

A STUDY OF VENOUS LEG ULCERS

Dissertation Submitted to

**THE TAMIL NADU
DR.M.G.R. MEDICAL UNIVERSITY**

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**M.S. (General Surgery)
BRANCH - I**



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CERTIFICATE

This is to certify that this dissertation entitled "**A STUDY OF VENOUS LEG ULCERS**" is the bonafide original work of **Dr.V.ARUN**, a post graduate student in the General Surgery Department, in partial fulfillment of the requirement for M.S., (Branch I) General Surgery examination of the Tamil Nadu Dr.MGR Medical University to be held in March 2008.

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DECLARATION

I, **Dr.V.ARUN**, solemnly declare that this dissertation "**A STUDY OF VENOUS LEG ULCERS** " is a bonafide record of work done by me in the Department of General Surgery, Government Stanley Medical College and Hospital, Chennai under the guidance of Prof **Dr.M.L.Shyamala, M.S.**, Head of the Department, Department of General Surgery, Government Stanley Medical College and Hospital, Chennai – 600 001.

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INTRODUCTION

“Venous ulceration” is defined as ulceration of the malleolar skin in the presence of perforating vein incompetence with or without deep vein incompetence.

Ulceration of the lower leg is a common symptom that will affect 2 % of people in their life time. Its prevalence increases with age from 0.5 % among patients over 40 to 2 % among those who are 80. As the proportion of elderly people in the population increases, we can expect a rise in the present estimated number of leg ulcers unless a more educated approach to its management is taken.

Though important advances have been made in the management of leg ulcers, India lags far behind the standards set by the European countries. Treatment is fragmented, poorly taught and inadequately researched. The average time taken to heal an ulcer is about 6 months and some persist for years.

Good management depends on accurate diagnosis, simple and appropriate care of the wound and treatment of underlying cause.

AIM OF THE STUDY

The objective of this study is to analyze the incidence of Venous leg ulcers in different age groups and sex, to know the relationship between socio economic status and venous ulcers, to compare duration of ulcer and symptoms, to analyze underlying pathology, the side of ulcer, to evaluate the causes, the recurrences and complications and finally to find out the best suited procedure.

PATIENTS AND METHODS

Patients with clinical diagnosis of venous ulcer between 2005 June to 2007 April (only inpatients) were included in this study. Among 116 patients who had venous ulcerations 111 were due to varicose veins and 5 patients had DVT.

A detailed history including symptoms, duration of ulcer, mode of development, occupation, previous surgery, claudication were taken. The patients were examined in both standing and recumbent postures. The location, size, floor of the ulcer and secondary changes in the leg including pigmentation, periostitis of underlying bone, mobility of ankle joint were noted. Regional lymph nodes were examined. Legs were examined individually for varicosities. Saphenofemoral / Saphenopopliteal and perforator incompetences were made out by Trendelenburg, multiple tourniquet test and Schwartz test. Deep vein assessed by Perthe's test. Perforator incompetences were localized by palpating the deep fascia for defects. Arterial pulses of both feet examined to rule out arterial components.

Abdominal and pelvic examination were done to rule out tumors, dilated suprapubic veins and ascitis. Doppler and Duplex imaging was used to rule out DVT and localize perforator incompetence in all cases.

Routine investigations of blood and urine were done. Swab taken from the wound and antibiotics prescribed according to culture and sensitivity. Patients were treated with initial Hydrogen Peroxide and Povidone Iodide until wound became healthy and then normal saline dressing was done daily. Elastocrepe bandage was applied from the level of head of metatarsal up to the knee. The limb was kept elevated by raising the foot end of the bed. 110 patients underwent surgery. The choice of surgery was determined by the extent of disease and patients general condition. The procedures done included Trendelenburg operation, complete stripping of long saphenous vein, multiple avulsion, sub-fascial ligation and extra-fascial ligation of perforators.

After performing the surgery a layer of pad and cotton bandage was applied, over which elastocrepe bandage was also applied. The dressings were changed on the 3rd POD. Patient was allowed to walk after 12 hours. The sutures were removed after 10 days. They were advised to avoid prolonged standing and to wear elastocrepe bandage for 6 months.

HISTORY OF VENOUS LEG ULCERS

Hippocrates was the first person who apparently recognized some relationship between leg ulcers and venous disorders. A Roman physician Aurelius Cornelius celsus (25 BC – 50 AD) advised the use of plasters and linen bandages in the treatment of ulcers and also treated varicose veins by avulsion and cauterization. During the dark ages those with unsightly and ulcerated legs were denied simple remedies because it was believed that bandaging varicose legs would reintroduce black bile into the circulation and madness.

Richard Wiseman described a laced stocking in 1676.

During the 18th century it began to be realized that leg ulcers were not necessarily accompanied by visible varicose veins.

John Gay in 1868 described about the perforating veins of the calf and ankle, recorded the fact that ulcers could occur in the absence of varicose veins if there had been post thrombotic damage to deep veins and introduced the term ‘Venous Ulcers’. He described clot formation and post thrombotic recanalization. He correctly described the sub-fascial course of proximal short saphenous vein in upper 1/3rd of calf.

Homan divided ulcers into those associated with varicose veins of legs, easily cured by removal of these veins and post phlebotic ulcers, rapid in development, always refractory to palliative treatment not cured

by varicose veins alone and needed excision. Home n in 1916 emphasized that sapheno femoral ligation was a vital step in the prevention of the recurrence of varicosities.

Linton of Boston described about the sub fascial ligation of incompetent perforating veins.

Harold Dodd showed that improved skin healing could be obtained by placing the incision for sub fascial ligation postero-medially rather than through medial liposclerotic skin.

Between 1961 and 1971 many follow-up studies were conducted on perforating vein ligation in the treatment of venous ulceration. The success rate was around 90%.In subsequent 10 years disappointing results in patients with deep venous incompetence as well as perforator incompetence have led to more careful appraisal and many surgeons now advice elastic hosiery alone for venous ulcer due to DVT. Sclerotherapy was accepted after Lincer of Tubigen clinic in 1911 discovered that bichloride of mercury was less toxic when injected parenterally.

THE ANATOMY OF THE VEINS OF THE LOWER LIMBS

VENOUS SYSTEM OF LOWER LIMB

The veins of lower limb may be divided into 3 groups.

1. Superficial
2. Deep
3. Perforating

SUPERFICIAL VENOUS SYSTEM

It consists of Great saphenous vein and small saphenous vein and their tributaries. These are situated beneath the skin in the superficial fascia. They are thick walled and valves are more numerous more distally. A large portion of their blood is drained into deep veins through the perforating veins.

LONG/ GREAT SAPHENOUS VEIN

It begins on the dorsum of the foot from the medial end of the dorsal venous arch and runs upwards in front of the medial malleolus, along the medial side of the leg and behind the knee. In the thigh it opens into the femoral vein by piercing the cribriform fascia(4 cm below and lateral to the pubic tubercle). The junction of long saphenous vein and femoral vein is clearly visible as white line. Saphenous nerve is closely applied to the long saphenous vein in the leg and ankle where it is at risk of damage by vein stripping.

This vein consists 10-15 valves which prevent backflow of venous blood encouraged by gravity. One valve is always present at the saphenofemoral junction. The vein is also connected to the deep veins by the perforating veins. Before piercing the cribriform fascia it receives 3 named tributaries:

- 1) Superficial epigastric
- 2) Superficial external pudental
- 3) Superficial circumflex iliac

SMALL SAPHENOUS VEIN

It is formed by the union of the lateral end of the dorsal venous arch with lateral marginal vein. It enters the back of the leg by passing behind the lateral malleolus. In the leg it ascends first lateral to tendocalcaneus and then along the middle line of the calf to the lower part of the popliteal fossa where it pierces the popliteal fossa and opens into the popliteal vein. It drains the lateral border of the foot, the heel and the back of the leg. It is connected with the great saphenous vein and with the deep veins and is accompanied by the Sural nerve.

DEEP VEINS

These are anterior and posterior tibial, peroneal, popliteal, femoral and their tributaries. They accompany the named arteries and are supported by the powerful surrounding muscles. The valves are more numerous than in superficial veins. The muscular veins are also valved except for the soleal veins. They are more efficient channels than the superficial veins because of the driving force of the muscular contraction.

PERFORATING VEINS

These are communicating veins between superficial and deep veins.

A. INDIRECT

These are not important and are 50 to 100 in number. These consist of small superficial vessels which penetrate the deep fascia to connect with a vessel in a muscle which in turn is connected to one of the deep veins. Ankle is having little muscle, therefore it has few indirect perforators. In ankle return of blood from superficial tissues depends only on direct perforators. This is the reason for ankle ulceration.

B. DIRECT

These are more important and consist of one long saphenous, two small saphenous and three perforating veins.

Small and long saphenous veins are also considered as large direct perforators because they are valved to prevent flow from superficial to deep venous system.

PERFORATING VEINS

They are fairly constant in position and related intimately to the posterior border of tibia and fibula and are valved. As a rule they are accompanied by an artery.

1. Thigh Perforator (Adductor canal perforator): It communicates between long saphenous vein and femoral vein.
2. Leg Perforators:
 - A. **Below Knee:** Communicating vein between long saphenous or posterior arch vein and posterior tibial vein.
 - B. **Medial Ankle Perforators:** They connect with a series of venous arcades which characterize the lower part of the large constant posterior arch vein.
 - i) *Upper Perforator:* Found at the junction of middle and lower third of leg at the posteromedial border of tibia. It constantly communicates with long saphenous vein. It is three to four centimeters long.
 - ii) *Middle Perforator:* It lies 10 cm above the tip of medial malleolus and just behind the tibial margin. It is 3-4 cms long.
 - iii) *Lowest Perforator:* It is situated behind and below the medial malleolus and is 1 cm long.
 - C. **Lateral Perforator:** A constant lateral perforating vein is found at the junction of lower and middle third on the outer side of leg. It connects small saphenous vein with peroneal vein.

PHYSIOLOGY OF VENOUS FLOW IN LOWER LIMB

Veins contain two thirds of total circulating blood volume. Venous tone is mediated by sympathetic system. The most important function of superficial veins of lower limb is thermo regulation.

Deep veins mainly act as passive blood conduits. Blood is returned to heart by means of four pumps namely:

1. Foot Pump.
2. Abdominal Pump
3. Respiratory Pump.
4. Calf Pump.

Foot Pump: Blood is expelled from plantar veins by their intermittent stretching during foot movements, rather than by pressure of the sole of the foot on the ground.

Respiratory Pump: Shallow inspiration increases the total venous return slightly. But deep inspiration almost certainly reduces venous return.

Calf Pump (Peripheral heart): *Disordered function of calf pump is the single most important factor in the etiology of venous ulceration.*

Calf muscle pump has four important components

1. Dilated valveless sinusoids within the soleus and gastrocnemius.
2. Direct perforating veins.
3. Numerous valves in the communicating and deep veins.
4. A layer of tough deep fascia which surrounds the calf muscles.

Although it has been suggested that all communicating veins (the long and short saphenous veins as well as the lower calf perforating veins) are subjected to high venous pressure in the presence of deep venous incompetence and reflux, when deep vein valves are incompetent, the pressures inserted on the lower calf and ankle perforating veins are significantly higher than those of either the long or short saphenous veins.

Resting foot venous pressure reflects the height between the right atrium and foot and in erect position is normal 80 – 90mm Hg. Following exercise, by raising the heel repeatedly off the ground, the pressure falls to about 25 mm Hg. Standing motionless, the foot venous pressure takes 25- 30 seconds to return to previous high resting levels, but venous reflux results in a rapid return to resting pressure following exercise.

Calf muscle pump function may become disordered as a result of muscle weakness, saphenous incompetence, deep vein incompetence or

obstruction or perforating vein incompetence with or without deep vein incompetence. All these abnormalities will adversely affect pressure profiles in the dorsal foot vein. Most venous ulcers occur in the malleolar regions which are drained by the corona phlebectatica, the network of small veins communicating with the calf and the ankle perforating veins.

At present measurement of calf muscle pump function can be done by dorsal foot venous pressure measurement or by plethysmography. Measurement of flow and pressure changes in the ankle flare veins is extremely difficult but, *it is the disorders of this venous network that are of prime importance in the etiology of venous ulcerations. In the prevention of recurrent alteration, all points of venous reflux communicating with the malleolar venous network must be accurately identified and effectively controlled.*

THE PATHOLOGY OF VENOUS LEG ULCERS

These ulcers occur most commonly over the medial malleolus and less frequently over the lateral surface of the ankle. A true varicose ulcer may lie anterior or posterior to these sites, usually directly over a large varix. Venous ulcers vary in size from only a few centimeters to giant ulcers which may lie circumferential and involve the whole of the **gaiter area** of the lower leg. They are often infected when first seen but infection responds rapidly to daily dressing – non irritant dressings, firm compression bandaging and appropriate antibiotics.

A clean venous ulcer has a base composed of healthy pink granulations covered perhaps by a little debris and edges consist of sloping pink epithelium. The granulation tissue represents the surface of capillary loops. A venous ulcer is usually surrounded by pigmented indurated skin (**lipodermatosclerosis**), the result of peri-capillary fibrin deposition.

BACTERIOLOGY OF LEG ULCERS

Most ulcers are infected when first seen. **Staph. aureus** is the most commonly cultured organism. Anerobic organisms are also found.

VENOUS DISORDERS RESPONSIBLE FOR ULCERATION

1) VARICOSE VEINS

They are defined as distended, tortuous superficial veins with incompetent valves. It has been supposed that valvular incompetence starts at the saphenofemoral junction and maybe the result of pressure from a gravid uterus or even from pressure from a loaded sigmoid colon.

Histologically there is disorganization of vein wall architecture leading to decrease in elasticity.

Varicose veins are more common in women than in men in western countries and usually appear for the first time during the first pregnancy. They often disappear after the baby is born, only to reappear and persist

during and after the second pregnancy. Already weakened walls become further weakened and dilated in response to increased estrogen and progesterone.

Varicose veins may present as small local varices without the evidence of either long or short saphenous incompetence.

II) THE POST- THROMBOTIC SYNDROME AND PERFORATING VEIN INCOMPETENCE.

Following thrombosis fibroblasts, mast cells, polymorphs and histiocytes invade the vein wall and the occluded lumen is usually restored by a combination of thrombus reaction and recanalisation. Instead of the vein wall becoming thin and weak, as in primary varicose veins, the walls of previously thrombosed veins are thicker and less distensible than those of normal veins, due to collagen deposition by fibroblasts in the process of recanalisation. The lumen is usually patent but irregular. In the process of recanalisation the delicate valves are either destroyed or become permanently adherent to adjacent vein wall. **Venous incompetence is therefore the direct result of valve incompetence rather than vein wall dilatation resulting in incompetence of normal valves as in varicose veins.**

Direct perforating vein incompetence may result from one of the following causes or a combination of these:

1. Long standing primary varicose veins
2. Following local thrombophlebitis
3. Following DVT & recanalisation
4. Ulcers resulting from combined effects of perforating and deep vein incompetence are the most difficult to treat and most likely to recur

THE MICROCIRCULATION IN VENOUS ULCERATION.

Although there is little doubt about venous ulceration that it results from high ambulatory venous pressures in the subcutaneous veins of the lower leg and ankle , there is still controversy about the precise effect of such pressure on the skin microcirculation. The three current theories are : fibrin cuff theory , white cell entrapment and microthrombus formation.

FIBRIN CUFF THEORY

The rise in venous pressure causes dilatation of horizontal subcapillary venous plexus with grossly coiled elongated capillary loops and a reduction in the number of capillaries supplying epidermis.

Burnard et al showed that fibrinogen 'leaks' from these dilated capillaries ,forming a *peri-capillary fibrin cuff which was thought to be responsible for reduced diffusion of blood oxygen to tissues* Both

systemic and tissue *fibrinolytic activity is significantly reduced in patients with venous hypertension.*

Fibrin cuff theory has given rise to an entity called “LIPODERMATOSCLEROSIS” or “LIPOSCLEROSIS” (indurated pigmented skin).

WHITE CELL ENTRAPMENT

White cells are trapped in circulation in small area of skin immediately proximal to the malleoli in dependent legs. This in turn leads to localized areas of ischemia due to occlusion of capillaries by WBCs which leads to ulceration. The white cell entrapment might contribute to liposclerosis.

III) MICROTHROMOBOSIS

Ehrly & Colleagues suggested that long term stimulation of fibrinolysis by low dose urokinase can increase TcPO₂ values and lead to a reduction in ulcer area.

In the healing stage of management, this hypertension is counteracted by appropriated elastic compression bandages or stockings. In the definitive treatment and in the prevention of recurrent ulceration attention must be paid to identifying all points of venous incompetence

responsible for such hypertension and correcting these by appropriate measures in each case

DIAGNOSIS

HISTORY AND EXAMINATION

Energetic attempts to heal ulcers and to prevent their recurrence are often unsuccessful due to the failure to appreciate that diagnosis and treatment of the underlying cause of the ulcer is very more important than treatment of the ulcer itself.

HISTORY

In taking the patient's history it is important to note the **duration** of the ulcer whether there was any **precipitating** cause and whether there is any past history of **deep vein thrombosis** or **varicose veins**.

It is also important to enquire whether the patient has experienced symptoms suggestive of ischemia, intermittent **claudication** or rest pain. A venous ulcer may be locally painful but if the patient complains of **pain in the toes or forefoot** suspect ischemia.

A history of DVT does not necessarily mean that ulcer is venous in origin. Ulcer of mixed venous and arterial origin are not uncommon in elderly.

Even symptoms of claudication may cause diagnostic confusion as post thrombotic venous obstruction may result in severe calf pain on walking, so called “**Venous Claudication**”

Diabetic ulcers are usually situated on the sole of the foot, but may occur on the leg and all new patients attending an ulcer clinic must have routine urine checkup.

Other forms of **vasculitis** may some times present with leg ulcers. **Hypertension** may occasionally present with leg ulceration, though there is some doubt whether the ulcer described by Martorell is infact a distinct entity.

EXAMINATION

A full **general examination** must be carried out and this must include **blood pressure** measurement. Examine the conjunctiva for evidence of **anaemia**. **Leg pulses** must be palpated especially in edlerly to rule out ischemia.

Ankle pulses are ofter difficult to feel in the ulcerated and oedematous leg and Doppler ultrasound examination must then be used.

Careful **abdominal examination** is done to exclude abdominal aortic aneurysm and other masses.

Primary familial varicose veins are the result of an inherited collagen and smooth muscles defect in the vein wall and any abdominal mass in such is likely to be coincidental.

Leg swelling can certainly occurs as a result of iliac vein compression by tumour spread, particularly if this involves **pervenous lymphnodes**. **Carcinoma of prostate** must therefore be excluded in men and **uterine or ovarian malignancy** in women.

During abdominal examination, the **groins** must be examined for the presence of **dilated collateral vein**, the result of iliac venous occlusion. These most often appear as dilated subcutaneous vein rather than true varices. Post thrombotic iliac vein occlusion and its attendant physical signs, most ofter affects the left leg.

INSPECTION

The colour and temperature of the toes gives a good indication of blood flow. Both patient with venous insufficiency and those with ischaemia may have cyanosed feet, but cyanosis secondary to venous congestion will show rapid refilling after firm finger pressure to empty superficial vessels, while an ischaemic foot will have a slow refilling time as long as 4 or 5 seconds.

Then **ulcer** is examined carefully. It is often broadly stated that venous ulcers most commonly occur in the skin over the malleoli, most usually the medial and often occurs in an area of pigmentation or lipodermatorsclerosis. Ischemic ulcers are usually on the anterior surface

of the leg and dorsum of the foot and diabetic ulcers are most commonly on the sole of the foot. Ulcers are however notorious mimics and it is quite common for an ischemic ulcer to occur over the medial malleolus to all appearances looking exactly like a venous ulcer.

Following general and pedal pulse examination on the examination couch, the patient is asked to stand so that thorough examination of the leg veins can be performed. The lower leg and foot are first inspected for the evidence of cyanosis which may not have been evident in the supine position, particularly if venous in origin.

The **surroundings of the ulcer** are carefully inspected for evidence of **lipodermatosclerosis or pigmentation**, typical of venous ulcer and the malleolar skin is equally carefully inspected for the presence or absence of an **ankle venous flare**. The fine network of venules which has been described as the **“Corona Phlebectatica”** is the **single most constant indicator of calf perforating vein incompetence**.

An ankle flare is a far more reliable indicator of perforating vein incompetence than “fascial defect” palpated on the medial surface of the calf. Indentation in the subcutaneous superficial fat can often be felt, but these are more likely to represent superficial varices than defects in the deep fascia and this is a unreliable method of examination.

Cough impulse in the groin is not be recommended as a test for saphenofemoral incompetence.

PALPATION

The aim is to locate the incompetent valve communicating the superficial & deep veins.

Trendelenburg Test

A positive Trendelenburg Test indicates either in competency of the saphenofemoral valve or in competency of communicating vein allowing blood to flow from the deep to superficial veins. Positive test are indications for operation.

TOURNIQUET TEST

Tourniquet is tied around the thigh or the leg at different levels after the superficial veins have been made empty by raising the leg in recumbent position.

The patient is now asked to stand up. If the veins above the tourniquet fill up and those below remain collapsed, it indicates the presence of incompetent communicating vein above the tourniquet. Similarly if the veins below the tourniquet fill rapidly where as veins above tourniquet remain empty, the incompetent communicating vein must be below the tourniquet. Thus by moving the tourniquet down the leg in steps or by applying multiple tourniquet one can determine the position of the incompetent perforators.

In case of short saphenous incompetence application of venous tourniquet to the upper thigh increases the strength of reflux as shown by faster filling time.

THE PERCUSSION TEST (SCHWARTZ TEST)

1) The watching fingers are placed over the foramen ovale or the popliteal fossa and the dilated saphenous vein or a varicose tributary in the lower leg is tapped briskly with fingers of the other hand. The dilated vein transmits the pulse wave thus produced and an impulse can easily be felt by the watching fingers. Strictly speaking the **“Distal Tapping & Proximal watching” test only indicates a dilated vein**

As per David Negus the tapping test with or without help from Doppler Ultrasound remains the mainstay of examination for saphenous incompetence.

PERTHE’S TEST

This is primarily intended to know whether the deep veins are normal or not. A tourniquet is tied around the part of the thigh tight enough to prevent any reflux down the vein. The patient is asked to walk quickly with the tourniquet in place. If the communicating and deep veins are normal, the varicose vein will shrink whereas if they are blocked the varicose vein will be more distended.

AUSCULTATION: Continuous machinery murmur will be heard in A.V. fistulas.

DIAGNOSIS

METHODS OF INVESTIGATIONS

Priorities in the investigation of leg ulcers:

As we are concerned only in obtaining essential information regarding underlying cases of leg ulceration there is no need for complicated and expensive equipment.

Non Invasive Investigations

- 1. Doppler Ultrasound (Hand held instrument is adequate)**
- 2. Photoplethysmography**
- 3. Strain gauge plethysmography**
- 4. Foot volumetry**
- 5. Duplex scanning.**

Invasive Methods

- 1. Angiology – Venography and Arteriography (Only if needed)**
- 2. Foot venous pressure measurement**

Photoplethysmography when combined with Doppler ultrasound and venography provides sufficient information for clinical management.

3. **Formal venous pressure measurement** – essential in diagnosis of the iliac vein occlusion requiring surgical correction.

4. **Radionuclide phlebography** – This test is used to look for the patency of veins

DOPPLER ULTRASOUND

Two types of Dopplers are available. Continuous wave and pulsed devices. In continuous device one crystal generates the sound waves continuously and an adjacent crystal samples the back scattered sounds. In pulsed Doppler the same crystal generates and receives sound waves. An electric gating system examines sounds return at different times, thereby measuring flow velocity at a given tissue depths.

TECHNIQUE OF DOPPLER ULTRASOUND

FOR VENOUS EXAMINATIONS

Doppler transducer is placed over the femoral, popliteal and posterior tibial veins and held at an angle of 60° approximately. A strip chart recorder notes the change during respiration (spontaneous) or during augmentation manoeuvres (Valsalva or calf and thigh compression)

Reverse flow on standing, during inspiration or with tourniquet localization can be determined for evaluation of valvular incompetence using the directional Doppler probe with the patient upright and holding on to a rail to ensure leg relaxation.

Femoral venous incompetence is shown by reverse flow on standing or abdominal compression. With a below knee tourniquet to occlude the greater and lesser saphenous veins, popliteal valve incompetence is suggested by reverse flow on release of compression as well as by to and fro fluctuation on calf muscle compression. To and fro fluctuation are also heard over incompetent perforating veins.

PHOTOPLETHYSMOGRAPHY

It is very useful in distinguishing oedema of venous origin from non venous causes. It is a method of detecting chronic venous insufficiency by assessing the venous filling time of an extremity.

STAIN GAUGE PLETHYSMOGRAPHY

It is very useful for diagnosing venous obstruction

It is useful in detecting DVT & to evaluate the functional status of patients with chronic venous insufficiency.

IMPEDENCE PLETHYSMOGRAPHY

It is based on ohm's law.

Because blood takes the path of least resistance in the body, a constant current results in a decrease in voltage between two reference points as the blood volume of a limb increases and vice versa. It is most useful in DVT evaluation.

FOOT VOLUMETRY

It is useful in research (e.g effectiveness of stockings) but more convenient for nonulcerated leg.

DUPLEX SCANNING

It permits examination of greater and lesser saphenous veins as well as the perforating veins in the superficial system and femoral, popliteal and calf veins in the deep system.

Saphenofemoral incompetence, saphenous vein diameter, wall thickness, the diameter of the saphenous tributaries and valvular competence with in the remaining saphenous system are assessed.

Duplex scanning of the deep venous system for valvular competency assesses 3 phenomena

- 1) Direction of blood flow
- 2) Value thickness
- 3) Value Co-aptation

LOWER EXTREMITY VENOGRAPHY

a) ASCENDING VENOGRAPHY :

It is performed with or without ankle tourniquet. It is commonly done with the patient supine and one tourniquet at the ankle and another above the knee. After Percutaneous puncture of small vein on dorsum of the foot with 20 or 22 gauge needle manual injection of 50 – 100 ml of MEGLUMINE DIATRIZOATE 60% is given. The flow of the contrast is observed fluoroscopically and pertinent radiographs are taken of the leg in front and lateral projections and of the thigh, groin and pelvis in frontal projections.

When percutaneous venepuncture is not possible because of severe pedal oedema a cut down on the dorsum of the foot almost always provides an appropriate vein. A rarely used alternative is intraosseous venography if superficial veins are not available for study. Most ascending venography are performed to confirm a clinical suspicion of thrombophlebitis. **Venography is 95% accurate in showing DVT.**

It is also useful in evaluation of venous malformation, varicose veins, trauma and postphlebotic limb.

b) DESCENDING VENOGRAPHY :

Here a catheter is percutaneously inserted into femoral vein. It can distinguish competent from incompetent venous valves. In patients with severe valvular incompetence one sees retrograde passage of contrast through the valves with little or no central flow.

RADIONUCLIDE PHLEBOGRAPHY

It involves injection of radionuclide sodium pertechnetate (Tc 99) with subsequent dynamic imaging of the venous flow pattern of radionuclide using a gamma camera. This test is to look for the potency of vein. The presence of **venous thrombosis** can be determined by

- 1) **Dilution or absence of a portion of normal course of a major venous segment.**
- 2) **The presence of collateral venous pathway.**
- 3) **Delay in appearance of the radio nuclide hot spots.**

DIFFERENTIAL DIAGNOSIS OF LEG ULCERS**I) VENOUS ULCER :****A) RELATED TO LOCAL VARICOSITY**

The ulcer related entirely to superficial varicosity arising from saphenous incompetence and reflux is rare. Careful examination of

patients with varicose veins and ankle ulceration will usually demonstrate perforating vein incompetence.

Varicose ulcers' one constant feature is that they are invariably related to a large dilated varicose vein.

The patient will usually have a long history of primary varicose veins, often with a similarly affected patient.

Examination will show large calf and ankle varices, most usually arising from a grossly distended long saphenous vein. This can be confirmed by the percussion and tourniquet tests. There may be patches of varicose eczema. Peripheral ischaemia be excluded by careful palpation of ankle pulses and Doppler ultrasound.

B) ULCERATION RELATED TO PERFORATING / DEEP VENOUS INCOMPETENCE

Venous ulcers are most commonly found over the medial malleolus, less commonly over the lateral malleolus, occasionally even become circumferential. They vary in depth from 1 or 2 mm to extending down to the deep fascia. They are always much deeper than varicose or vasculitis ulcers.

Venous ulcers are almost invariably accompanied by an ankle venous flare of fine superficial venules and usually by pigmentation and

lipodermatosclerosis. These physical signs are seen accompanying smaller ulcers, very large ulcers may include all the skin normally occupied by the venous flare or liposclerosis. Most venous ulcers are infected when the patient first presents to the hospital and the ulcer base may be obscured by purulent debris and slough. History of Local trauma is common.

If patient has chronic iliac vein occlusion then there may be symptoms of venous claudication – bursting calf pain after walking some distance.

Examination of the limb with venous ulceration has two main purposes. Firstly to identify all the points of venous reflux and possible venous obstruction and secondly to exclude arterial insufficiency as a contributory factor.

Doppler ultrasound examination may be helpful in identifying long saphenous incompetence but its chief value is in identifying sites of perforating vein incompetence and also popliteal and short saphenous incompetence.

Strain gauge plethysmography is useful in investigating possible proximal venous obstruction. Duplex scanning, if available can be used to investigate popliteal and saphenous reflux, perforating vein incompetence and proximal venous obstruction.

Ascending venogram may be needed in order to provide more precise information about the site of incompetent perforating veins than is possible with non invasive methods.

ISCHAEMIC ULCERATION

Ischaemic ulcers may be situated on the toes or forefoot in an area of cyanosed and obviously pre-gangrenous skin. Another common site is heel, particularly in bed ridden patient. The ulcers are accompanied by severe rest pain. History of ischaemic rest pain is common in severe cases and lesser degrees of ischaemia are usually associated with intermittent claudication. The patient is often a heavy cigarette smoker. These ulcers may present as pressure sores of heel.

Ischaemic ulcers occur in an area of obviously ischaemic skin. All pulses must be palpated. In case of pedal oedema Doppler ultrasound should be used. Ischaemic ulcers can result from small vessel insufficiency particularly in Diabetes, TAO and the vasculitides. The pressure gradient between the ankle and toes normally should not exceed 30 mm Hg. Arteriography is often mandatory. Ulcers may respond to Lumbar sympathectomy or prostacycline infusion.

ULCERATION OF MIXED ARTERIAL AND VENOUS ORIGIN

It is very common in elderly. It is often not recognized that there is an ischaemic element until months or even years of conventional

dressings and compression bandaging fail to achieve satisfactory healing. Careful examination of foot pulses is the most important. Doppler ultrasound examination of ankle pulses and observation of toe pulse waveform by PPG may be helpful. It is often necessary to perform both arteriography and ascending venography to perform full evaluation.

A.V.FISTULA

Congenital AV fistula usually affects the leg and the dilated surface veins may be mistaken for simple varices until their pulsation is noted. The patient or parents may have been aware of increased limb growth. The ulcer is usually indistinguishable in its features from a typical venous ulcer. Careful examination of the surrounding skin will show pulsation of the dilated veins and their bruit can be heard with a stethoscope. Arteriography is necessary to demonstrate the site of the fistula and to evaluate the possibility of therapeutic embolization – AV fistula formation may also follow inadequate ligation of tributaries of the long saphenous vein, when the latter is used for an insitu femoropopliteal graft.

VENOUS MALFORMATION

The Klippel Trenauney syndrome is a congenital disorder usually affecting one limb only and consists of a port-wine stain on the lateral surface of the thigh, extensive varicose vein also predominantly on the

lateral surface of the thigh and limb hypertrophy. Venous ulceration may develop in unusual sites.

CONTACT DERMATITIS

RHEUMATOID AND OTHER VASCLITIC ULCERS

STEROID ULCERS

HYPERTENSIVE ULCERS

DIABETIC ULCERS

They may be the result of peripheral neuropathy, ischaemia or infection or often combination of all these factors. Neuropathic diabetic ulcers are the result of pressure and are therefore usually found on the sole of the foot, commonly under the first metatarsophalangeal joint or the heel. Ischaemic diabetic ulcers may affect the toes or forefoot or the heel, particularly as pressure sores in bed ridden patients.

Peripheral ischaemia in diabetes is often attributed solely to small vessel disease. Diabetics have a higher than normal incidence of atherosclerosis which may result in stenosis or occlusion of the main leg vessels. Arteriography may be necessary, even if ankle pressures are apparently within normal limits as the increased stiffness of diabetic arteries may lead to abnormally high pulse pressures.

TRAUMATIC ULCERATION

They may be the result of accidental trauma, self induced trauma or the result of injection sclerotherapy or operative surgery. Many patients with venous or ischaemic ulceration give a history of precipitating trauma. All patients presenting to surgical out patient department with abrasions or lacerations of the lower leg should be carefully examined for signs of venous reflux or arterial insufficiency. Abrasions and lacerations of the lower leg and ankle are always slow to heal, particularly in the elderly, even in the presence of normal arteries and veins.

LYMPHOEDEMA ULCERS

Ulceration is rare in lymphoedema. These ulcers are usually small, indolent and painless and occurs on the anterior surface of the ankle or lower leg. Photoplethysmography will distinguish lymphoedema from venous oedema. The cause of lymphatic insufficiency may be investigated by isotope or x-ray contrast lymphography.

TROPICAL ULCERS

MALIGNANT ULCERS

Squamous cell carcinoma developing is an established venous ulcer is known as **Marjolin's ulcer**. Squamous carcinoma should be suspected if there is any overgrowth of tissue in the base or at the edge of

the ulcer. Squamous cell carcinoma may also develop per primam on the leg and mimic venous ulceration.

Basal cell carcinoma may occasionally occur on the leg. Its appearance is less likely to mimic venous ulceration than squamous cell carcinoma and diagnosis is established by biopsy.

THE TREATMENT OF PRIMARY ULCER

- 1) *Ulcer cleaning and dressing*
- 2) *Compression bandaging*
- 3) *Elevation*
- 4) *Skin grafting*
- 5) *Pharmacological treatment*

ULCER CLEANING AND DRESSING

Slough in the ulcer base may *impede healing* and if not too extensive or thick this can be removed by various applications. Important principle of any dressing is that the dressing should be non-irritant and the exudate from the ulcer surface should be absorbed. Paraffin gauze dressings (Jelonet) have excellent non-adherent properties and are easy to apply and remove but occasionally they may give rise to allergic reactions.

COMPRESSION BANDAGING

Venous ulcers require firm compression to counteract venous hypertension and effect healing.

The main disadvantage of elastic stockings is their liability to soiling from exudate. There may be difficulty for the patient to apply at home.

Medium weight elasticated bandage (elastocrepe) should be used and this is then covered by tubular gauze to increase the degree of compression and to prevent the bandage slipping and losing its compression.

The essential features of compression bandaging in the treatment of leg ulceration are :

1. Adequate pressure (>15 mm Hg at the ankle)
2. Graduated compression from ankle to knee.

The importance of adequate compression has been demonstrated by Mc Collum by applying a three layer bandage – wool, and elastic compression bandage and a Coband dressing (3m) over the top – 60% of venous ulcers will heal within 8 weeks.

ELEVATION

Patients being treated for venous ulceration by dressings and compression bandaging must be firmly advised to avoid prolonged standing and to rest with the leg well elevated as much as possible. The legs must be elevated to heart level by lying back on a sofa or bed so that the foot and ankle are at or above the level of right atrium.

SKIN GRAFTING

Most venous ulcers heal in a mean 3 months with dressings and firm compression bandaging. Only a minority therefore require skin grafting. Venous ulcers can be grafted either after ligation of incompetent perforating and saphenous veins, with excision of the ulcer bed or at the same time as venous surgery is undertaken. It is wiser to treat grossly infected long standing chronic ulcer by Cockett's technique of delayed grafting following surgical excision of grossly infected tissues.

SSG usually taken from ipsilateral thigh are applied. Pinch grafts can also be used. They are taken either from the lower abdominal wall or from ipsilateral thigh. Cross leg flap or vascularised free flap are also used for non healing leg ulcers.

BISGAARD METHOD

The Bisgaard Method of treatment consists of Massage in elevation to the whole leg, Passive movements to maintain the mobility of foot and ankle, Active movements, to the calf muscles in elevation and in standing (with bandages on), Teaching correct walking placing heel down first and using the calf muscles to lift the heel and a firm elastic (e.g. blue line) bandage is applied spirally from the base of the toes to the knee so that the movements in walking alternatively stretch and relax the bandage and produce an added venous pumping effect.

PHARMACOLOGICAL TREATMENT OF LEG ULCERS

1) VENO ACTIVE DRUGS

a) **STANZOLOL** : It has a long term effect in stimulating natural fibrinolysis. It has significant side effect like androgenic action. So it is not recommended for liposclerosis or venous ulceration.

b) **DEFIBROTIDE (Fibrinolytic enhancement)**: Combined treatment by defibrotide and elastic compression is more effective in reducing both liposclerosis and ulceration and in improving microcirculating parameters and capillary permeability.

c) **PENTOXIFYLLINE** They inhibit granulocyte aggregation and production of oxidants from granulocytes. Lecocytes

elaborate either tissue type plasminogen activator or urokinase and that, through this mechanism, pentoxifylline can increase the rate of fibrinolysis.

2) VASOACTIVE PROSTAGLANDINS (Vasodilator and inhibition of platelet adhesiveness)

Intravenous PGE1 is shown to improve the healing rates of venous ulcerations.

3) VENORUTON: It is a semisynthetic flavinoid. It inhibits microvascular permeability and oedema formation. When it is combined with elastic stockings there is an increased rate of venous ulcer healing.

4) UROKINASE: Microthrombi in the vicinity of leg ulcers have been demonstrated by a number of authors. Urokinases effectiveness is yet to be evaluated in lysing the microthrombi.

5) TISSUE PLASMINOGEN ACTIVATOR CONTAINING OINTMENT (TPA): When applied topically for 12 weeks they heal the wound by its wound healing properties but not by fibronolytic property.

6) FACTOR XIII – When it is applied topically there is distinct improvement in healing of venous ulcers (Significant improvement in granulation tissue and marked reduction of secretion from the ulcer).

DEFINITIVE TREATMENT OF VENOUS ULCERS

Before performing surgery or injection sclerotherapy, the ulcer should be healed if possible.

Injection Sclerotherapy

Common sclerosants used are 1)Ethanolamine oleate 5% 2) Sodium tetra decyl sulphate 3%.

Technique

To make the venous occlusion permanent, injections must be given into an empty vein so that the wall adheres to the wall, and there is no intervening blood clot or thrombosis which will certainly recanalise . The needle is inserted into the vein with the patient sitting down and with the leg in a horizontal position. A latex foam pad is put over the site of the injection and along the whole length of the vein and compression bandaging applied. Injections should be given at the site of perforating veins and following this the patient is encouraged to exercise to the maximum.

THE SURGICAL TREATMENT OF VARICOSE VEINS

a) Surgery of the long saphenous vein

Before the surgery varices are marked with marker pen.

A Skin crease incision is made 3 cm below the inguinal ligament, extending laterally for about 5 cm from the adductor longus origin. The long saphenous vein is identified and followed to its termination at the foramen ovale where the superficial pudendal artery crosses beneath it.

The tributaries are dissected, ligated and divided. The long saphenous vein is divided close to the saphenofemoral junction which is then ligated.

A MYER'S metal stripper or one of its modifications is passed down along with long saphenous vein through its divided end and tied into place with a silk ligature. If competent valves are present in the upper part of the vein, the stripper will be unable to pass and the vein should then be simply ligated with a Chromic Catgut Suture. This retrograde stripping avoids unnecessary stripping of a normal saphenous vein with competent valves and also indicates the length of vein which requires stripping.

The stripper will normally pass down and incompetent long saphenous vein as far as upper calf where its main varicose tributaries join it. Distal to this point most long saphenous veins are normal in diameter and their valves are competent. Therefore no need to strip them.

Avoiding interference with normal distal long saphenous vein has two advantages

1) The normal segment of vein can be used for future coronary artery by-pass grafting if required.

2) Damage to saphenous nerve is avoided.

The most common complication of stripping the proximal long saphenous vein is severe haematoma formation in the thigh. Attempts to prevent this by tight bandages are usually unsuccessful. A Redivac can be used to prevent this. This is attached to stripper across immediately before the vein is stripped. The drain is removed after 24 to 48 hrs.

Between performing the groin dissection and stripping the long saphenous vein, individual varices are avulsed through very small stab incisions made with a No.11 blade. The leg is then elevated and firmly bandaged with a crepe bandage from toes to thigh. After first 12 hrs patient is allowed short walks. The crepe bandage can be removed and replaced by a tight (Class I) compression stocking 24 to 48 hours post operation.

SURGERY OF THE SHORT SAPHENOUS VEIN

It can be combined with long saphenous stripping. Termination of short saphenous vein is notoriously unreliable and often difficult to feel by simple palpation. The patient lies in prone position. Popliteal fossa is

explored through a 7-10 cm long skin crease incision. Deep fascia is opened in the line of the incision. The sural nerve is identified and preserved and short saphenous vein is identified between the heads of gastrocnemius.

The vein is carefully traced the popliteal fat towards its termination (feel for the pulsation of the popliteal artery). The tributaries may need division. The short saphenous vein is ligated just superficial to the junction of the gastrocnemius veins using 2-0 black silk. Opinion varies regarding stripping since there is no constant equivalent to Hunterian perforating vein. More distal stripping may lead to sural nerve damage. Stripping should be limited to upper 2/3 rd of calf. Bandage is applied as for long saphenous vein.

SURGERY OF THE CALF AND ANKLE PERFORATING VEINS

1) THE LINTON OPERATION (COCKETT'S MODIFICATION)

Linton recommended subfascial ligation of all perforating veins through different approaches by long incisions. Cockett's modification is approaching the perforating veins subfascially by small incision by posterior approach.

The sites of medial incompetent direct perforating veins are marked on the skin with ink. The incision lies over these, usually 2.5 – 5

cm posterior to the posterior border of the tibia. An incompetent perforating vein is usually 3 mm or more in diameter. Skin wound is closed and firm elastic compression dressing applied for two to three weeks post operatively.

The modified Linton subfascial approach can also be used to divide incompetent lateral perforating veins, the incision being placed just posterior to the fibula.

THE COCKETT OPERATION

It is extra-fascial division and ligation of incompetent calf perforating veins by straight incision lying one finger breadth behind and parallel to the posterior border of the tibia. Starting nearly half way up the leg it is extended to a point one inch above and behind the medial malleolus. The perforators are identified and ligated at their points of entry into the foramina in the deep fascia.

THE DODD OPERATION

This is posteromedial subfascial approach avoiding both medial and lateral indurated areas. Incision is placed 2.5 cm medial to the middle and the lower end is swung still more medially to avoid the Achilles tendon.

SUBFASCIAL ENDOSCOPIC LIGATION OF PERFORATORS

Subfascial endoscopic identification, bipolar coagulation, and division of in-competent perforating veins in patients with chronic venous insufficiency was first introduced by Hauer in 1985. A small incision, outside the area of trophic changes and induration of skin and subcutaneous tissue, allows the interruption of perforating veins under direct vision with minimal morbidity. Failure rate of ulcer to heal after this method varies from 2.5% to 11%. This failure may be due to limited accessibility of perimalleolar perforating veins that allow considerable manoeuvrability of endoscopic instruments.

Patterns of venous insufficiency should be determined by colour flow duplex ultrasound prior to surgery. Perforating veins are defined as vessels that penetrate the fascia and constitute continuously traceable connection between superficial and deep venous system. The only criterion for insufficiency is reverse venous flow as demonstrated by Doppler during relaxation phase after active dorsiflexion of ankle or manual compression of foot. Insufficiency is determined if reverse flow persists for >0.3 sec.

Endoscopic subfascial ligation can be performed with or without tourniquet. Mediastinoscope and clipping instruments are often used. All medial and dorsal perforating veins, that could be found are to be ligated

and divided. Patient can be mobilized on 1st post operative day and can be treated by ambulant compression therapy using short stretch bandages.

SURGERY OF DEEP VENOUS REFLUX

Deep venous reflux, usually post – thrombotic, is the most important factor in the aetiology of venous ulceration. Following calf perforating vein ligation, recurrent ulceration is inevitable in the presence of venographic evidence of deep vein damage.

Operations to prevent deep venous reflux may be divided into four groups:

- 1) Deep vein ligation
- 2) 'Sling' operation
- 3) Valve repairs, and
- 4) Valve transposition or transplantation.

It is disappointing that so many ingenious efforts to restore valvular function to the deep veins have shown poor or at best indifferent long –term results

MASTER CHART

<i>Sl. No.</i>	<i>Name, Age/sex</i>	<i>I.P.no.</i>	<i>Occupation</i>	<i>Duration of ulcer</i>	<i>Ulcer site</i>	<i>Varicosity</i>	<i>SFI</i>	<i>PI</i>	<i>Treatment given</i>
1.	Chockalingam /M/40	037856	Laborer	6 months	L-MM	LSV	+	MA	A+D+E
2.	Sabari/M/ 33	035467	Laborer	6 months	L-MM	LSV	+	MA	A+D+E
3.	Munusamy/M/35	030987	Laborer	6 months	R-MM	LSV	+	MA	A+D+E
4.	Ponrangan/M/23	036475	Laborer	6 months	L-MM	LSV	+	MA	A+D+E
5.	Prakash/M/45	034221	Printer	6 months	L-MM	LSV	+	MA	A+D+E
6.	Paneerselvam/M/54	036486	Laborer	6 months	R-MM	SSV	-	MA	B+E
7.	Gopalakrishnan/M/28	031233	Laborer	1 year	L-MM	LSV+SSV	+	MA+ BK	A+D+B
8.	Chandru/M/35	031564	Laborer	1 year	R-MM	LSV	+	MA	A+D+E
9.	Ulaganathan/M/65	030986	Laborer	8 months	L-MM	LSV	+	MA	A+D+E
10.	Ramasamy/M/34	034252	Laborer	6 months	L-MM	LSV	+	MA	A+D+E
11.	Henry/M/29	004533	Tea-Master	6 months	L-MM	LSV	+	MA	A+D+E
12.	Veeramuthu/M/34	029403	Laborer	6 months	R-MM	LSV	+	BK	A+D+C
13.	Pandian/M/38	023434	Laborer	1-1/2 years	L-MM	LSV	+	BK	A+D+C
14.	Govindan/M/47	024367	Laborer	6 months	R-MM	LSV	+	MA	A+D+E
15.	Babu.K./M/43	024356	Laborer	1 year	B/L	LSV	+	MA	A+D+C
16.	Firdaus/M/43	024311	Laborer	1-1/2 years	L-MM	DVT	-	-	F
17.	Karthikeyan/M/52	026343	Tea-Master	6 months	R-MM	LSV+SSV	+	MA	A+D+B
18.	Chinnamani/M/26	035578	Laborer	6 months	L-Dorsum	LSV	+	MA	A+D+C
19.	David/M/48	045363	Laborer	6 months	R-MM	LSV	+	MA	A+D+E
20.	Seethapathy/M/35	037589	Laborer	6 months	L-MM	LSV	+	MA	A+D+E
21.	Velu/M/38	045217	Laborer	6 months	R-MM	LSV	+	BK	A+D+C
22.	Kuppan/M/54	020674	Ironing	8 months	L-MM	LSV	+	MA	A+D+E
23.	Boobalan/M/51	021315	Laborer	1 year	R-Dorsum	LSV	+	MA	A+D+E
24.	Narayanan/M/43	023164	Laborer	1 year	R-MM	LSV	+	MA	A+D+E
25.	Zakhir/M/46	035423	Laborer	8 months	L-MM	LSV	+	LA	A+D+E
26.	Thangadurai/M/29	015353	Laborer	1 year	R-MM	LSV	+	MA	A+D+E
27.	Silambarasan/M/23	043312	Printer	6 months	L-MM	SSV	-	MA	B+E
28.	Kandasamy/M/48	035477	Laborer	6 months	L-MM	LSV	+	BK	A+D+C
29.	Ramesh/M/43	045372	Laborer	6 months	R-MM	LSV	+	MA	A+D+E

30.	Sakthivel/M/26	035533	Tea-Master	6 months	L-MM	LSV	+	MA	A+D+E
31.	Sabarathinam/M/55	029408	Laborer	1 year	R-MM	LSV	+	MA	A+D+E
32.	Pazhani/M/38	034978	Laborer	1 year	L-MM	LSV	+	MA	A+D+E
33.	Kalingarayan/43/M	035596	Laborer	1 year	R-MM	LSV	+	MA	A+D+E
34.	Narasimhan/M/44	020985	Ironing	6 months	R-MM	LSV	+	MA	A+D+E
35.	Kondiah/M/59	046596	Laborer	6 months	L-MM	LSV	+	BK	A+D+C
36.	Tamizhselvan/M/35	023764	Laborer	6 months	R-MM	SSV	-	MA	B+E
37.	Pavendran/M/54	026596	Laborer	6 months	L-MM	LSV	+	MA	A+D+E
38.	Babu.s./M/43	042840	Laborer	6 months	R-MM	DVT	-	-	F
39.	Srinivasan/M/45	036405	Laborer	1 year	B/L	SSV	-	MA	B+C
40.	Bangariah/M/66	034222	Laborer	1 year	R-MM	LSV	+	MA	A+D+E
41.	Bhushanam/M/46	023740	Laborer	6 months	L-MM	LSV	+	MA	A+D+E
42.	Kotiah/M/48	037475	Laborer	6 months	L-Dorsum	LSV	+	LA	A+D+E
43.	Bhaskar/M35	025448	Laborer	8 months	L-MM	LSV+SSV	+	MA	A+D+B
44.	Kumar/M/23	037354	Ironing	1 year	L-MM	SSV	-	MA	B+E
45.	Vetri/M/37	027242	Ironing	6 months	R-MM	LSV	+	MA	A+D+E
46.	Pitchumani/M/34	028406	Laborer	6 months	L-MM	LSV	+	MA	A+D+E
47.	Ganesan/M/54	045390	Laborer	6 months	R-MM	LSV	+	BK	A+D+C
48.	Giridharan/M/43	025649	Laborer	6 months	L-Dorsum	SSV	-	MA	B+E
49.	Monoharan/M/45	034486	Laborer	1 year	L-MM	LSV	+	MA	A+D+E
50.	Bharathi/M/61	030574	Housewife	8 months	L-MM	LSV	+	MA	A+D+C
51.	Seethiah/M/39	046390	Laborer	6 months	L-MM	LSV	+	MA	A+D+C
52.	Vayupandi/M/32	023659	Laborer	6 months	R-MM	DVT	-	-	F
53.	Stephen/M/34	036486	Laborer	6 months	L-MM	LSV	+	LA	A+D+E
54.	Varadhan/M/45	034121	Laborer	6 months	R-MM	LSV	+	MA	A+D+E
55.	Saravenan.G./M/43	037454	Ironing	6 months	L-MM	LSV	+	MA	A+D+E
56.	Rajendran/M/54	046489	Laborer	1-1/2 years	B/L	LSV	+	MA	A+D+E
57.	Thiagarajan/M/56	046589	Laborer	1 year	R-MM	SSV	-	BK	B+C
58.	Jaganmohan/M/41	034367	Laborer	6 months	L-MM	LSV	+	MA	A+D+E
59.	Sundaram/M/39	042121	Laborer	6 months	L-Dorsum	LSV	+	MA	A+D+E
60.	Karnan/M/33	025284	Laborer	6 months	R-MM	LSV	+	MA	A+D+E
61.	Sasikala/F/36	030561	Housewife	6 months	R-MM	LSV	+	BK	A+D+E
62.	Emmanuel/M/43	027243	Tea-Master	6 months	L-MM	-	-	BK	E
63.	Pakkirisamy/M/27	026489	Laborer	1-1/2	L-MM	LSV	+	MA	A+D

				years					
64.	Lifath ali/M/54	024439	Laborer	6 months	R-Dorsum	LSV	+	MA	A+D+E
65.	Janardhanan/M/43	026476	Laborer	6 months	L-MM	LSV	+	BK	A+D+C
66.	Nedunchezian/m/43	027240	Ironing	6 months	L-MM	LSV	+	MA	A+D+E
67.	Siva/M/34	045297	Laborer	8 months	L-Dorsum	SSV	-	MA	B+C
68.	Janardhanan/M/56	042198	Ironing	6 months	L-MM	LSV	+	MA	A+D+C
69.	Rex/M/34	042523	Printer	6 months	R-MM	LSV	+	BK	A+D+E
70.	Khadar/M/35	024437	Laborer	8 months	L-MM	LSV+SSV	+	MA+ BK	A+D+B+E
71.	Raghu/M/36	036596	Laborer	6 months	L-MM	DVT	-	-	F
72.	Mariamamma/F/43	019455	Housewife	6 months	B/L	LSV	+	MA	A+D+E
73.	Gunasekaran/M/32	026406	Laborer	1-1/2 years	L-MM	SSV	-	BK	B+C
74.	Manikandan/M/37	025490	Laborer	8 months	L-MM	LSV	+	MA	A+D+E
75.	Sankar/M/52	027638	Laborer	8 months	R-Dorsum	LSV	+	BK	A+D+C
76.	Kattabomman/M/35	026548	Laborer	8 months	L-MM	LSV	+	MA	A+D+E
77.	Jaya/M/33	026487	Laborer	6 months	R-MM	LSV	+	MA	A+D+E
78.	Malini/F/40	035407	Housewife	6 months	L-MM	SSV	-	LA	B+C
79.	Kaliappan/M/40	027636	Laborer	6 months	L-Dorsum	LSV	+	MA+ BK	A+D+C
80.	Ramadoss/M/59	034232	Tea-Master	6 months	R-MM	LSV	+	MA	A+D
81.	Puviarasan/M/43	025374	Laborer	6 months	L-MM	SSV	-	MA	B+C
82.	Kulothungan/M/25	026274	Laborer	6 months	R-MM	LSV	+	BK	A+D+E
83.	Arokiadass/M/35	025438	Printer	6 months	L-MM	LSV	+	MA	A+D+C
84.	Murugan/M/32	046478	Laborer	8 months	R-MM	DVT	-	-	F
85.	Satish Kumar/M/54	026254	Laborer	8 months	L-MM	LSV+SSV	+	MA	A+D+B
86.	Balachander/M/43	012343	Laborer	8 months	R-MM	LSV	+	MA	A+D+C
87.	Damodharan/M/34	036374	Laborer	1-1/2 years	R-MM	LSV	+	BK	A+D+E
88.	Raja/M/56	028726	Laborer	6 months	L-MM	SSV	-	MA	B+C
89.	Kamaraj/M/39	037345	Tea-Master	6 months	B/L	LSV	+	LA	A+D
90.	Mohammed/M/42	026347	Laborer	6 months	L-MM	LSV	+	BK	A+D+C
91.	Thanigavel/M/36	025332	Tea-Master	1-1/2 years	R-MM	LSV	+	MA	A+D+E
92.	Quereshi/M/43	026490	Laborer	8 months	L-MM	LSV	+	BK	A+D+C
93.	Parthiban/M/34	012263	Laborer	8 months	R-MM	LSV	+	MA	A+D+C

94.	Sakkarai/M/38	027253	Laborer	1-1/2 years	L-MM	DVT	-	-	F
95.	Poongavanam/M/30	027243	Tea-Master	8 months	L-MM	SSV	-	BK	B+C
96.	Ramachandran/M/54	025478	Laborer	1-1/2 years	R-MM	LSV	+	BK	A+D
97.	Lingaiah/M/52	045373	Laborer	6 months	L-MM	LSV	+	MA	A+D+E
98.	Varadharajan/M/37	037648	Laborer	1-1/2 years	R-MM	LSV	+	MA	A+D+E
99.	Poonguzhali/F/42	030874	Housewife	6 months	L-MM	SSV	-	MA	B+C
100.	Venkiah/M/31	020586	Laborer	6 months	R-MM	SSV+LSV	-	BK	A+D+B
101.	Shanmugam/M/61	046607	Laborer	6 months	L-MM	LSV	+	MA	A+D+E
102.	Salauddin/M/43	037348	Laborer	1-1/2 years	L-Dorsum	LSV	+	MA + BK	A+D+C
103.	Dinesh/M/37	036458	Laborer	8 months	L-MM	LSV	+	MA	A+D+E
104.	Amudhan/M/39	048462	Printer	8 months	R-MM	LSV	+	MA	A+D+C
105.	Bharani/M/31	037659	Laborer	6 months	L-MM	DVT	-	-	F
106.	Kuppusamy/M/30	037586	Laborer	6 months	L-Dorsum	LSV	+	MA+ BK	A+D+C
107.	Jayagopal/M/46	027689	Laborer	6 months	R-MM	LSV	+	MA	A+D+E
108.	Uthirapathy/M/54	024375	Laborer	6 months	L-MM	LSV+SSV	+	MA	A+D+B
109.	Jayaraman/M/45	034436	Laborer	6 months	L-MM	SSV	-	BK	B+C
110.	Marimuthu/M/32	032648	Laborer	1 year	R-MM	LSV	+	MA	A+D+E
111.	Selvam/M/39	036478	Laborer	1-1/2 years	L-MM	LSV	+	LA	A+D+C
112.	Anand/M/29	025232	Tea-Master	8 months	L-MM	SSV	-	MA	B+C
113.	Krishnan/M/54	034434	Laborer	8 months	R-MM	LSV	+	LA	A+D+E
114.	Sashivardhan/M/38	046462	Laborer	6 months	L-MM	LSV	+	MA	A+D
115.	Muthu/M/28	025233	Laborer	6 months	R-Dorsum	LSV	+	MA	A+D+C
116.	Murali/M/40	035223	Laborer	6 months	R-MM	LSV	+	MA	A+D+E

A— Trendelenberg operation

D—Stripping of varicosities

B— SPI Flush ligation

E—Flush ligation of perforators

C— Multiple stab avulsions

F—Conservative

MA- medial ankle

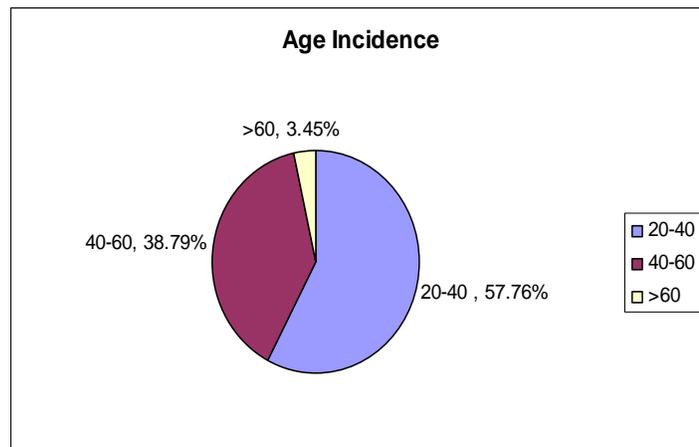
LA – lateral ankle

MM—medial malleolus

BK – below knee

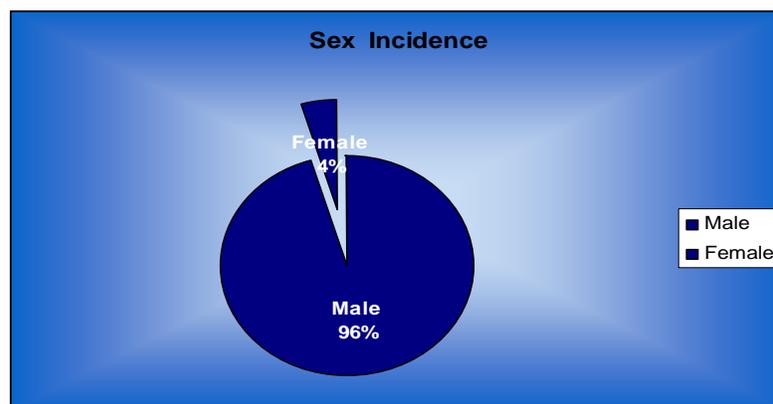
RESULTS OF THE STUDY

AGE INCIDENCE



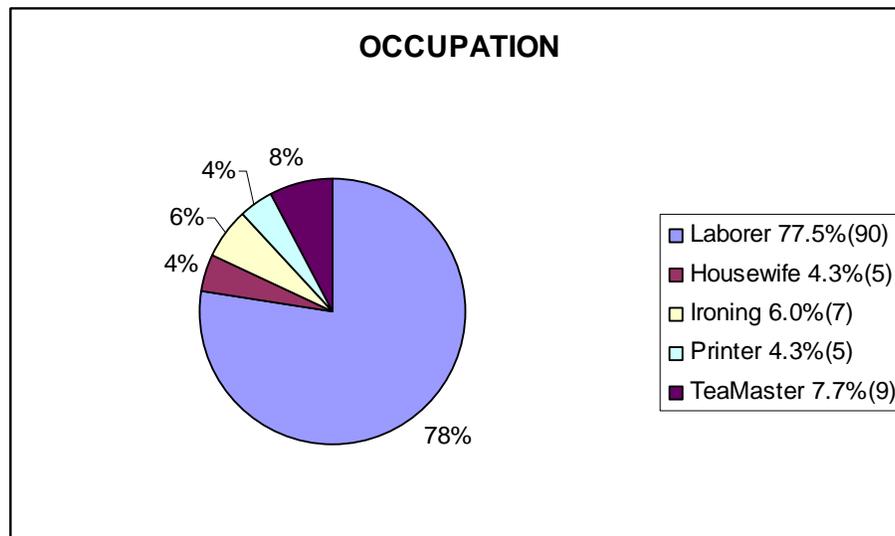
In my study the majority of patients with Venous Leg Ulcers were in the age group of 20-40 years, which constituted 57%. The youngest patient was 20 Years old and the oldest was 65 Years old.

SEX INCIDENCE



Mainly Males were affected.

OCCUPATION



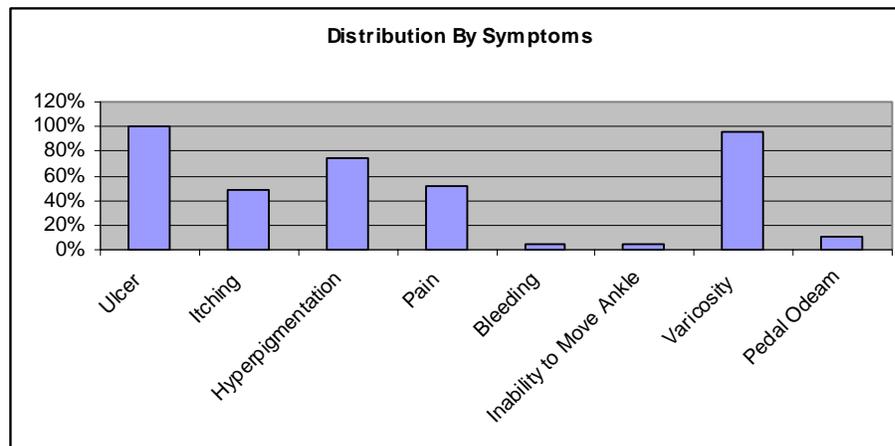
Most of the patients were laborers who work in standing posture most of the time.

INCIDENCE OF LATERALITY

SIDE	No. Of Patients	Percentage
Left	65	56%
Right	46	40%
Bilateral	5	4%

Left side ulcers were more common than right side. Among inpatients bilateral disease (Ulcer) was present in only 5 patients.

SYMPTOMS



Ulcers were multiple(≥ 2) in 5 patients. 48% patients complained of itching (so also pain). History of bleeding was present in only one patient with varicose ulcers.

- a) Urine – All diabetic patients are excluded from the study
 - Only in 18 patients urine examination revealed trace of albumin in urine.
- b) Haemogram – Only one patient showed Hb of 8grms % who was unfit for surgery. All others showed a Hb of >10 gms%.
- c) No abnormal rise of ESR was noticed in any of the patients.

WOUND SWAB FOR CULTURE & SENSITIVITY

Organism Grown	No. Of Patients	Percentage
Staph. aureus	43	37%
Klebsiella	13	11%
E.Coli	17	15%
Pseudomonas	8	7%
Mixed	8	7%
No Growth	27	22%

These results were comparable to British reports. Staph aureus was a common organism presented in 37% of ulcers. No growth was found in 22% of patients. Gram –ve organisms were found in 29% cases.

RESULTS OF EXAMINATION OF 116 LIMBS

Defect	No.	Percentage
1.Long Saphenous Varicosity	103	89 %
2. Saphenofemoral incompetence	92	79%
3. Perforator incompetence	100	86%
Medial ankle	92	79%
Lateral ankle	3	3%
Calf	6	7%
Below knee	65	51%
Mid thigh	30	20%
4. Small saphenous varicosity with SPI	8	3%
5. Combined small & long saphenous varicosity	5	3%
6. Bilateral disease	5	7%
7. DVT (Perthe's Sign +)	5	2%

Among 116 patients admitted with venous ulcers five patient had DVT five had bilateral disease (Varicose Vein). Combined small and long saphenous disease was found in 5 patient.

DETAILS OF SURGERY DONE

Surgery done (on 110 limbs)	No. of Limbs	Percentage
Long Saphenous Ligation and or stripping	103	89%
Short Saphenous Vein Ligation	9	8%
Multiple Avulsion	19	16%
Cockett's Procedure	9	8%
Conservative Treatment	11	10%

Among 116 patients 110 limbs were operated. 103 patients underwent long saphenous ligation and or stripping. Cockett's extrafascial ligation done in 8 cases. Multiple avulsion done in 19 cases. Short saphenous vein ligation done in 8 patients.

COMPLICATIONS OF SURGERY

Complication	No.	Percentage
1. Wound Infection	17	15%
2. Wound Haematoma	4	4%
3. Wound gaping	9	8%
4. Anaesthesia related (Headache)	24	20%

CONCLUSION

From the study 116 cases of venous leg ulcers I was able to arrive at some conclusions.

1) Though venous ulceration is described as a disease of elderly, it is found equally common among young adults as per my study. This could be due to more prolonged standing occupations and perhaps young adults are more prone to traumatise themselves while working.

2) Male : Female incidence is 24 :1 i.e. Females in Chennai are more resistant to venous ulceration when compared to males. Females here are more homebound and they can relax more in between their working hours.

3) DVT as a cause of venous ulcer as per my study is 5/116 (approx. 4%). This is pretty less when compared to Western Statistics.

4) Our Indian patients have excellent tolerance. Even with leg ulcers they continue to work. This is evidenced by a history of upto 7 years duration of leg ulcer. This may be due to indifference and ignorance.

5) Leg ulceration is common in poor people below poverty line. (Perhaps we can not compare with the available data alone because many

middle class and most high class people never come to Govt. Hospital). This may also be related to their nutritional status.

6) The common associations of ulcer are hyper pigmentation followed by pruritis and pain, especially on prolonged standing which is relieved by keeping legs elevated. Pain is most often felt at the ulcer site and some times also felt involving entire leg.

7) Left sided venous ulcers are more common than (R) side ulcers. This may be due to loaded sigmoid colon compressing veins draining the (L) lower limb.

8) Wound haematoma though described as a common post operative complication, it is rare where absolute meticulous haemostasis is achieved during surgery.

9) Failure to ligate the incompetent medial ankle perforator is the most common cause of recurrent venous ulcer. At the same time even when all the perforators are ligated meticulously the ulcer recurs after some time. This shows that our understanding is not yet complete.

10) We have to spend more time in teaching the patients regarding compression bandages, and to demonstrate them how to wear it.

11) Arterial component contributing towards venous ulcer were excluded by clinical palpation of pedal pulses. In few cases difficulty encountered in palpating which was overcome with the help of Doppler.

12) Stripping of great saphenous vein was performed upto just above ankle in almost all cases. This stripping should be limited to upper calf, since distal to this Part most long saphenous vein is normal in diameter and can be used for Bypass Surgeries.

14) Though the patients were advised to come for follow-up only half of the patients responded (55 out of 110). One month post surgery most were found to heal except in five patients who did not wear elastocrepe as advised.

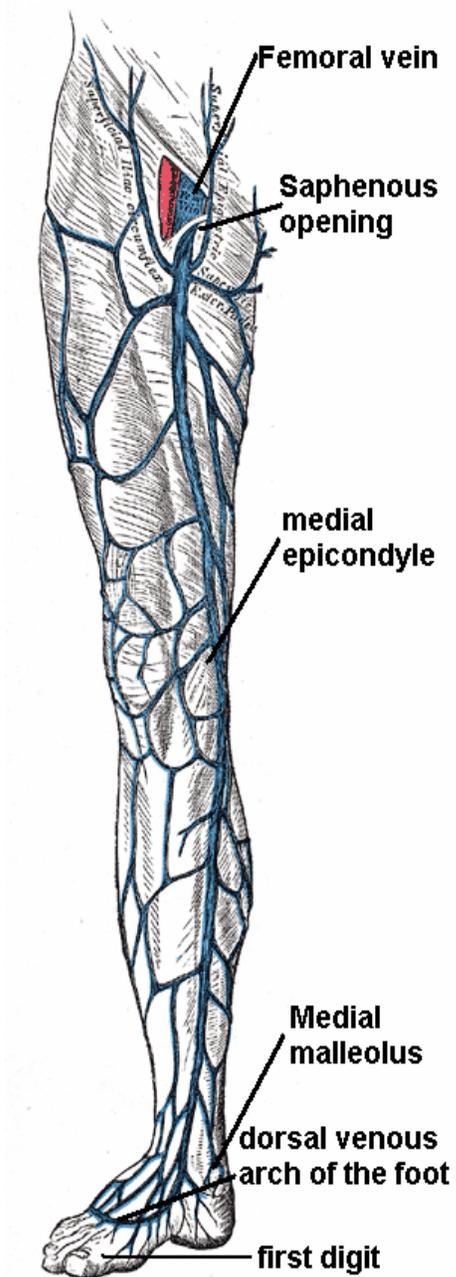
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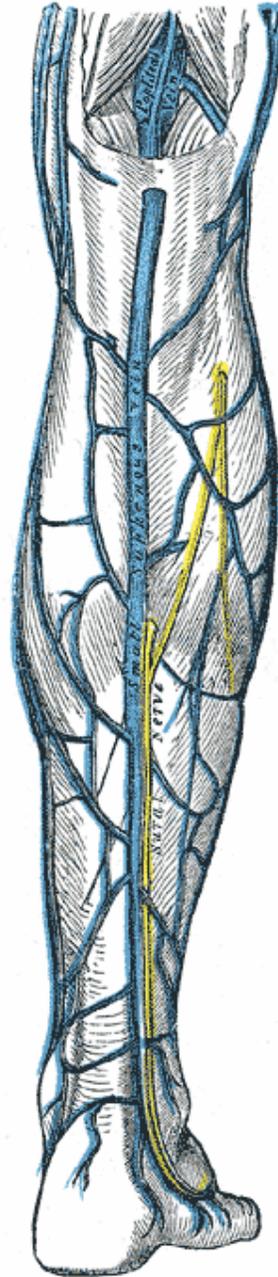


Large ulcer above ankle

GREAT SAPHENOUS VEIN



SHORT SAPHENOUS VEIN



- a) Small Saphenous
- b) Long Saphenous

Incompetence	SF Valve / SPI	
(Clinical + Doppler)	Perforator	a) MA (Medical Ankle)
		b) LA (Lateral Ankle)
		c) MC (Mid Calf – part of medical ankle)
		d) BK (Below knee)
		e) MT (Mid thigh)
	Perthe's Test (DVT)	
	Pedal Oedema	
	Movement of ankle joint	

Periosteal thickening
 Regional Lymphadenopathy
 Cardiovascular Examination

S1

S2

Murmurs

Peripheral pulses	(L)	(R)
- Radial		
- Ulnar		
- Brachial		
- Femoral		
- Popliteal		
- Dorsalis pedis		
- Posterior tibial		

Abdominal Examination :

Free fluid

Mass

Organo megaly

Varicocele

Investigations :

Hb%

Urine - Alb.
- Sugar

Blood - Urea
- Sugar

ESR

Swab for C/S

Doppler

Biopsy if any from edge

Operation : Conservative/ Ligation/Ligation & Stripping/EFL/SFL/ SSG

Result at the time of discharge : Wound healing / static

Result after one month : Wound healing / static.