Varicose veins.

History.

The word varicose is derived from the Latin word "varix" which means twisted. In 1550 BC, the Ebers papyrus recommended avoidance of surgical treatment for varicose veins (VV). Hippocrates (460 – 375BC) wrote a treatise "On Ulcers". He recognised the correlation between VV and leg ulcers and recommended the use of two layers of bandages to produce firm compression for the treatment of ulcers. He stated that "*whatever cannot be cured by medication is cured by the knife, what cannot be cured by the knife is cured by the searing iron, whatever this cannot cure must be considered incurable*". Celsus (53BC to AD7) also advocated plasters and then linen bandages for leg ulcers. In addition, he recommended avulsion of VV with a blunt hook. Ambrosie Pare reported the association of VV with pregnancy. He treated ulcers by rigid diet, purgatives, bleeding and bed rest. He also used compression bandages. Richard Wiseman (1696) invented a laced stocking made of leather (preferably dog skin as it was soft) as a compression devise. Theodor Billroth (1878) stated that "*every operation upon veins may become dangerous to life by complications with thrombosis and emboli, you will agree with me when I declare that operation for varices utterly uncalled for".*

Fredrich Trendelenburg (1890) a German surgeon, described great saphenous vein (GSV) ligation at the junction of the middle and lower thirds of the thigh using a longitudinal incision. This was followed by 10 days of bed rest.

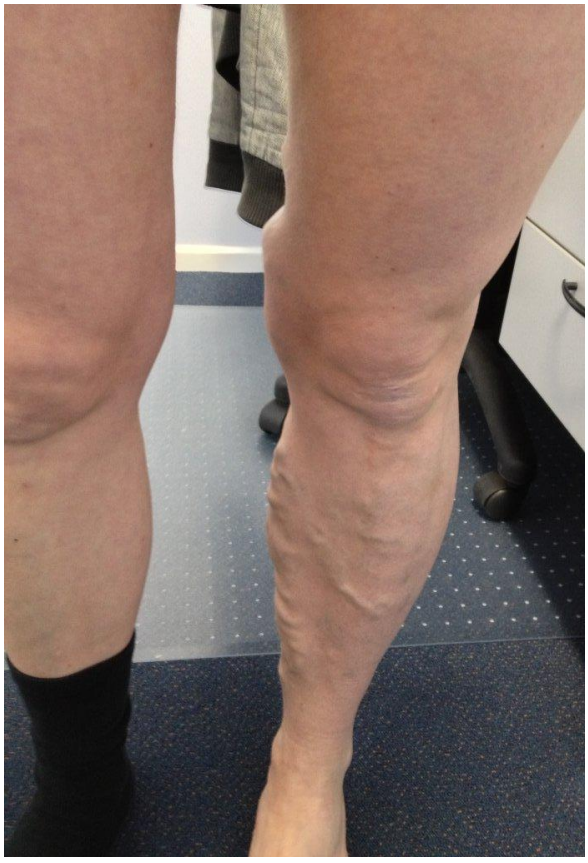


***Figure 1** – Left leg varicose veins*

Definition.
The World Health Organisation definition of VV is "abnormally dilated superficial veins."

Classification.

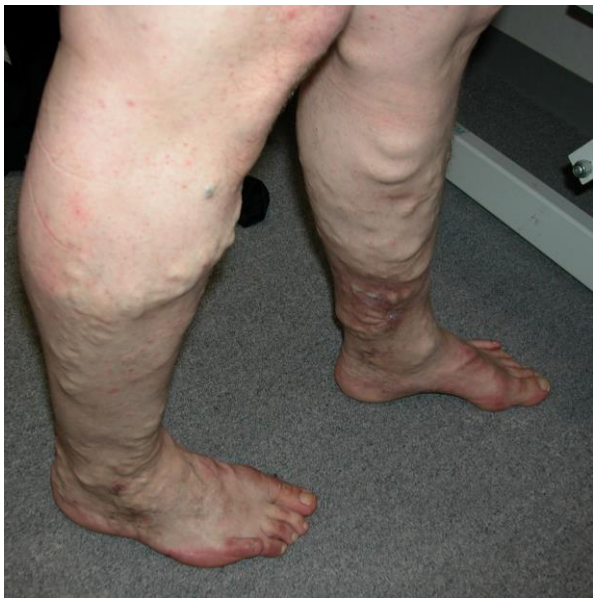
- Primary, which includes telangectasia or spider veins
- Secondary, usually postphlebitic
- Congenital venous malformations
- Acquired AV fistula

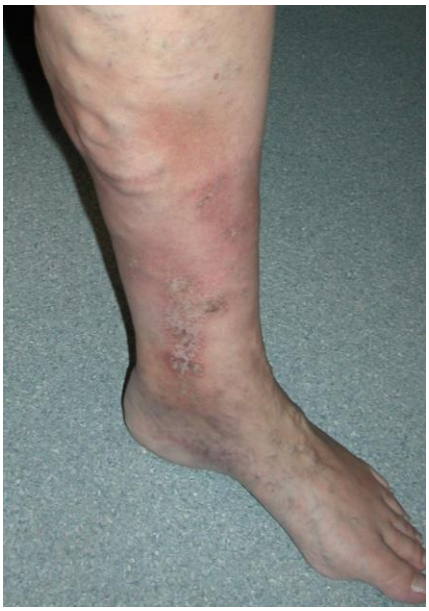
CEAP classification.
For the CEAP classification VV are defined as subcutaneous dilated vein 3mm or more in diameter which may also be tortuous.

C (clinical class)	C0	no visible or palpable signs of venous disease
	C1	spider, telangiectasies or reticular veins
	C2	varicose veins
	C3	oedema
	C4	a pigmentation or eczema
		b lipodermatosclerosis or atrophie blanche
	C5	healed ulcer
	C6	active venous ulcer
	S	Symptomatic – ache, pain, tightness, skin irritation, heaviness, muscle cramps
	A	Asymptomatic
E (etiology)	Ec	congenital
	Ep	primary
	Es	secondary
	En	no venous cause identified
A (anatomy)	As	superficial vein
	Ap	perforator
	Ad	deep veins
	An	no venous location identified
P (pathophysiology)	Pr	reflux
	Po	obstruction
	Pr,o	reflux and obstruction
	Pn	no venous pathophysiology identifiable

Reticular veins are dilated non palpable subdermal veins 1 – 3mm in diameter. Telangiectases are dilated intradermal venules less than 1mm in diameter. Telangectasia is a confluence of dilated intradermal venules. They are also called spider veins or thread veins. There is no connection between the saphenous system and the reticular network which feed telangiectatic veins. Varicose veins and telangiectases commonly co-exist and are associated with similar symptoms. There is no evidence that treating one system impacts on the treatment of the other and no evidence to suggest which system should be treated first (Ruckley *EJVES* 2008).
Atrophie blanche is localised whitish atrophic skin surrounded by dilated capillaries and sometimes hyperpigmentation (Figure 2b). It is a sign of severe Chronic Venous Disease (CVD).
Corona phlebectatica is a fan shaped pattern of small intradermal veins which used to be called an ankle flare.
Eczema is an erythematous dermatitis which may progress to blistering, weeping or scaling.

Lipodermatosclerosis is localised chronic inflammation and fibrosis of the skin and subcutaneous tissue. It is associated with scarring and contracture of the Achilles tendon.
Pigmentation results from extravasation of blood and deposition of haemosiderine.
Venous ulcer – full thickness skin defect.





A **b**

Figure 2a*. Varicose veins with lipodermatosclerosis.* ***B.*** *lipodermatosclerosis and atrophie blanche*

Venous clinical severity score (Vasquez JVS 2010).
Designed for serial assessment of venous disease.

	None = 0	Mild = 1	Moderate = 2	Severe = 3
Pain, aching, heaviness, fatigue, soreness, burning of venous origin		Occasional, not restricting regular daily activity	Daily, interfering with but not preventing regular daily activities	Daily, limits most regular daily activities
Varicose veins – more than 3mm diameter in standing position		Few, includes corona phlebectatica (more than 5 blue telangiectasias)	Confined to calf or thigh	Involves calf and thigh
Venous oedema		Limited to foot and ankle	Extends above ankle but below knee	Extends to knee and above
Skin pigmentation due to venous disease	None	Limited to perimalleolar area	Diffuse over lower 1/3 of ccalf	Wider distribution above lower 1/3 of calf
Inflammation, cellulitis, erythema, venous eczema, dermatitis		Limited to perimalleolar area	Diffuse of lower 1/3 of calf	Wider distribution above lower 1/3 of calf
Induration, chronic oedema with fibrosis, includes white atrophy and lipodermatosclerosis		Limited to perimalleolar area	Diffuse of lower 1/3 of calf	Wider distribution above lower 1/3 of calf
Active ulcer number	0	1	2	3 or more
Active ulcer duration		Less than 3 months	3 months to 1 year	More than 1 year
Active ulcer size		Less than 2 cm	2 – 6cm diameter	More than 6cm

				diameter
Use of compression therapy	Not used	Intermittent	Wears stockings most days	Full compliance

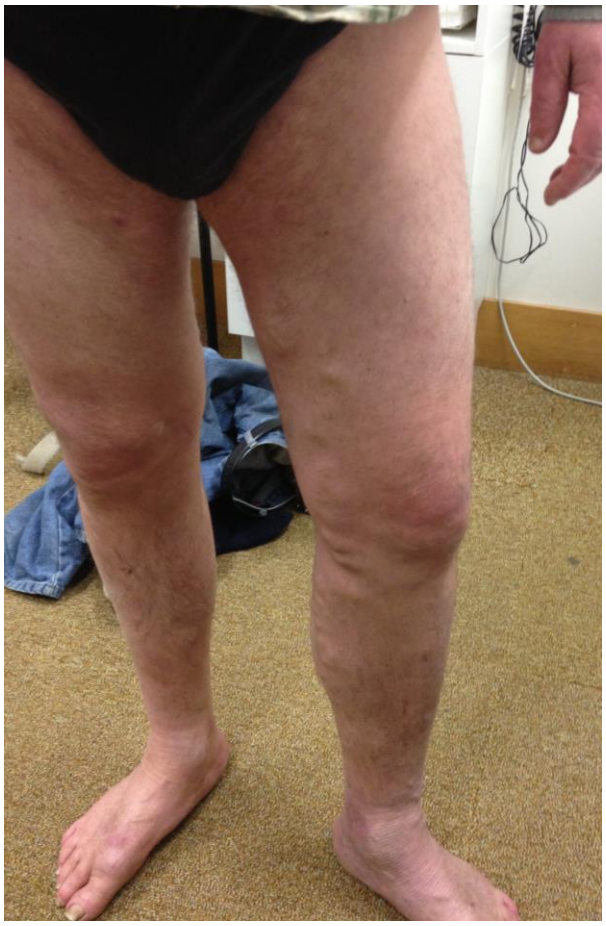


Figure 3. Varicose veins in distribution of left great saphenous vein

Epidemiology.

Varicose veins are common – 40% of men and 32% of women aged 18 – 64 years have VV (Campbell *BMJ* 2006). 50% of the population over 40 years have some form of VV. 0.5% have superficial varicosities with associated venous stasis and ulceration. 10 - 15% of men over 15 years of age and 20 – 25% of women have VV. In the Basel study of 4500 healthy working people, 55% were found to have some form of venous disease.

Risk factors:

- Age – over 50 years. Edinburgh Vein Study found that the prevalence increased from 11.5% in 18 – 24 year olds to 55.7% in 55 – 64 year olds.
- Family history – in 70 – 85%. If both parents have VV, 90% develop VV, if one parent was affected, males have a 25% chance and females 62% chance of developing VV, compared to 20% chance if neither parent was affected. Inheritance appears to be autosomal recessive.
- Sex – F:M 6:1 decreasing to 2:1 with advancing age. Framingham study showed prevalence of 1% in men vs. 10% in women less than 30 years and 57% vs. 77% in men and women over 70 years. Thought to be related to high oestrogen levels.
- Multiparity – VV usually occur in first three months of pregnancy. Progesterone inhibits SMC contraction in the vein wall. May also be due to mechanical obstruction and fluid retention. Parity with three or more births is an independent risk factor for VV.
- Oral contraceptive -
- Standing vocation – more than 4 hours of standing per day. Prolonged sitting is also a risk.

- Obesity – especially in women. May be related to increased intra abdominal pressure. Gastric bypass surgery has been shown to correct venous stasis in almost all patients.
- Constipation –
- History of DVT
- Smoking

Anatomy.

The superficial and deep veins of the leg are joined via communicating and perforating veins. The superficial veins are within the superficial compartment of the leg bounded deeply by the muscular fascia and superficially by the dermis. This tissue is called tela subcutanea or subcutaneous tissue. The subcutaneous tissue contains the saphenous veins and their tributaries. Within the subcutaneous tissue is a separate saphenous compartment bounded superficially by the saphenous fascia and deeply by the muscular fascia. The saphenous fascia contains the saphenous veins, accompanying arteries and nerves. Saphenous tributaries, accessory, collateral and communicating veins lie external to this compartment.

Perforating veins perforate the muscular fascia to connect superficial and deep veins. Communicating veins interconnect veins within the same system i.e. either deep or superficial.

The direction of flow is normally from superficial to deep, aided by valves within the perforating veins and the function of the calf muscle pump.

Duplication of the great saphenous vein (GSV) occurs in 10%.

The confluence of superficial inguinal veins corresponds to the veins of the saphenofemoral junction. These tributaries include the external pudendal vein, the superficial circumflex iliac vein, the superficial epigastric vein, the superficial dorsal vein of clitoris or penis, anterior labial or scrotal veins.

An anterior accessory great saphenous vein can be found anterior to the saphenous compartment and a posterior accessory vein posterior to the saphenous compartment. The later used to be called Leonardo's vein or posterior arch vein.

The term superficial accessory great saphenous vein is for any segment ascending parallel to the GSV but more superficial than the saphenous compartment or saphenous fascia. Similarly, the superficial accessory small saphenous vein ascends parallel to the SSV but superficial to the saphenous fascia.

The anterior thigh circumflex vein is a tributary of the GSV or anterior accessory great saphenous vein which ascends the anterior thigh obliquely, sometimes originating from the lateral venous system. Similarly, the posterior thigh circumflex vein ascends obliquely in the posterior thigh. It may originate in the SSV, in its cranial extension or in the lateral venous system.

The cranial extension of the small saphenous vein courses in the groove between biceps femoris and semimembranousus muscles to end in the inferior gluteal vein. A cranial extension of the SSV that communicates with the GSV via a posterior thigh circumflex vein is often called the vein of Giacomini.

There may be intersaphenous veins connecting the GSV and SSV in the calf. The lateral venous system represents the remnant of the embryonic vena marginalis lateralis.

Absence of ileofemoral venous valves is found in 40% of normal population.

In patients with GSV incompetence, this incompetence starts at the saphenofemoral junction (SFJ) in approximately 70%. In the remainder, the SFJ is competent but the more distal GSV is incompetent below the subterminal valve.

The common femoral vein is formed by the femoral vein and the deep femoral (or profunda femoris) veins. The femoral vein originates from the popliteal vein.

The perforators of the leg are divided into:

- Para-tibial – connect the GSV with the posterior tibial veins, close to the medial surface of the tibia (Sherman in the lower and mid leg and Boyd in the upper leg).

- Posterior tibial - (Cockett) connect the posterior accessory great saphenous vein with the posterior tibial veins.
- Anterior leg perforators – pierce the anterior tibial compartment and connect to anterior tributaries of the GSV to anterior tibial veins.
- Lateral leg perforators connect veins of the lateral venous plexus with the fibular veins.
- Medial gastrocnemius perforators
- Lateral gastrocnemius perforators
- Intergemellar perforators – connecting the SSV with calf veins, also called mid calf perforator of May.
- Para Achillean perforators – connecting SSV with fibular veins, also called perforator of Bassi.
- Medial Perforators of knee
- Suprapatellar perforators
- Lateral knee perforators
- Infrapatellar perforators
- Popliteal fossa perforators
- Perforators of the femoral canal – Dodd
- Anterior thigh perforators – pierce quadricepts femoris
- Lateral thigh perforators
- Posteriomedial thigh perforators – pierce adductor muscles
- Sciatic perforators – in midline of posterior thigh
- Posterolateral thigh perforators – pierce biceps femoris and semitendinosis (perforator of Hach)
- Pudendal perforators

Pathophysiology.

Various theories for developing VV include:

- Arteriovenous communications – suggested by the pO_2 in VV being increased when compared to normal veins. Increased pressure and turbulence may lead to vein dilatation.
- Valvular incompetence – valve incompetence leads to retrograde flow, increased pressure and vein dilatation. But VV can also occur below competent valves.
- Vein dilatation leading to valvular incompetence.

The media of vein walls consists of 3 layers of SMC: an inner longitudinal layer, which is thickened at valve sites; a circular layer; and an outer longitudinal layer. The inner and outer layers interdigitate with scafolds of extra cellular matrix (ECM) composed of collagen, elastin, SMC and proteoglycans. The adventitia is composed of irregular longitudinal muscle fibres, collagen, fibroblasts, SMC and vasa vasorum. In VV, there are vein wall structural abnormalities with hyperplasia of the endothelium, increased collagen, decreased elastin and disruption of SMC architecture. The SMCs become enlarged. The adventitia has increased SMC, fibroblasts and collagen. Thrombus in various stages of organisation may be found. Matrix metalloprotinases (MMPs) may play a role. The vein wall shows alternating thickening and thinning with aneurysm formation. There are increased inflammatory cells (mast cells and monocytes) found in the vein wall. Replacement of smooth muscle by collagen (I and III) is characteristic of VV. There is also fragmentation and attenuation of elastic fibres.

In VV there is increased fibrinolytic activity, increased prostaglandin production, increased endothelin 1 production and reduced expression of endothelin B receptor.

Secondary VV are usually due to deep venous thrombosis. Valve damage leads to reflux. Perforators are often also damaged and lead to incompetence.

VV appear to be linked to a candidate marker D16S520 on chromosome 16q24. VV are also associated with the FOXC2 gene. Kim has identified three complementary deoxyribonucleic acids (cDNA) prominently expressed in patients with VV. These cDNAs have similarities to the L1M4 repeat sequence of clone RP11-57L9, RP11-29H13 and Alu repetitive sequence of human tropomyosin 4 messenger RNA, suggesting that altered expression of these elements affects the structure and function of the vein wall by altering the actin binding proteins involved in the assembly and regulation of the cells contractile mechanisms (Kim J *Surg Res* 2005).
Segmental GSV hypoplasia occurs in limbs with VV with SFJ incompetence more frequently than in healthy limbs (25% vs. 12%). The hypoplasic segment does not permit venous reflux

The oedema seen in those with varicose veins may be due to lymphatic dysfunction (Suzuki *ESVS* Nice 2008).

Varicose veins demonstrate atrophy of valves and widening of the valvular annulus. The valves contain less collagen.

Structural changes in the vein wall contribute to pathological weakening and resultant dilation. Overproduction of collagen type I, decreased synthesis of collagen type III, and disruption of the arrangement of smooth muscle cells and elastin fibers have been observed in histological studies of varicose venous segments. Increased levels of tissue inhibitors of MMP's observed in VV specimens may favour the deposition of extracellular matrix material in the vein wall. Increased levels of transforming growth factor β1 and fibroblast growth factor β have also been observed in the walls of varicose veins and may contribute to structural degradation.

In animal models, the concentration of neutrophils, monocytes, macrophages, and lymphocytes and levels of MMP's increased in venous valves exposed to high pressures for prolonged periods of time. Over time, the venous valves exposed to high pressures demonstrated adverse remodelling with decreases in leaflet length and thickness. Turbulent flow, reversal of flow, and decreases in shear stress promote inflammatory and prothrombotic changes that may further contribute to loss of structural and functional integrity of the vein wall and valve leaflets.

Congenital
Klippel Trenaunay syndrome – Maurice Klipple was a French neurologist and Paul Trenaunay was a physician who described this condition in 1900. It is a congenital, non hereditary venous abnormality without AV fistulas. Varicose veins (72%) are found on the lateral aspect of the thigh, there is a port wine stain (98%), and there can be unilateral soft tissue and bone overgrowth (67%). Recent studies suggest a defect in VG5Q gene, an angiogenic factor. It can affect the upper limb in 5 – 10%. There is a 10 fold increase in post operative thromboembolism.

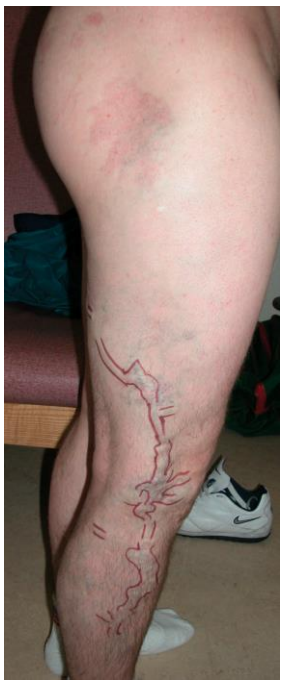


Figure 3. Kipple Trenaunay syndrome in a young man. Note port wine stain on upper thigh.

Parks Weber syndrome – Similar to Kippel Trenaunay syndrome except that there are AV fistulae. First described by British dermatologist Frederick Parkes Weber in 1918. This is a high flow arteriovenous malformation with cutaneous, subcutaneous and intramuscular involvement. Usually affects a single limb, 95% lower limb. May be assictaed with sinal AVM's. Many patients have mutation in RASA1 gene

CLOVES syndrome.
Congenital, lipomatous, overgrowth, vascular malformations, epidermal nevi and spinal/skeletal anomalies and or scoliosis.

Chuvash polycythaemia – 74% of patients with this disorder have VV. Autosomal recessive disorder caused by Arg200Trp mutation (C598T) of the Hippel-Lindau gene which causes dysfunction in oxygen tension sensing. Endemic in Chuvashia (Russia) and Ischia (Italy).

Natural history.
The natural history of VV remains unclear. It has been suggested that 3 – 6% of patients with VV will develop venous ulcers. It has also been suggested that 28.6% of those with VV progress to more serious venous disease after 6.6 years.

Clinical findings.
The relationship between VV and symptoms is controversial. Whilst it has been suggested that VV may cause aching pain, leg fatigue, leg heaviness, itching, symptoms worst on the first day of the menstrual period, swelling, cramping, throbbing, bleeding, skin changes and ulceration, asymptomatic superficial venous reflux is found in 39% of the population. In the Edinburgh Vein study, lower limb symptoms were common irrespective of the presence of VV with 48% of all women complaining of aching legs (Bradbury *BMJ* 1999). The majority of lower limb symptoms have a non venous cause. Symptoms which are more suggestive of venous origin are aching, itching and heaviness. These should be relieved by elevation. In a study by Campbell (*EJVES* 2007) symptoms responded poorly to surgery but there is evidence that surgery for VV improves quality of life (Michales *Health technology assess* 2006). There is some evidence that there is an exaggerated sense of the risk of varicose veins in patients (NICE 2013) together with high expectations of treatment success.

In a study by Daravel in 2009, at 6 months after surgery for VV the percentages where pre-operative expectations were not met: Symptoms Pain 20% Itch 21% Tingling 18% Cramp 23% Restless legs 22% Swelling 27% Heaviness 18% Other factors Appearance of the legs 12% Choice of clothes that can be worn 25% Performance at work 25% Relationships 14% Enjoyment of leisure activities 30%.

Conway (*Vascular* 2011) reported a lack of association between disease severity as graded by CEAP classification and patient reported symptoms. This differs from the study of Chiesa (*JVS* 2007) where symptoms were found to correlate with CEAP classification.
Signs of chronic venous insufficiency (swelling, pigmentation, oedema in the gaiter area) are more common with secondary VV.
Only 40% of patients with superficial venous reflux on duplex have varicosities on examination.
Hand held Doppler examination has an accuracy of only 70%.
There have been reported deaths from bleeding from varicose veins, but this is exceedingly rare (Jelev *EJVES* 2011).

In those presenting with VV, 73% have saphenofemoral junction incompetence in duplex scan, 13% have short saphenous incompetence (Conway *Vascular* 2011).

The Aberdeen Varicose veins score is said to be a useful tool in the assessment of chronic venous disease and quality of life.

Drugs.
Venacura.
The manufactures claim that Venacura is clinically proven to reduce appearance and discomfort associated with spider veins. It contains Diosmin, a bio-flavinoid. It is a once a day pill (600mg). It is said to reduce spider veins, relieve swelling and inflammation of the legs, reverse chronic venous insufficiency, relieve leg ulcers and relieve haemarroids. I do not believe a word of it.

Calcium dobesilate (Doxium).
Has been shown to reduce cramps and restless legs.

Flavonoids.
Decrease leg oedema.

Horse chestnut seed extract.
May help with leg oedema.

Compression therapy.
Improves both symptoms and venous haemodynamics. Particularly useful in reducing oedema. Used for chronic venous insufficiency and symptomatic VV but compliance is variable.

Table 1. Compression stocking classification.

	UK standard	*European standard*
Class 1	14 – 17 mmHg	20 – 30 mmHg
Class 2	18 – 24 mmHg	30 – 40 mmHg
Class 3	25 – 35 mmHg	40 – 50 mmHg

A systematic review has reported that compression significantly reduced pain scores when compared to placebo. Heaviness, cramps and swelling are also improved.
Because of the poor quality of evidence for the benefit of compression stocking, the 2013 NICE guidelines had a real downer on compression stockings. However, the American College of

Phlebology guidelines state that compression therapy is an effective method for the management of symptoms related to superficial venous disease.

Sclerotherapy.

Traditionally reserved for residual VV following surgery and small veins such as telangiectasia and reticular veins. Sclerosing agents:

Polidocanol (Aethoxysclerol) -
Sodium tetradecyl sulphate (STD) (Fibrovein). Maximum dose – 10 – 15ml of 1% solution.
Hypertonic saline (20 – 24%) -
Sodium morrhuate -
Historical agents – ferric chloride, iodine, glycerine.

Cochrane reviews:

Injection sclerotherapy for varicose veins – type of sclerosant, local pressure dressing, degree and length of compression have no significant effect on efficacy of sclerotherapy. Sclerotherapy should be limited to recurrent VV and thread veins.

Technique - Vein should be empty of blood.

Complications – pigmentation, cutaneous necrosis, superficial thrombophlebitis, allergic reaction. Recurrence of new lesions is usually found at different locations.

Results - In a randomised trial of sclerotherapy vs. surgery, with sclerotherapy 82% were cured at one year but only 7% were cured at six years. Results were better with non truncal VV. Foam sclerotherapy may improve the results (Alos *EJVES* 2006).

Ultrasound guided foam sclerotherapy (UGFS).

First described by Cabrera in the early 1990's. There is a reduced risk of nerve injury when compared to surgery. It is usually reserved for veins less than 5- 7mm in diameter but this is not a universal stipulation. Foam is created by mixing the sclerant with air in a ratio of 1:4 or 1:5 (2ml of 1% polidocanol or 3% aethoxysclerol with 6mls of air). The foam is made simply by passing the liquid (2ml) from one syringe to another (6ml) a number of times through a three way tap. It is thought that the foam gives better endothelial contact as it has a greater surface area than just the liquid. An 18, 20 or 25 gauge IV cannula or needle is inserted into the vein. I find that a micropuncture kit or a conventional 4 French angio sheath is useful in patients with fat legs (deep veins). The vein is cannulated under ultrasound guidance and foam is injected with ultrasound monitoring. Compression of the SFJ with the ultrasound probe should prevent foam escaping into the deep system. When the foam is moving too slowly, asking the patient to take some deep breaths can improve the flow. With leg elevation to 45 degrees, the incidence of "embolic" complications is reduced (Bergan *Vascular* 2007). Graduated compression stocking then applied and worn for one week. Also useful for recurrent SFJ (Darvall *EJVES* 2011) and saphenopopliteal incompetence.

There have been case reports of visual disturbance during the procedure, which may be related to a patent foramen ovale which is present in 10 - 20% of the population, and DVT's (up to 6%). Skin pigmentation occurs in 32%, cutaneous necrosis in 1%, allergic reaction, lower back pain in 4% (NICE guidelines 2007). Other side effects – chest tightness, dry cough, dizziness.

Using carbon dioxide instead of air is said to reduce the incidence of complications and using physiological gas (70% carbon dioxide, 30% oxygen) was shown to improve the success rate and reduce the incidence of skin staining (Beckitt *EJVES* 2011 from Plymouth). However, in a debate at Charring Cross 2014, 60% voted that air was the best gas for making foam.

In a randomised trial of conventional surgery vs. UGFS (Figueiredo *EJVES* 2009), 3 months treatment success was noted in 78% who had USGFS compared to 90% following surgery.
In a randomised study of 3 layers of Pehahaft bandage (STD pharmaceuticals UK) plus TED stocking for 24 hours then TED stocking for two weeks vs. 5 days of the bandages and TED stocking plus 2 weeks of TED stocking, there was no advantage for the longer bandage regime when phlebitis or skin discoloration were compared (O'Hare *BJS* 2010).

Varithena is a commercially available foam made from polidocanol.

Surgery.
In a randomised clinical trial comparing surgery with conservative treatment for uncomplicated varicose veins, surgical treatment provided better symptomatic relief and significant improvements in quality of life (Michaels *BJS* 2006).

Technique.
The SFJ is usually one inch lateral to and one inch below the pubic tubercle. The lower edge of the fossa ovalis, through which the GSV passes, contains the superficial external pudendal artery which may need to be divided to fully expose the SFJ. The medial posterior tributary and the lateral anterior tributary are important in SFJ ligation. Tributaries should be dissected back to their first branch prior to ligation. Some would advocate ligation of the medial deep perforating branch, but others feel this is not required. The SFJ is ligated with a vicryl transfixion suture and the stump then oversewn with prolene (see below). GSV stripping reduces recurrence rates from 40% to 17%. The GSV is bifid in 10% and both should be stripped. Inversion stripping reduces the morbidity of stripping. The GSV tunnel is then flushed with local anaesthetic and adrenaline using an infant feeing tube as this is reported to reduce haematoma formation.

In short saphenous surgery, the saphenopopliteal junction should be marked by pre operative duplex scanning because of its variable positioning. Beware of the popliteal perforating vein, which joins the popliteal vein laterally, as being the source of short saphenous varicosities and may require ligation. The fact that this tributary to the popliteal vein is often missed may account for some recurrences in short saphenous surgery (De Palma *Phlebology* 2006). The short saphenous vein is generally not stripped due to the risk of nerve injury but it can reduce recurrent saphenopopliteal incompetence at one year from 33% to 13% (Earnshaw *The Evidence for Vascular Surgery* 2007).

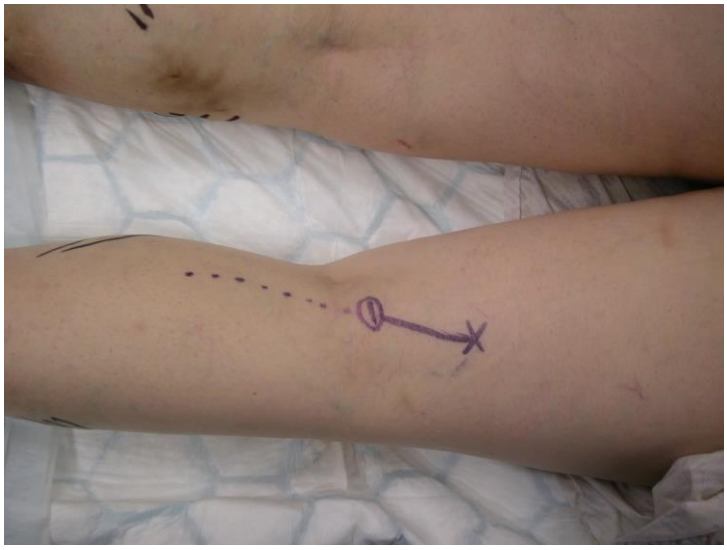
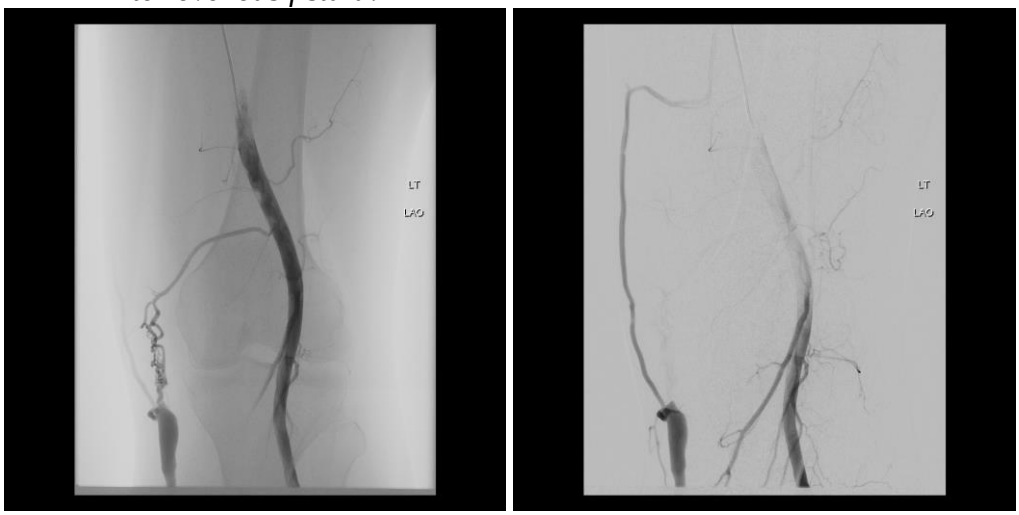


Figure . *Pre operative duplex marking of a high saphenopopliteal junction – X marks the junction.*

Complications.

- *Femoral vein and femoral artery injuries* – 0.02%.
- *Recurrence* – 7 – 10% at one year and 21% over 10 years. Initially attributed to inadequate groin dissections or inadequate surgery. In a randomised controlled trial, pre operative duplex scan was shown to reduce the rate of recurrence and redo surgery (2% vs. 9% at two years) but increased the overall costs of surgery (Blomgren *Phlebology* 2006). Groin recurrence has been shown to be increased with larger groin wound incisions. Incisions of less than 3cm have the lowest recurrence rate. But, there is also a correlation between smaller groin incisions and incomplete SFJ ligation. In a randomised study, use of an absorbable suture to ligate the SFJ followed by Prolene to close the stump had the lowest neovascularisation rate (3% vs. 11%), (Frings *EJVES* 2004). Closing the cribriform fascia or pectiueus fascia does not reduce the incidence of recurrence. Using a prosthetic patch over the ligated junction did not reduce the incidence of recurrence (Winterborn *EJVES* 2007). Operative mistakes occur in 5% and can be excluded on a post operative scan (3 months). Neovascularisation can be due to dilatation of pre-existing veins e.g. in vasa vasorum of femoral vein or true angiogenesis, possibly stimulated by the residual exposed endothelium, groin haematoma or groin wound infection.
- *Infection* – Up to 13% groin wound infection rate. In a randomised trial, IV antibiotics significantly reduced wound related problems following VV surgery (Coughlin *ESVS Abstracts* 2006). This has also been shown in retrospective studies (Singh *Ann Vasc Surg* 2012). Putting a Betadine soaked swab in the wound was also shown in a small trail to reduce the risk of groin wound infection (Walker)
- *Haematoma* – May be reduced by the use of a tourniquet and post operative compression bandaging. Flushing the GSV tunnel with local anaesthetic and adrenaline significantly reduces postoperative pain and haematoma formation (Nisar *EJVES* 2006).
    - *Cochrane review* - Surgery for VV: use of tourniquet – reduces blood loss from 107 – 133mls to 0 – 16ml. No difference in complications.
- *Nerve injury following VV surgery.* The saphenous nerve (L3, 4) descends with the superficial femoral artery and pierces the roof of the lower quarter of the adductor canal where it comes to lie deep to sartorius. Here it gives off an infrapatella branch to supply skin medial to the knee and distal patella. The main nerve pierces the fascia lata just above the knee and appears in the subcutaneous tissues at the level of the knee between the sartorius and gracilis. Here the nerve lies deep and posterior to the GSV but below this the two come to lye together, at about 2-3 cm below the tibial tuberosity. Below this the nerve and its branches are wrapped around the GSV. The nerve supplies skin of the calf and medial surface of foot. 50% of patients suffer saphenous nerve injury with ankle to groin strip. This is reduced to 4 – 23% by groin to ankle strip. Stripping to the level of knee also reduces the risk (from 39% to 7%). If nerve injury does occur, symptoms improve over time but a small number go on to develop saphenous neuritis. The sural nerve (S1, 2) arises from the tibial nerve in the popliteal fossa and then descends in the back of the leg to the posterior surface of the lateral malleolus. The sural nerve may pierce the deep fascia above, with or below the short saphenous vein (SSV). It supplies the skin of the lower half of the posterior calf, the lateral part of dorsum of the foot and the lateral side of the little toe. The nerve can sometimes be found within the vein wall. Most would not strip the SSV due to the risk of sural nerve injury but there is little data in the literature to back this up. The common peroneal nerve is at risk of avulsion as it winds around the neck of the fibula. The tibial nerve is at risk behind the medial malleolus.
- *DVT and PE* – DVT occurs in 0.15%, PE occurs in 1:600 and causes death in 1:15 000. Incidence of DVT was 5% although more than 80% of these were in the calf veins (van Rij *BJS* 2004). Overall incidence of VTE was 0.5%, more common in those having bilateral surgery (0.62% vs. 0.3%) and those having redo surgery or short saphenous surgery (Sutton *Ann R Coll Surg Engl* 2012).
- *Recurrence*.

- *Arteriovenous fistula*.



a. b.

Figure . a Left SFA angiogram showing filling of below knee GSV following GSV stripping. B. Delayed angiogram showing contrast draing up leg fro AV fistula. Treated by embolisation.

Surgery for recurrent varicose veins.

Pre operative duplex is a must. My preferred technique is to use a vertical incision, to dissect the common femoral artery and then dissect medially to completely expose the common femoral vein. I find it wise to control the proximal and distal common femoral vein as I have experienced life threatening bleeding during this operation. The GSV may need to be marked pre operatively to ensure this is adequately stripped.

Complications:

- *Recurrence –*

Table 2. A randomised trial of PTFE patch insertion for recurrent GSV varicose veins, (Winterborn *EJVES* 2007):

	Patched	Not patched
Number randomised	20	20
Number of legs available for follow up at 6 weeks	16	16
At 6 weeks, number with groin wound infections requiring antibiotics	2	2
At 6 weeks number of seromas	3	1
At 6 weeks, number of intact SFJs	0	0
Number of one year follow ups	14	16
Recurrent VV at one year (p = 0.26)	7	4
Duplex confirmed neovascularisation at one year	3	3
Two year follow up – number of legs	16	16
Recurrent VV at two years	8	4
Duplex confirmed neovascularisation at two years	5	4

The study was stopped early as the authors switched to foam sclerotherapy for recurrent VV.

- *Groin wound seromas/lymph leaks*
- *Groin wound infection*

Radiofrequency ablation.

Radiofrequency (RF) ablation results from thermal energy being delivered to the vein causing vein wall collagen contraction, vein wall thickening, endothelium destruction and inflammation resulting in fibrosis and permanent vein occlusion.

Technique.

VNUS system using ClosureFAST catheter. The GSV is cannulated at the knee and through a 7 French sheath a catheter is advanced under ultrasound to the SFJ. Cannulation of the vein is aided by proximal tourniquet and topical application of GTN. Keeping the leg warm and use of warm gel packs can help to keep veins dilated. The catheter tip is placed 2cm distal to the SFJ. The catheter may require passage over a guidewire in tortuous veins. Tumescent analgesia – provides analgesia but also compresses the vein. Use spinal needle (21 gauge) connected to bag of IV fluid containing dilute local anaesthetic. The vessel is heated to around 120°C. May reduce the incidence of nerve injury. Complicated by skin burns (3 – 5%), saphenous nerve injury, DVT (0 – 16%). A grade 2 compression stocking is then applied.
Advantages from EVOLVeS randomised study (small numbers, total of 86 limbs) were improved QOL in early follow up, earlier return to normal activities, earlier return to work. Recurrence rate 14% for CLOSURE vs. 21% for open surgery at 2 years and no difference in venous scores at 2 years. Thus no great advantage in the long term.
90% success at 5 years.
Suitable for non tortuous veins of less than 12mm diameter but recently larger veins have been treated successfully (Calcagno *Vascular and Endovascular Surgery* 2009). Usually requires sclerotherapy or avulsions for residual VV.
A randomised trial (Stotter *Phlebology* 2006) showed that RF ablation, when compared to traditional invagination stripping, was associated with less bruising, less pain, quicker return to normal activities (mean of 7 days vs. 14 days) and there were no DVT's or PE's. Only 20 patients in each group. Although at one year there were no physician assessed differences between the groups, patients who had RFA were more satisfied.
Is has been reported that concomitant ambulatory phlebectomy increases the risk of DVT (JVS 2009). This study also showed that RF ablation in patients with a previous history of DVT is safe and does not have a high rate of procedural DVT.
Proebstle has reported a 5 year occlusion rate for veins treated by RF as 91.9% (BJS 2015).

Endovenous laser therapy (EVLT)
First reported by Boné in 1999. Works by delivering laser energy (heat) to create steam bubbles resulting in vein wall damage and subsequent fibrosis. Only 51% of varicose veins are suitable for EVLT, mainly due to the tortuosity of the GSV. Increased bruising (24 – 100%), thrombophlebitis. Laser wavelength of 810 to 1320nm. Increased laser energy results in reduced recannalisation rates but this can still occur in up to 22% of cases. Nerve injury rates of 4 – 36% and skin burns in 2 – 7% have been reported. Use of tumescent analgesia may help with this. In approximately 20%, following the procedure, thrombus is visible at the SFJ but this seems to resolve after a few weeks.
97% success at 5 years.

Technique.
As for RF, the GSV is cannulated with a 19G needle at the level of the knee or the most distal aspect of vein incompetence under ultrasound guidance. The laser fibre is then introduced and the tip placed 2 cm distal to the SFJ guided by ultrasound. The aiming beam of the laser can also be used to confirm position. Perivenous tumescent analgesia is then administered along the vein to be treated. Options for tumescent analgesia – 440ml normal saline plus 50ml 1% lignociane with adrenaline plus 10ml sodium bicarbonate or 420ml normal saline plus 60ml 1% lignocaine plus adrenaline plus 20ml sodium bicarbonate, or 1 L normal saline plus 50ml 1% lignocaine plus 1 ml of 1:10000 adrenaline, or 4 degrees cold normal saline (Chong *Phlebology* 2006). Laser energy is then applied while withdrawing the laser catheter 1 – 2mm at a time. The leg is then bandaged with a compression bandage e.g. Panalast (Lohmann and Rauscher International, Germany) for one week. Some would replace this with a compression stocking for a further 2 weeks.
Michael Gough has reported that patients on warfarin do not need to stop the warfarin prior to laser ablation (EJVES 2009).

Complications.
DVT 0.57 to 16% and PE 0.17%. Occasional reports of AV fistula and mesenteric ischaemia. Skin burns, the incidence of which is reduced by tumescent analgesia. Saphenous nerve injury in 1 – 36.5%. Most cases resolve. Saphenous nerve injury is prevented by GSV cannulation above the knee and tumescent analgesia. Phlebitis in 1.6 – 12%. Treated with NSAID. Bruising in 23 – 100%. May be related to vein perforation. Rarely can result in permanent hyperpigmentation. Compression bandages following treatment decrease the amount of bruising. External iliac arteriovenous fistula (Wheatcroft *vascular* 2014)

Results.
In a comparative study (Vuylsteke *Phlebology* 2006) of 84 patients undergoing conventional surgery and 80 patients undergoing EVLT, EVLT was associated with better QOL scores, reduced sick leave (4 days vs. 19 days for unilateral procedures and 9 days vs. 23 days for bilateral procedures), less post operative analgesia and improved patient satisfaction. Groin haematomas were less common with EVLT (3% vs. 0%), wound infections less (3% vs. 0%), there were a total of 3 burns with EVLT, bruising was less with EVLT, paraesthesia was less with EVLT (28% vs. 14% for unilateral procedures and 19% vs. 11% for bilateral procedures). 20% of EVLT patients had induration along the GSV post operatively. There were 3 partial recannalisations and 4 full recannalisations in the EVLT group. EVLT cost more, mainly due to the laser and laser fibre.
Rates of GSV occlusion range from 87.9% to 100%. Recannalisation occurs in up to 5%.

In a comparative study looking at recurrence and neovascularisation 2 years after surgery and EVLT, there was no difference in the frequency of recurrent VV (6.6% vs. 7%), but neovascularisation was more frequent after surgery. It was recommended that more than 70J/cm laser energy was required to reduce the risk of recannalisation and recurrence with EVLT (Theivacumar NS *VSSGBI abstract book* 2007).

In the Recovery study where Closure fast was compared to a 980nm laser, those who has Closure fast had less post operative pain, less bruising and less paraesthesia.

Not all patients require phlebectomy following RFA or EVLT. In a study by Carradice (*ESVS Nice* 2008), 50% of those who had EVLT required avulsions post operatively.

In a randomised study comparing EVLT and conventional surgery, recurrence at one year was the same but there was more pain after EVLT in the post operative period (Pronk *EJVES* 2010).

In a randomised study of combining saphenofemoral ligation with EVLT, (Disselhoff *EJVES* 2011), the rate of recurrence was the same in both groups.

In a 2013 study in the JVS the 5 year clinical recurrence with conventional surgery was 55% and with EVLT it was 48%.

In a 2014 meta-analysis (Pan *Phlebology* 2014) comparing EVLT with high tie and stripping, there was no difference in recurrence rates but there were fewer bleeding and haematoma complications (1.28% vs. 4.8%) wound infection (0.33% vs. 1.91%) and paraesthesia (6.7% vs. 11.3%) with EVLT compared to surgery.

One novel approach reported at the MEET meeting in Nice 2014 was to use EVLT to cause proximal vein spasm and then inject foam distally.

Studies comparing RFA with EVLT.

Goode, ESVS Nice 2008 (Nottingham) – RFA was less painful than EVLT and produced less bruising.
RECOVERY trial (JVIR 2009) showed less pain with RF

DVT and EHIT with RF and EVLT.
Reported incidence of DVT with RF is 0.5%. EHIT occurs in 1 – 16%. Kabnick classification. Thus there may be a rational for giving LMWH at the time of the procedure, especially in high risk patients. There appears to be a higher risk of DVT and EHIT when phlebectomies are performed at the same time. Incidence of clinically significant PE following endovenous treatment is 0.01%. For EHIT II, treat with aspirin alone. Shutz has reported (JVS Venous and lymphatic disorders 2015) with EVLT an incidence of EHIT of 5.28% with risk factors being higher CEAP C class, increased number of veins treated, larger veins, higher energy used, concomitant phlebectomies (6.6% vs 3.1%), lower laser wavelength. Deep venous reflux is also a potential risk factor. It is recommended that the RF or laser tip be at least 2.5cm from the junction to the deep system to reduce EHIT. Reduced mobility after concomitant phlebectomy may account for the higher EHIT rate.

CLASS trial (NEJM 2014).
A randomised trial from 11 centres in the UK comparing conventional surgery, EVLT and USFS. The used sodium tetradecyl sulphate for the foam (3% for trunks and 1% for varicosities) and used a maximum of 12ml of foam per session. EVLT was performed under LA and followed by foam sclerotherapy for residual varicosities at 6 weeks if required (one centre performed concurrent phlebectomies). The primary outcome measure was patient reported disease specific quality of life measured using the Aberdeen Varicose vein questionair and patient reported generic quality of life using the EuroQoL 5 dimension self report questionair and the Short form 36 survey. Secondary outcomes included duplex follow up a 6 weeks and 6 months to assess for ablation rates. Initial planned sample size was 1015 patients. This was revised to 779 patients when interim analysis showed better than expected results for AVVQ.
6592 patients were assessed, 3369 (51%) met entry criteria and 798 (23.7%) consented to participation of whom 785 were included. Approximatly 57% were female, most had GSV incompetence (84%), interestingly approximately 10% had deep venous reflux. At least 50% in each group had C2 varicose veins.
At 6 weeks 38% of the foam patients had treatment with foam of residual varicosities at 6 weeks compared to 31% in the laser group.
At 6 weeks and 6 months, AVVQ score was significantly higher (indicating worse disease specific QOL) in the foam group compared to the surgery group, but the difference was moderate. There was no difference in the surgery vs laser group. There was no significant difference in the groups using the EQ-5D or SF-36 physical component score at 6 months.
At 6 weeks and 6 months there were fewer residual varicosities as assessed by both patient and a nurse in the surgery group than the foam group, but the difference were small. There were fewer residual varicose veins in the surgery group than the laser group at 6 weeks but not at 6 months.
The frequency of complete ablation of the GSV was 84.4% in surgery group, 83.3% in laser group and 54.6% in the foam group at 6 weeks.
The frequency of procedural complications was lowest with laser (1%) than foam (6.2%) and surgery (7.1%). At 6 weeks and 6 months, complications were greatest in the foam group (mainly lumpiness and skin staining) in the foam group.
Numbness at 6 weeks was (laser vs. foam vs. surgery) 11.4% vs. 5.7% vs. 17.9% and a 6 months 9.2% vs. 4.0% vs. 15.6%. Persistent tenderness and discomfort at 6 weeks, 21.2%, 46%, 31.5%. Skin staining at 6 months 17.4% vs. 36.6% vs. 10.2%.
The conclusion was that Moderate differences in disease specific QOL favoured surgery. All treatments had similar clinical efficacy but there were fewer complications with laser. Ablation rates were lowest with foam.

General comments about RF and laser ablation.
This issue of when to do the phlebectomies is unresolved. In as many as 50% of patients with visible varicosities, following ablation of the major trunks, the varicosities disappear in 50%. Many therefore recommend waiting 2 – 3 months before doing the phlebectomies, which can again be performed under local anaesthetic.

Powered phlebectomy (Trivex).
General, epidural or spinal anaesthetic is usually required. Local tumescent anaesthetic and conscious sedation can be used in some patients. Incisions of 2 – 3mm in length and vertically orientated in the thigh and calf and transversely at the level of the knee following Langer lines allow access of an illuminator and resector for resection of clumps of varices. Up to 23% incidence of saphenous nerve injury. In a randomised study comparing stab avulsions with powered phlebectomy, powered phlebectomy reduces the number of skin incisions but was associated with significant bruising, more sever and persistent pain and there was no improved quality of life.

Subfacial endoscopic perforator surgery.
Saphenous and sural nerve injury 10 and 2%. Contraindications – non ambulatory patient, extensive deep venous occlusion, chronic arterial occlusive disease, infected ulcer, morbid obesity. Relative contraindications – diabetes, RA, scleroderma. Perforators are marked by duplex. Torniquet occlusion. Advantages – reduced wound complications.

External valvular stents.
Venocuff. After 5 years 90% have competent SFJ. However, only 34% of patients are suitable as the GSV must have a diameter less than 10mm and no gross tortuosity or varicosity of the GSV.

Coil embolisation.
DVT rate of 0.5%.

Glues.
There are now a number of these on the market including Saphenon and Venaseal. The idea is that a small drop of glue (0.09ml) is injected into the vein and external compression is then applied to stick the walls together. The advantage is that tumescence and a compression stocking are not required.

The whole concept of compression after any varicose vein intervention has been questioned by some when on CT there is no effect on GSV diameter in the thigh with currently available compression stockings.

Follow up after venous interventions.
In a debate at Charring Cross 2014, 64% voted against the motion that duplex scanning is mandatory after treatment for saphenous reflux.

Superficial vein thrombosis - Superficial thrombophlebitis (STP).
Incidence 3 – 11% of the general population. 78% are female. Risk factors are previous thromboembolic episodes, long haul flight, pregnancy, oral contraceptives, HRT, immobilisation, age over 60, obesity (Karathanos *EJVES* 2012), recent surgery, trauma, sclerotherapy, IV drug abuse and IV cannulae. Mean age at presentation is 60 years. It is most common in the saphenous veins and their tributaries but can also occur in the upper limb. The GSV is involved in 60 – 80% of cases. Bilateral in 5 – 10%. Patients with STP have a higher prevalence of hypercoagulable states. Patients with spontaneous STP without VV or extending to the main trunk of the GSV should be screened for hypercoagulability. Weak relationship between STP and malignancy. Trousseau's syndrome – migratory thrombophlebitis with malignancy.

11% have extension into the deep veins, increased risk if the above knee GSV is involved. Thus patients with STP should have their deep veins scanned. 6 – 44% of cases are associated with DVT and PE occurs in up to 33% of those with thigh STP.
Inflammatory response in vein wall. Painful indurated erythematous vein.
Differential – gout, cellulites, lymphangitis, fat necrosis, vasculitis.

Mondor's disease – uncommon. First described by Henri Mondor 1939. Breast pain and tenderness. Mainly in the upper outer quadrant of breast or in submammary fold. Treat symptomatically.

Treatment.
No role for antibiotics, NSAID creams or heparinoid creams. If there is infection, debridement and antibiotics (usually staphylococcal).
Aspirin and NSAID decrease local pain.
Compression stockings if tolerated.
LMWH for 4 weeks, extended if associated DVT.
Repeat duplex scan after 10 days to assess extension of thrombus. If thrombus migrates towards SFJ, urgent SFJ ligation is appropriate
Surgical options – local thrombectomy, vein ligation, excision and stripping, sclerotherapy.

CLAISTO, a randomised study of fondoxaparanu (2.5mg SC od) in STP reported that embolic complications were reduced by 85% with no increase in risk of bleeding complications. (*NEJM*). NNT 20.

Cochrane review 2007 – Treatment for superficial thrombophlebitis – LMWH and NSAID appear the current best therapeutic option. LMWH for one month.

Varicoceles.
Incidence in general male population is 15%. This rises to 30% of infertile men. It may be due to absence or insufficient testicular vein valves. It is more common on the left, possibly due to a longer vein and its communication with the left renal vein. 46% of young men with VV will have a varicocele (Bolcal *Phlebology* 2006). 85% of patients with a varicocele will have lower limb venous disease. Varicoceles are related to obesity, standing occupation, constipation and smoking. Endovascular coil embolisation has become the treatment of choice.

Pelvic varicose veins and pelvic congestion syndrome.
This is a cause of pelvic pain (dyspareunia, chronic pelvic pain, bladder irritability) and also recurrent leg varicose veins. It is usually due to ovarian vein incompetence and can give rise to vulval and recurrent varicose veins. The choice of pre intervention imaging is unclear, ranging from transvaginal ultrasound, CTV and MRV. Endovascular embolisation seems to work well. Whether pelvic varicosities should be treated before leg varicose veins is controversial. At the Charring Cross meeting in 2014, 36% felt that pelvic vein reflux must be treated before treating leg veins.

Vulval varices.
Coil embolisation of the ovarian veins results in resolution of vulval varices in 87% (Castenmiller *ESVS Amsterdam* 2010). Foam embolization of the anterior branch of the internal iliac veins is also effective. Access via the right internal jugular vein can be used. Complications include embolisation of the coils (Ratnam *Cardiovasc intervent radiology* 2008).

Nutcracker syndrome.
Caused by compression of left renal vein between the superior mesenteric artery and aorta resulting in left renal and gonadal vein hypertension. It can cause left flank pain, haematuria, varicoceles.
Can be treated by renal vein transposition.

Haemorroids.
Calcium dobesilate (Doxium) has been shown in a randomised study to provide symptomatic relief from acute symptoms of haemaorrhoidal disease.