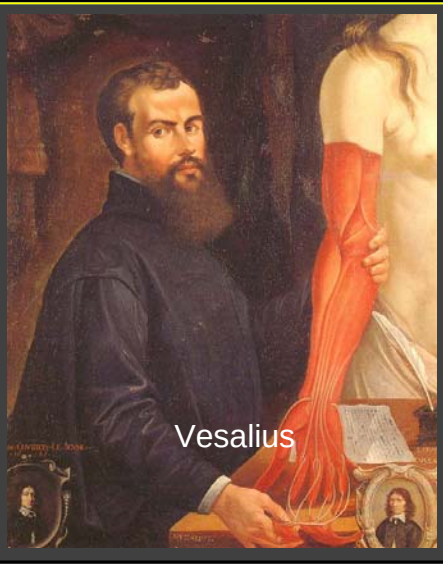


Anatomy



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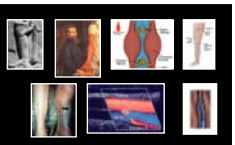
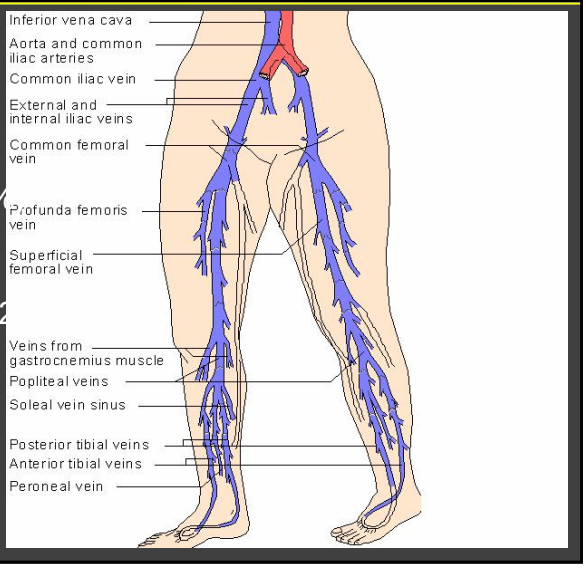


Deep Vein Anatomy

Valves of the Deep System

Com. Iliac V = 0
External iliac V = 1 (67%
Com Fem V = 1
Superficial Fem V = 3
Popliteal V = 1-2
Paired Veins BK =1v /2cm



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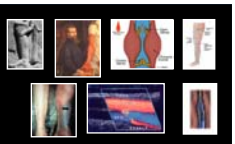
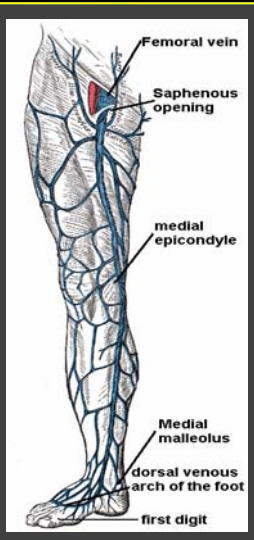
Saphenous Vein Anatomy





GSV
Minimum 6 valves
Max 14-25 valves

LSV
Median 7-10 Range 4-13

All valves more frequent BK

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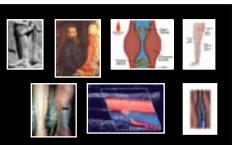
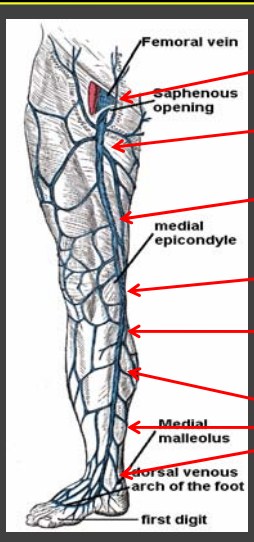
Perforator Vein Anatomy





SaphenoFemoral Junction

Huntererian perforators

Dodd's Perforators

Boyd's Perforators

Paratibial Direct at 24 cm from sole

Cockett's I,II,III perforators
Medial mal., II 7-9 cm, III 10-12 cm

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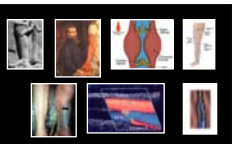

Venous Physiology

Hydrostatic P = weight of the column of blood

$P_{RA-D} = \frac{1.056 gm/ml \times 960 cm/sec^2}{1333 dynes/sec^2}$

= 0.77mm Hg /cm height

Avg male 174 cm

IVC at Rt Atrium P = 0
Malleolus ~ 122 cm P = 94 mm Hg

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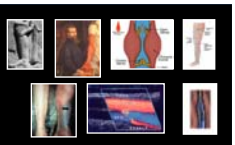

Venous Physiology

Pressure is transmitted to the Skin and SubCut

Venous capacitance maxs at ~ 30 mm Hg

30 mm Hg starts the microcirculatory changes associated with inevitable fibrosis and ulceration

Hydrostatic Pressure is transmitted through the valvless capillary bed favoring transudates

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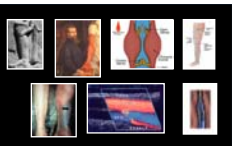
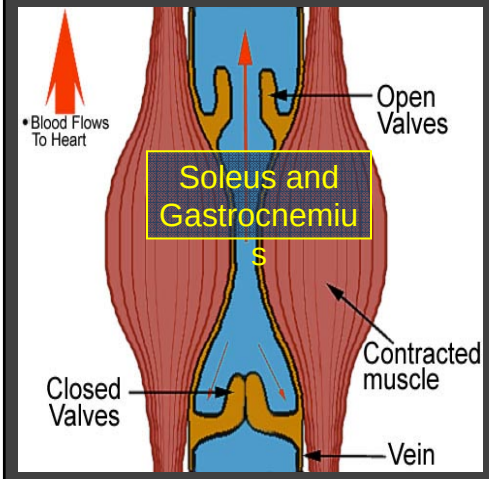
Venous Physiology





Calf muscle Pump

60% ejection fraction = 3cc/100ml tiss.

Pressure lowered further by repetitive contractions

After Contractions stop:
31 secs to achieve normal hydro. Press.
90% of volume takes > 70 secs

Pump is primed by the foot vein plexus

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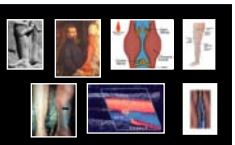
Venous Physiology



Human
Resting Hydro P drops to 22 mm Hg within 12 steps
Calf Pump generates 230 mm Hg
Fascial envelope = 150 mm Hg

The rigid fascial compartment prevents increased capacitance adaptation, hence blood is pushed to a low pressure system

The skin and subcut. tissue are elastic and will hold the transudate; interstitial pressure reaches 40-50 mm

Ulceration is linearly correlated to P> 30 mm and venous filling of > 7 mls/sec

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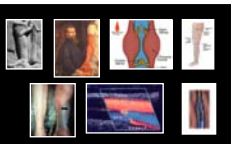
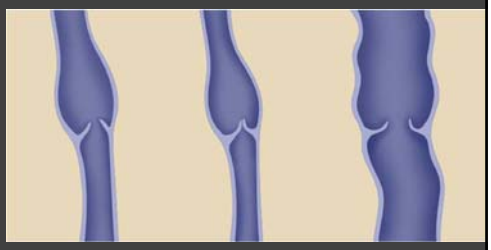


Macro Failure Mode

1. Vein valves fail
2. Results in reflux and poor drainage of venous blood
3. Column of blood causes pressure to build up in leg veins with subsequent transudate
4. Increased pressure causes surface veins to enlarge and become varicose
5. "Axial Failure Mode"



New Model

Can be Multicentric in Origin

Skip Lesions

Inflammatory role in Valve Failure

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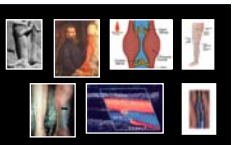

Venous Macrocirculation

Review of Macro Patho-Physiology

- Primary valvular failure with reflux – 80%
- Pure outflow obstruction - 5%
- Mixed reflux + obstruction – 15 %
- 85% Superficial Veins
- 15% Deep venous system But has normal hemodynamics
- C_5EAP and C_6EAP 43% will have Super. + Deep reflux
- C_6EAP (ulcers) have a history of DVT in 33%
- After DVT
  - 12 % return to normal duplex criteria
  - 41% have postphlebitic syndrome
  - 13 % develop ulcers

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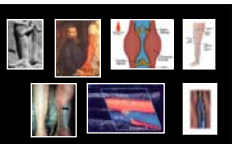
Venous Microcirculation



Micro Functional Changes with Venous Hypertension

1. Reduction / reversal / Stagnation of blood Flow
2. Increased Capillary Pressure
3. AV Shunting adjacent to ulcers
4. Decreased O2 tension near ulcers

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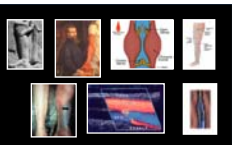
Venous Microcirculation



Anatomic Changes secondary to Venous Hypertension

1. Microlymphangiopathy
2. Dilatation and elongation of the Capillaries
3. Occlusion of capillaries by Leukocytes
4. Reduction in the number of functional capillaries
5. Increased capillary leakage of proteins and RBCs
6. Fe^{++} Deposition / WBC activation
7. Collagen Deposition/ fibrosis (> lipodermatosclerosis)

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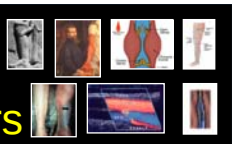
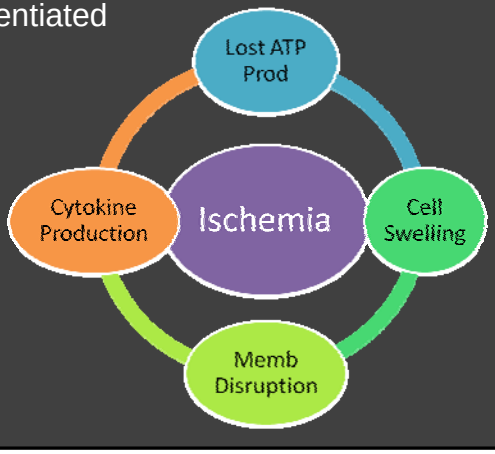


Ischemia Reperfusion in Ulcers

Implicated in both arterial and venous ulcerative breakdown
In venous ulcers, the Isch.-Reperfusion cycle is repetitive/potentiated



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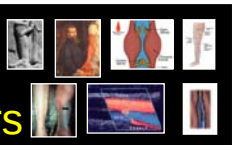
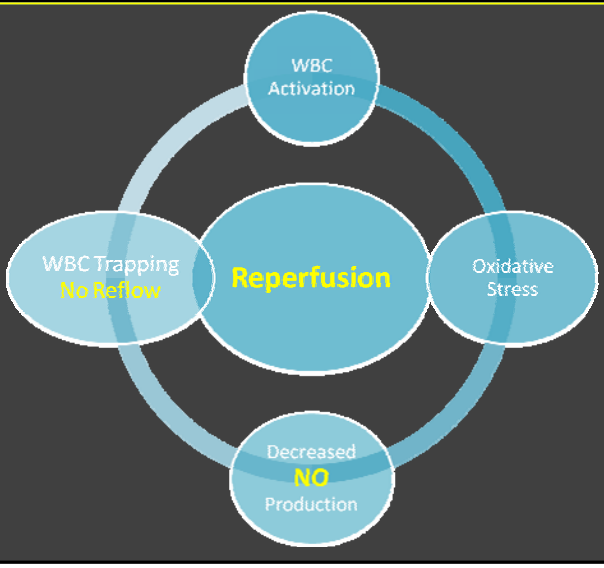


Ischemia Reperfusion in Ulcers



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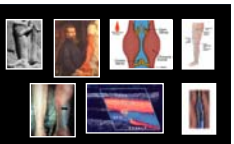
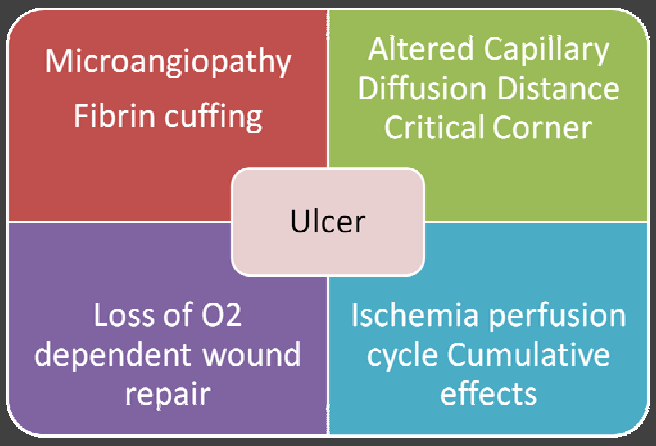


Skin Breakdown



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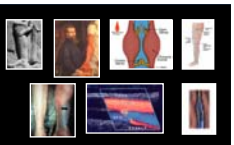
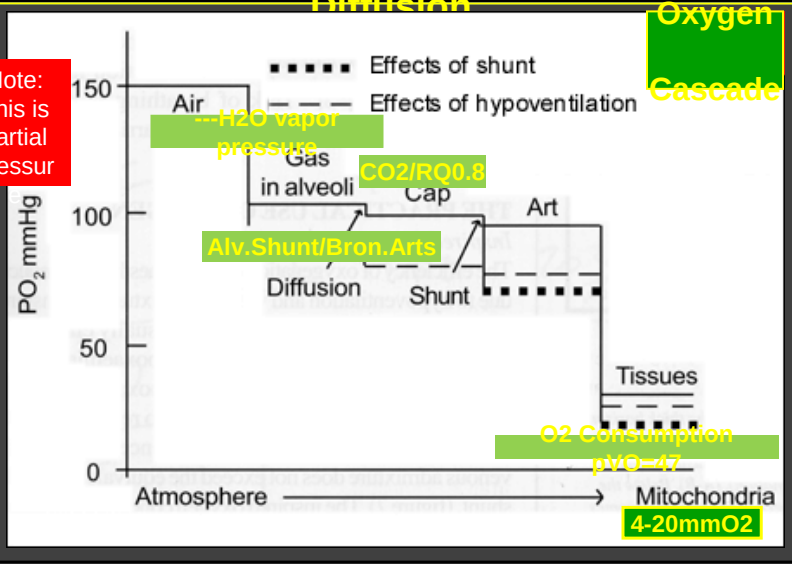


Oxygen Delivery and Diffusion

Oxygen Cascade

Note: This is partial pressur



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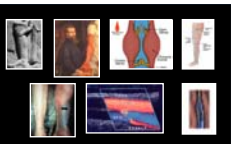

Oxygen Delivery and Diffusion

4 Fundamentals

1. **Wounds occur in subcutaneus tissue**
2. **Wound healing is dependent upon oxygen diffusion**
3. **The driving force of diffusion is partial pressure**
4. **"Normal" partial pressure O_2 = 65 mm Hg**

Hypoxia ≠ Ischemia

Diffusion Distances increase in injured tissues

Tissue beyond the diffusion distance can be ischemic in spite of having normal PaO2, O_2 Sats and CaO_2

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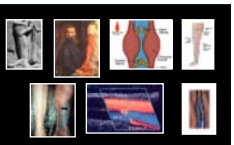
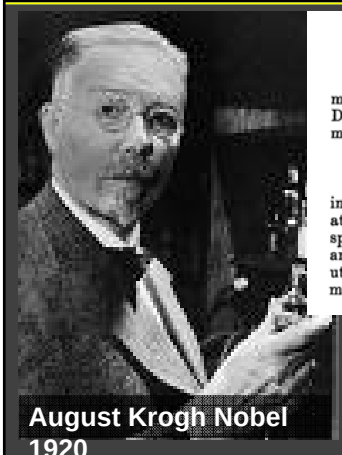


Oxygen Delivery and Diffusion



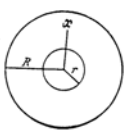
August Krogh Nobel 1920

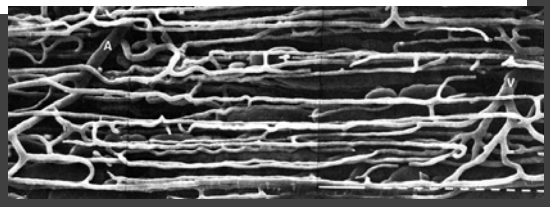
August Krogh: The Anatomy and Physiology of Capillaries, p. 270

Not being much of a mathematician myself, I have asked my friend, the Danish mathematician, Erlang, to work out such a formula for me. It runs

$$T_0 - T_x = \frac{10^4 p}{d}\left(\tfrac{1}{4}R^2 \cdot \log_{nat}\frac{x}{r} - \frac{x^2 - r^2}{4}\right)$$

in which T_0 and T_x are the oxygen pressures (in atmospheres) in the capillary and at the point x, respectively, d is the diffusion constant as defined above, and p is the number of cc. of oxygen used up per minute by 1 cc. of muscle; the distances r, x, and R are measured in cm.





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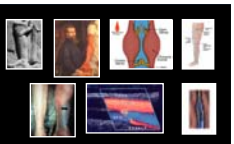
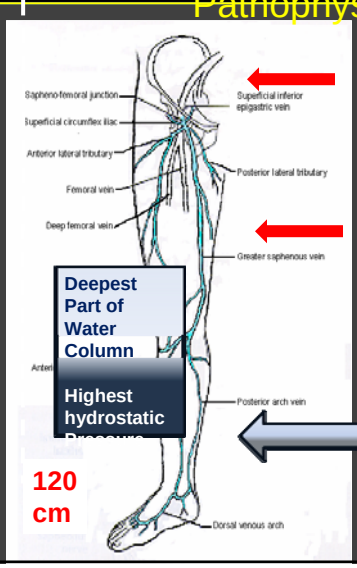


Review of Pathophysiology



- **Venous Insufficiency / Reflux**
- **Fluid Overload /Rising Tissue Pressure**
- **Swelling**
- **Micro Reperfusion Injury**
- **Leukocyte Activation / Infl.Cascade**
- **Oxidative Stress / O2 radicals**
- **Skin Ulcers/Eczema/ lipodermatosclerosis**

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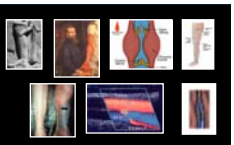

Prevalence

An estimated 25 million people in the U.S. have varicose veins

- ❖4.5 million new diagnoses are made annually
- ❖2 to 6 million have more advanced forms of venous insufficiency (swelling or skin changes)
- ❖Nearly 500,000 have painful venous ulcers
- ❖*Only 1.7 million actually seek treatment*

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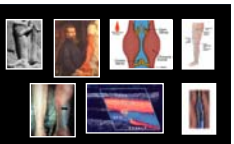

Prevalence

- In the U.S. it is estimated that 72% of women and 42% of men will experience varicose veins by the time they are in their 60s
- Prevalence is highly correlated to age and gender
- Over 2 million working hours are lost annually in the US and $1 billion is spent each year on venous ulcers alone

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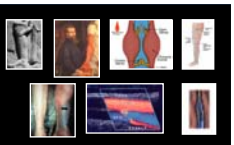
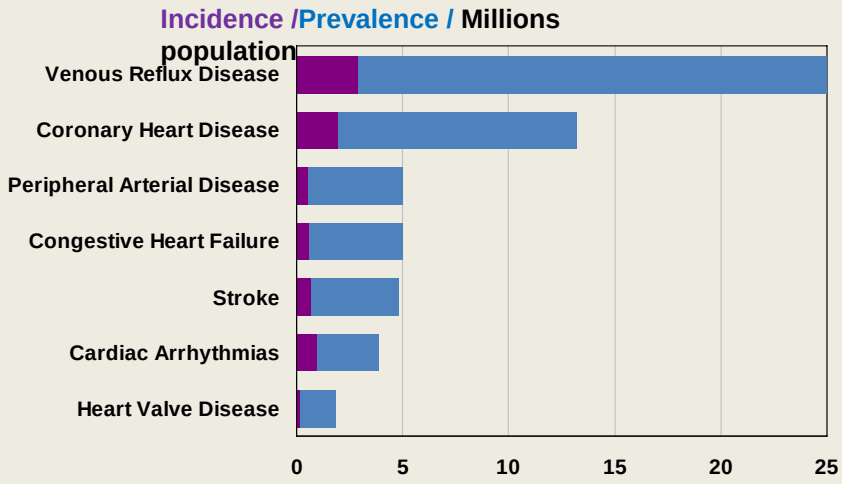


Vein Disease in Perspective



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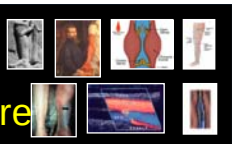

Mechanisms of Diagnostic Failure

Failure to recognize;

- Saphenous System Reflux present in 85% patients with Sxs
- High Quality Duplex scanning is NOT “r/o DVT”, Must Look for Reflux/Obstruction
- The disease is always progressive

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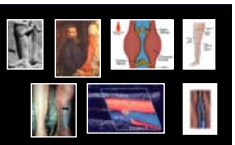

Signs and Symptoms

- Itching
- Burning
- Swelling
- Aching
- Fatigue
- Night Cramps
- Restless Leg Synd.
- Recurrent Cellulitis
- Non healing ulcers

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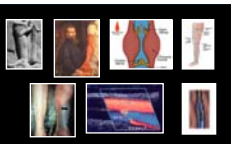

Revised CEAP Classification

Consensus Statement J Vasc Surgery Vol 40, No 6

Clinical Classification (0-6)

A classification system for visible and palpable signs of venous disease
Does not assess or classify subjective symptoms such as leg pain, aching, heaviness, etc.

Etiology
Anatomy
Pathophysiology

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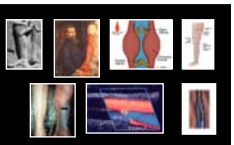

Revised CEAP Classification

Consensus Statement J Vasc Surgery Vol 40, No 6

C 0 No visible Disease

C 2 Varicose Veins

C1 Spider Reticular

C3 Swelling Edema

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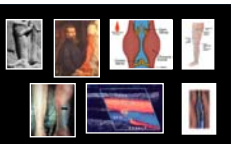

Revised CEAP Classification

Consensus Statement J Vasc Surgery Vol 40, No 6

C 4
Pigmentation/Ecz.

C 5
HealedUlcers

C 6 Active Ulcer

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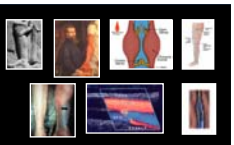

Treatment Options

Compression Therapy

Egyptian / Hippocrates 5th Century B.C.

Effects

- Reduction in Intradermal Edema
- accelerates blood flow in capillaries

Requires minimum pressure of 30-40 mm Hg

Long term patient compliance is very low

80% Effective in healing if done under strict controls

Steep Learning curve and difficult to apply

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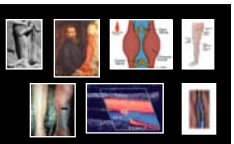

T.E.D. Hose

Cheap, Old School, Obsolete

14-18 mm Hg

Non gradient ; constant pressure = non physiologic

Designed to increase flow velocity in the supine patient

Tourniquet effect at knee ; exacerbating edema

Does not address venous hypertension

Ineffective compared to SCDs, Gradient Stockings

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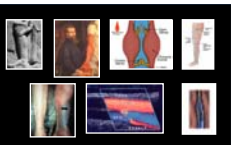

Compression Therapy Failure

Guidelines for the treatment of Venous Ulcers. WoundRepRegen. 2006 ;649-662 C Wound Healing Society

"..compressive therapy.. Does not address the increased ambulatory venous pressure (venous structural defects) that is the underlying cause of the ulcer."

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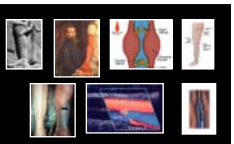

Vein Stripping (Real Old School)

- General or regional anesthesia
- Minimum of two incisions
- Ligation of all SFJ tributaries
- GSV tied to stripper and pulled out of the leg
- Recovery time of up to 2 weeks or greater

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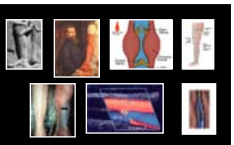

Vein Stripping Complications

Hematoma	<10%
Paresthesia 6.5%**	
Infection	<1%
Phlebitis	<.1%
Deep vein thrombosis	0.1%
Pulmonary embolism	<.1%
Lymphocele	2.5%

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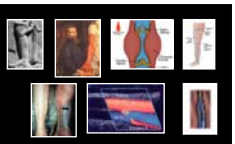
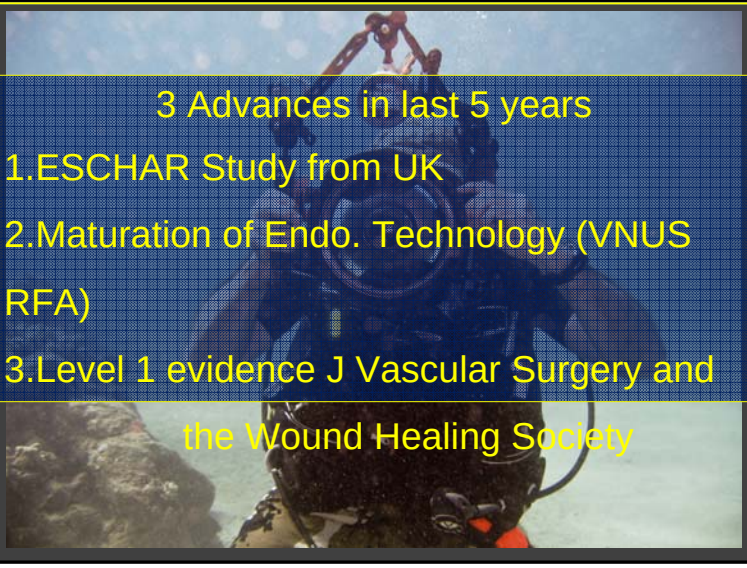


Focus on Advances



3 Advances in last 5 years

1.ESCHAR Study from UK

2.Maturation of Endo. Technology (VNUS RFA)

3.Level 1 evidence J Vascular Surgery and the Wound Healing Society

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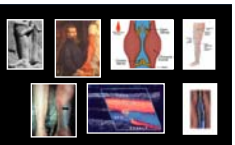

ESCHAR Study

BMJ 2007;335;83

Long term results of compression therapy alone versus compression plus surgery in chronic venous ulceration ESCHAR): randomised controlled trial

Duplex Imaging Stratification for Deep Reflux

212 legs investigated

112 multi layer compression therapy

102 compression therapy plus surgery

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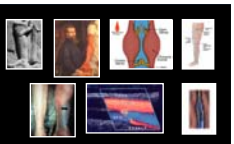

1. ESCHAR Study Results

Conclusions Surgical correction of superficial venous reflux in addition to compression bandaging does not improve ulcer healing but reduces the recurrence of ulcers at four years and results in a greater proportion of ulcer free time.

Saphenous surgery abolished deep reflux in ten of 22 legs with segmental deep reflux and three of 17 with total deep reflux. Overall median (range) VRT increased from 10 (3-48) to 15 (4-48) s 1 year after surgery (P < 0·001).

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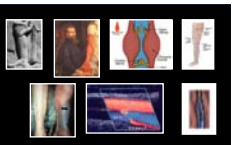
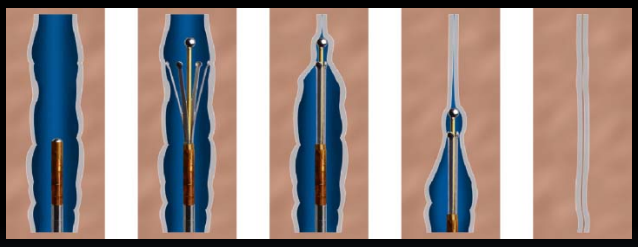


2. Radiofrequency Ablation

RFA: Resitive Heating of Collagen

Laser: Cavitation (boiling) of blood with tissue heating



Catheter inserted in refluxing vein

Catheter positioned, electrodes deployed

RF energy heats and contracts vein wall

Catheter withdrawn, closing vein

Denuded vein is physically narrowed

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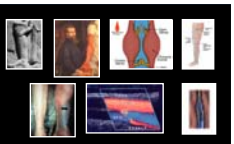
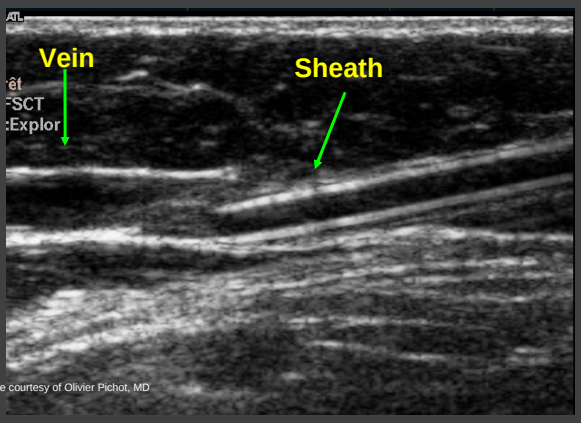
VNUS Radiofrequency





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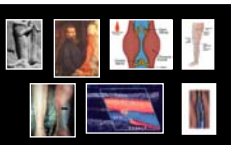
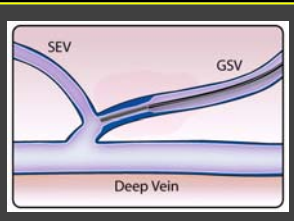
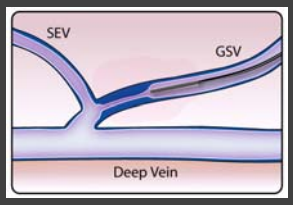
Treatment: ClosureFAST

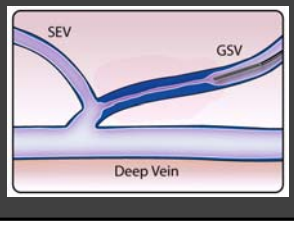




1. Apply external compression and deliver energy to vein segment

2. Withdraw catheter to next shaft marker, apply compression and deliver energy





3. Repeat withdrawal, compression and treatments until desired length treated

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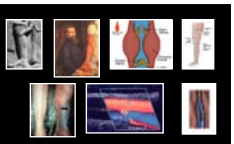
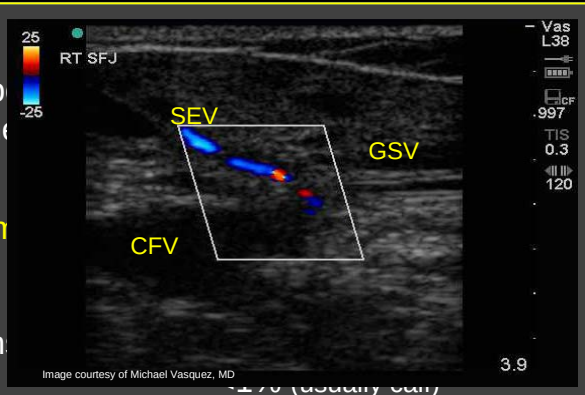


RFA Complications

Technical
Failure to access/p
Inadequate anesth

Post operative Com
Bruising
Parethesias
Infection; skin burn
DVT
Neovascularization



<1% (usually calf)
<.1% ?? Late??

Late Dilation and collateral formation

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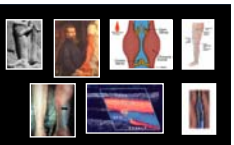
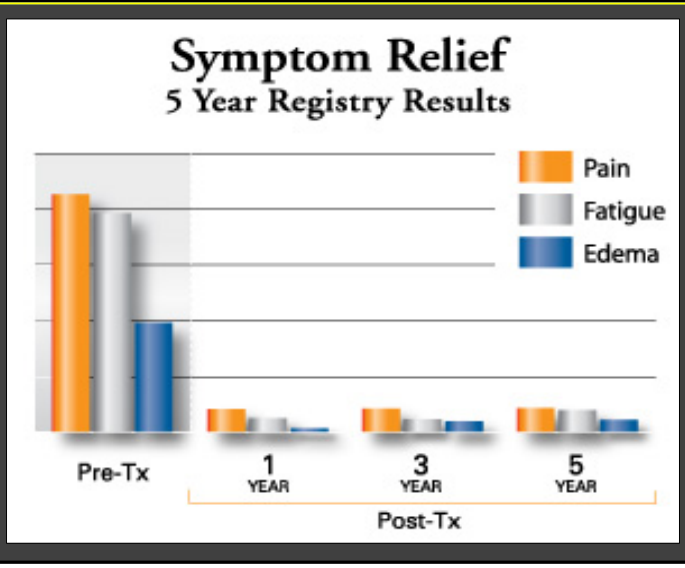


RFA Clinical Results



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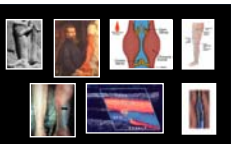

3. RFA Results

Journal of Vascular Surgery	Vol 42 no. 3 Sept 2005 p 502	1st Generation Technology

1,222 limbs treated 89% GSV Goal: Vein Occlusion / Stop Reflux

Identified 3 types of Treatment Failure 185/985 follow up of 1222

Type I Non Occlusive 12.4% of anatomic failure

Type II Recanalization 69%

Results	1 week	1year	5 years
Vein Occlusion	96%	87%	87%
Absence of Reflux	96%	88%	83%

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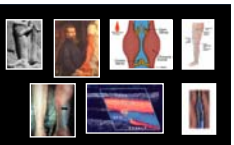

CVSPC / *Iowa Vein Clinic* Results

205 VNUS closures in GSV / LSV / Perforator

Closure Failure / Recurrence = 0

Access failure = 3

DVT = 0

Nerve injury / Paresthesias = 0

Neovascularization = 1

Bleeding / Infection / Thrombophlebitis = 0

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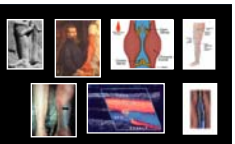

Vein Science Summary

1. Venous Insufficiency documented since antiquity
2. Venous hypertension can result in Ischemia Reperfusion
3. Relief of Venous Reflux is fundamental
4. Surgical (mechanical) Tx is superior to Compression
5. Endovascular repair is equal to or superior to surgery
6. Endovascular is better tolerated and cheaper