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The Treatment of Varicosities with special
reference to the lower extremities

Edward Joseph Clarence Panzer

Senior Thesis presented to the
College of Medicine, University
of Nebraska.

Omaha

1940

The material written upon this subject is vast. It has been the problem of the writer to set out in consequential order the trends of that particular period and try and embody the latest thoughts of the day with this subject.

In order to understand the treatment of varicose veins, it will be necessary to bring into the reader's mind the basic problems from which he can get some idea as to the workings of the methods used. Hence the Anatomy, Histology and Physiology of the veins are mentioned to refresh the reader's mind.

The importance of the treatment in varicosities cannot be stressed too much and it is a boon to a great many people that it has reached an efficient position, although far from perfect, at the present time.

Although there are other types of varicosities special attention is given to the lower extremities.

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CHAPTER I

The veins of the lower extremity.

The veins of the lower extremities (Lewis 57)

consist of:

1. Superficial veins
2. Deep veins
3. Communicating veins

The superficial veins consist of the great saphenous and small saphenous veins with their tributaries. Great saphenous vein starts in the medial marginal vein of the dorsum of the foot. It passes in front of the tibial malleolus, upward on the medial side of the lower leg passing behind the condyles of the tibia and femur and along the medial side of the thigh and passing through the foramen ovalis into the femoral vein about 3 cm. below the inguinal ligament. This vessel is the longest vein in the body. The small saphenous vein begins as a continuation of the lateral marginal vein behind the external malleolus and runs upward along the tendo calcaneus ligament which it soon crosses and travels upwards in the middle of the posterior portion of the leg. Reaching the popliteal fossa, it perforates the deep fascia and ends in the popliteal vein.

The deep veins consist of:

1. Plantar digital veins

2. Posterior digital veins
3. Anterior digital veins
4. Popliteal vein
5. Femoral

The plantar digital vein is formed from the plexuses of the plantar surfaces of the digits and with the veins of the dorsum of the foot form the deep plantar arch which after communicating with the great and small saphenous veins unite behind the medial malleolus to form the posterior tibial vein.

The posterior tibial vein follows the course of the lateral aspect of the tibia and goes with the anterior tibial to form the popliteal vein.

The popliteal vein formed from the anterior and posterior tibial veins ascends through popliteal fossa to the abductor magnus where it becomes the femoral vein. The femoral vein at first lies lateral to the femoral artery then behind it and at the inguinal ligament lies medial to the femoral artery. At about 4 cm. below inguinal ligament the femoral vein receives the profunda femoris (Clarkson 11). Near the termination of the femoral vein it is joined by the great saphenous which has the superficial circumflex iliac, the superficial external pudental and the superficial inferior

epigastric branches draining into it. These are above the juncture of the medial and lateral femoral cutaneous branches. These last two veins, particularly the medial, make frequent communications with the more distal branches of the long saphenous vein. Further important communications of the medial vein are with the superficial, external pudental and more posteriorly the inferior sciatic veins. (McPheeters 73,74).

The communications are veins that unite the superficial veins with the deep veins. Most of the blood is drained through these communicating branches into the deep veins which flows toward the heart. These facts are important and must be remembered. The number of communications (McPheeters 74), confirmed by Loder and Remy, are six in the foot, fifteen in leg and seven in thigh.

The larger veins mentioned contain valves which are so formed so that blood will flow toward the heart and not able to flow backward. (McPheeters 74) states "the deep veins contain more valves than the superficial veins." He lists them - Great Saphenous 3, Anterior Tibial 9, Post. Tibial 9 and the peroneal 7. The femoral according to Gray Anatomy contains 3 valves. Horizontal veins do not have valves according to

Meissen. There is a constant (McPheeters 73,74, Sabrazes 98) ostial valve at mouth of femoral vein.

It must be remembered that the superficial veins of the lower extremities are not well supported since they are situated beneath the superficial fascia. The deep veins on the other hand are well supported by aponeurosis and muscles.

The circulation in the lower extremities is aided by two factors (Sabrazes 98) muscular movement and by the aspiration of blood during diastole of the heart, by the increase in size of the thorax during inspiration which draws the blood from the veins toward the heart and the elasticity of the veins when they are dilated. McPheeters (73) doubts the latter portion of this statement. He believes it plays a part, but not a large part that some authors say. (McPheeters 74, Sabrazes 98)

The veins (Maximow and Bloom 67) consist of 3 layers, intima, media and adventitia. The intima consists of flattened endothelial cells supported by a layer of C.T. which may be thickened in patches. It also contains (Sabrazes 98) small bundles of smooth muscle fibers running parallel with the direction of the blood stream. According to Sabrazes (98) this

The musculature of these veins contributes in regulating the flow of blood and the veins can adapt themselves to the most varied circulatory conditions on account of their elasticity and contractility. The

hemorrhoidal veins, submucous veins of esophagus, varicosities in broad ligament, in subcutaneous veins of abdominal wall in liver cirrhosis, superficial veins of upper extremities.

The various states of varicosities are graded by several means. Sabrazes (98), McPheeters (73) grades them according to the individual anatomical structure of the veins. Bernstein (116) classifies the various states depending upon their pathological formation. McPheeters has the size of the varicose vein measured and has them divided into four groups with a fifth group that would include all others there-to-fore.

- | | |
|------------------------------------|-------------------------------------|
| 1. $\frac{1}{2}$ cm. in diameter | 3. $1-1\frac{1}{2}$ cm. in diameter |
| 2. $\frac{1}{2}-1$ cm. in diameter | 4. $1\frac{1}{2}-2$ cm. in diameter |

Clarkson (11) to me appears as the most useful for the average physician. One might combine McPheeters classification with that of Clarkson.

Clarkson's Classification.

Grade I. Trendelenburg's first test negative. The valve at the sapheno-femoral is competent. The vein does not fill from above with blood after it has been emptied. Injection above indicated.

Grade II. Complete valvular incompetence of

saphenous chain. The Trendelenburg test is positive. The hydrostatic pressure is 200/H₂O at the ankles. High ligation with subsequent injection.

Grade III. There is a valvular incompetence of saphenous trunk and also of deep communications. The hydrostatic pressure is the greatest in this instance and the Trendelenburg test is doubly positive. (High ligation and retrograde injection failing in this, some set operative procedure designed to interrupt and ligate the deep communication is indicated).

ETIOLOGY III

The causes of varicosities are many and varied. No specific one can be picked out and say it is the sole cause. I do believe this that there may be some instances in certain individuals that the exact cause may be stated.

Varices have been considered to be due to various conditions such as valvular insufficiency, congenital predisposition, the upright position, pregnancy and intestinal stasis. (Millet 68). However, all of these conditions can be eliminated because they are very common and cannot be considered as being definite causes. Millet (68) believes that the origin of varices should be searched for in a syndrome which is common

to all cases and not to heterogenous and variable cases. This condition may be possible but as was stated before, the syndrome might exist in those cases where the etiological factor was outstanding. Higgins (32).

Ever since man started in the upright position he never did get himself thoroughly adjusted to this position as proven by the varicose state.

Kashimura (McPheeters 73) explains the cause to be a loss of the nervous and muscular tone of the vein wall which allows the dilatation to take place. This Japanese surgeon states that there is an increased tone at first with a secondary compensatory overgrowth, particularly of the media and intima, then a terminal relaxation. It appears true because of the fact that the oriental people do most of their work in the sitting position or a squatting position. Yet it can also be noticed that the oriental people that do work requiring standing for long hours that they are prone to varicosities as much as the other individuals.

McPheeters (73) writes and states that Pierre Delbert approached the cause from a mechanical standpoint. Pierre Delbert's theory is that the original cause is the weakening of the external iliac veins which allows back seepage and creates more

pressure upon the saphenous valves. But then there are varicosities that appear both above and below competent valves. A factor which might be considered is that any increased abdominal pressure due to heavy lifting, coughing, sneezing or any condition which causes an increase in contracture of abdominal muscles. (McPheeters,73)

Horgan (36) believes that there is a congenital weakness of the middle layer of the vessel wall. This will cause a dilatation, either due to some secondary factor as extra pressure, age, occupation, etc., that will separate the valves causing a reflux of blood.

McCausland (71) does not hold to the pressure theory at least not as an important factor. Varicose veins develop early in pregnancy before any real pressure is started. It may, however, be a factor late in pregnancy because of a reduction in the negative intra-abdominal pressure.

McPheeters (73), de Takats (105) seem to think that the infection theory should be given serious consideration in all cases. The origin may either be hematogenous, embolic or be from direct extension from localized infection in a neighboring field, such as occurs following injuries and traumas. There would follow a low grade phlebitis or even a periphlebitis that would

not be recognized clinically. Even though not recognized clinically there would be a starting destruction of the vein wall. This would continue in its pathological course until the media layer became involved. This would be replaced by fibrous tissue, that although it has resisting power does not have elasticity. McPheeters (73,74) in this same book states that Thorel and others believe the infection comes through the vaso vasorum and directly involves the media. The fact that the development of varicose veins is so definitely associated with attacks of typhoid fever, diphtheria, pellagra, influenza, bronchial infection, pelvic infection in women, etc., most certainly lends positive evidence to the theory of an infection as a primary factor in the causation and development of varicose veins.

The inflammatory theory upheld by Noble (McPheeters 73) appears quite illuminating and upholds the ideas set forth by Delbert, Magnus, Hasebrock. It is apparent and logical to think that as the blood passes the valves portions become stagnated due to the fact that the vein is dilated above the valve. (de Takats 105, McPheeters 73). This would affect the valves if infection was present and would prove that the valves are affected first with the dilatation

of the veins secondary.

That heredity plays an important part in a small percentage cannot be doubted. A senior medical student, class of 1940, has a small varicosity on the anterior portion of his lower extremity. On further questioning it was found out that his mother and father both were afflicted with varicosities since childhood. The writer knows of another family near Lexington, Nebraska, that are afflicted the same way. The father had varicose veins as long as he can remember and two of his three children (age 16,20) have evidence of early varicosities of the lower extremities.

Sicard (102) states that varicosities develop because of an endocrine absence. McPheeters (73) thinks if this were so then it should be remedied by treating patient that was afflicted with the administration of an endocrine product. So far this has been unsuccessful. McPheeters (73) states that the spider bursts occurring in females after the menopause is in favor of the ovarian dysfunction. Also it has been brought out that there should be more varicosities noted in females that have hysterectomies and double ovariectomies, a condition which seldom does occur.

Age and sex play an important part according to McPheeters (73), McCausland (71), Millet (68). The older a person gets, it is thought that the valves are degenerating. The degeneration of the valves due to age, plus any secondary factor such as increased pressure, infection, etc., will tend to bring on the varicose state. According to Bernstein (116), McPheeters (73) most varicose veins occur before the 30th year (75%). McPheeters (73) series shows almost the same percentage except his highest percentage was in the ages from 30-50 years. It was also found in this series that women are afflicted much more than men. They are in about the same ratio up to 20 years of age, after that age the ratio is in favor of the females. Higgins (32).

One fact cannot be forgotten and that is the occupation factors. Higgins (32) When an individual is walking the action of the muscles on the deep venous system tend to help the flow of blood toward the heart. And in this case they tend to "milk" the blood from the superficial blood vessels. But when an individual is doing work that means standing in one location there is proof to be a definite lack of the movement of the blood. This will cause an excess amount of blood in the lower

extremities and cause excess back pressure upon the valves. I believe the important thing here is not so much back pressure upon the valves but a dilatation of the veins between the valves. Logically, this would cause a separation of the leaflets of the valves, permitting a reflux of blood in the segment below.

SUMMARY

1. Only in specific cases can an etiological factor be determined as the absolute cause.
2. There will be a small percent where the etiological factor can be traced to heredity. A greater percent can be attributed to infection and inflammation.
3. Still another group will come until the occupational factor.

TESTS IV

Mahorner and Ochsner (63,65) devised a test whereby the varicose veins are inspected on the patient. The size is noted when sitting and walking. After this the tourniquet is placed around upper one-third of thigh to compress superficial vein. The comparison as noted should be 50-75% less. Muscles milk into deep and out, then tourniquet removed and placed over middle one-third. Then lower one-third and observations

recorded.

This is to determine best type of therapy and possibilities of recurrence.

G. H. Pratt (93) uses two tourniquets to see if blow out needs ligation.

Sir Benjamin Brodie wrote about the test and later Trendelenburg picked it up. In 1891 Trendelenburg (reprint by Tice) gave to the world his now famous Trendelenburg phenomenon. According to Bernstein (McPheeters 73) he classifies the Trendelenburg into four groups.

Group I Trendelenburg positive. Where the flow is entirely from above downward and the spill is at the sapheno-femoral opening or from some communicating branches above the knee.

Group II Trendelenburg negative. Where the back flow comes from the communicating branches that are located below the knees. The valve at the sapheno-femoral juncture is competent and there is no reverse flow from above the knee.

Group III The Trendelenburg double. This condition arises when the varicose state is extensive and there is a reflux of blood from above (saphenous) and also a back flow from the communicating branch. In

other words this is merely a combination of one and two.

Group IV The Trendelenburg nil. In this condition the valves are competent but the veins themselves have become dilated and given way. Bernstein (Tice) states that this is the primary factor in the development of varicose veins. The veins become so dilated that the valves are unable to have their edges together causing a reflux of blood.

This test (McPheeters 73) can be carried out by having the patient stand. The examiner then notices the large varices, marking the exact spots of the larger varices. The patient is then allowed to lie down horizontal with the afflicted leg raised in the air so as to drain the veins. Then by pressing at the femoral-sapheno juncture, the patient is allowed to stand. The time that it takes for the veins to fill is noted very carefully. From 20-30 seconds is considered the time that it takes for the veins to be filled from the capillaries of the foot. If the time should be shortened then the deep communicating veins are involved. The portion involved will be the veins in the upper one-half of lower extremity. Now release the pressure above and notice if there is a swelling of the veins in the leg from the reflux of blood spilling

hand tap the large varices below. This test will locate the great saphenous vein and when marked will allow the injection of sclerosing solution into an empty vein while the patient is in the horizontal position.

Kilbourne (49,50) states that the Perthes test, which is a modification of the Trendelenburg tests, can determine whether the deep venous system is patent. Place a tourniquet just above the knee and have the patient walk. If the veins swell and the patient complains of pains which increase as he walks then the deep veins are not patent. This would indicate a block of the deep system and they are using the superficial veins as a "by pass". This is an absolute indication to not inject the superficial veins. This will be brought out more in detail later.

DIAGNOSIS AND DIFFERENTIAL DIAGNOSIS V

At a glance the diagnosis would look apparently easy but such is not the case. According to McPheeters (81) the history of the patient is very important. It should be detailed and to the point. Faxon (21) believes that a routine method of inquiry is an efficient means of evaluating the symptoms referable to the varicosities. It is true that be-

cause of the obvious nature of the veins, the patient attributes symptoms to them, which, from the story alone, can be ruled out as arising from this source. For example, any symptoms at or above the knee are, with few exceptions, not due to varicosities and a history of intermittent claudication is far more commonly attributable to arterial insufficiency than to stagnation in the venous channels.

The questioning concerning the local conditions should be such as to reveal matters relating to familial tendency to varicosities, duration and increase in size of the veins, edema, eczema, ulceration, heaviness of the leg and past episodes of phlebitis. Inquiry concerning systems, unrelated to the peripheral circulation is of obvious importance in forming some judgment of the patient's general condition.

When the history is taken and evaluated, then the examination of the patient as to the location and type of involvement is determined.

In order to come to a correct diagnosis, one must evaluate all those diseases that have pain and discomfort in the lower extremities. Much cannot be written here except to mention those diseases that

will confuse the unwary. Menopausal arthritis, flat feet, neuritis, diabetes, compensatory veins, Buerger's disease, Reymand's disease, arterio-venous aneurysms, Varicose veins secondary to the presence of pelvic tumors, compensatory veins due to a true blockage of the deep veins. McCausland (71)

The examiner has to be sure that there is no blockage of the deep system. This can be determined by the Trendelenburg-Brodie and the comparative test as given by Mahorner and Ochsner (63). Some believe that pregnancy is a contraindication (Dawkins 15), McPheeters new book (73) believes that they can be treated around the seventh month. McCausland (71) at the Los Angeles Fraternity considers them amendable to treatment if one is careful in choice of patient, and technic. Siegler (103). Any advanced, cardiac, pulmonary or renal diseases and diabetics are contraindications. (Dawkins 15) Marked cirrhosis of the liver is a contraindication. No attempt must be made to sclerose a vein which is the seat of an infection. (de Takats 105) After the attack has completely subsided, injections may be undertaken if necessary. Whether old deep thrombosis is a contraindication or not is a much debated point. Its

presence should be suspected if there is much swelling, edema or difference in the color and temperature of the two legs. (Dawkins 15, McPheeters 73,78,81, de Takats 105, Faxon 21)

TREATMENT VI

It is the plan here to give an idea of both the injection methods and the surgical procedure that have been tried in the past.

McPheeters (73,78), Johnson (44) writes that Hippocrates (1) thought to puncture the varicose veins in many places which would allow healing to take place by fibrosis. He did not advise cutting open the veins because of the occurrence of ulcers. In the first century, Celsus (2), as did Avicenna, would cut the veins out by cautery being careful of not touching the skin edges. Algeneta (2) exposed the vein by incision then passed needle with double thread under it and allowed blood to go out. They were then tied in the upper and lower portions of the involved veins. A big step was taken when Pravaz (McPheeters 73) invented his syringe in 1851. He would inject aneurysms with Perchloride of Iron, but got terrific reactions and sloughs. The French Lyons school used the Pravaz method in the treatment

for Varicose Veins. They soon became discouraged because of the results and complications.

In 1853 Chassaignac of St. Antoine Hospital of Paris (McPheeters 73) made a series of trials of this new method. The results were very unfavorable. Some were successful but in the largest proportion of the cases there were sloughs, abscesses, septic emboli. Becoming discouraged the treatment was given up. The peculiar and ironic feature of this was that they were on the right track but there was no knowledge of asepsis.

In 1854 Desgranges cured 16 cases by using "liquor Indo tannique" devised by Socquet and Gullermond (McPheeters 73). Its composition was Iodine 5 gms., tannus 45 gms., H₂O 50 gms. The dosage was 5-7 drops. The reaction obtained was milder than that given by Perchloride of Iron. The patient was also kept in bed ten to twelve days.

In 1875 Valette Douthwaite (118) used iodine and tannic acid. Patient had fortnight for recumbancy.

Then there was a flurry of different solutions used. Gugon (1876) used perivenous injections of Ergot of Rye. This proved very unfavorable. In 1878 the English physician used 5% alcohol but Broca

used 30%. Pupi changed to Chloral Hydrate and it was the start to find a different solution. All these solutions were given up by 1894, although even at this time Delore defended them. McPheeters (73)

The point I wish to make here was that the solutions used were blood coagulants and did not have the sclerosing effect upon the venous walls.

The second greatest advance was made by Trendelenburg in 1891 (Johnson 44,45) when he took Sir Brodie's explanation of the varicose state and also advised ligation (1891). Now we are approaching the idea of getting the surgeons and the medical men together on this problem.

In 1904 (Tance) injected 5% carbolic acid after ligating internal saphenous vein.

In 1905 and 1906, Horgan (36) Keller and Mayo (119), Homan (35) Babcock (117) advised stripping of entire saphenous vein above knee and extirpation en masse below knee. A special apparatus was made for this type of operation. Multiple small incisions were made along course of veins. The large incision was made in the upper third of thigh so as to expose the saphenous vein. It is ligated and the stripper placed over the distal portion, passed down the vein

tearing or cutting all of its branches. No effort was made to tie off bleeders. Small incisions below were made so as to draw the apparatus out. The leg was then bandaged tightly so as to produce hemostasis. This procedure is practically eliminated today although some men (Horgan 36) still use a modified form of the original method.

Schiasso in 1908 had a solution made up consisting of Iodine 1 gm., K.I. 1.6 gms., H₂O 100 gms. He divided the varicose vein below the knee, placed cannula into distal end and injected 30-50 cc. of this solution. The vein would then be ligated below the cannula.

Linser (McPheeters 73) used one-half 1% of Perchloride of Mercury in 1916 and had some success with it. But on further follow up cases, people were found to have complications of nephritis, stomatitis and enteritis. This treatment was soon abandoned.

Sicard (102) 1916-1917 was giving some luargol intravenously and noticed that the veins became obliterated without embolus. He thought it was the soda part of the salt. So in 1917 he used Carbonate of Soda and found out by experience that it was too caustic. Finally he chose Salicylate of Soda and up

to 1930 still recommended it. This contribution by Sicard was the first solution that did not solely act as a coagulant of the blood but rather caused an inflammatory reaction of the venous wall.

Homans (35) in 1916 and again in 1934 recommended ligation of the saphenous vein between terminations of deep femoral and internal saphenous vein in order to forestall pulmonary embolism in thrombosis of deep veins of legs and calf.

Genevrier in 1917 (25) used Quinine and Urethane which proved to be better than Sicard's solution and came closer to being the perfect sclerosing solution.

Dalton (Douthwaite 118) in 1928 used pure liquid carbolic acid but this was immediately dropped because of complications.

Douthwaite (118) in 1929 used neutral Hydrochloride with quinine. This solution was made up of Quinine Hydrochloride (B.P.) 4 gms., urethane 2 gms. and distilled H₂O 30 gms. He gave over 3,000 injections. On a smaller number of people na salicylate, glucose and Meisen's solution was used. He did not advocate the use of Biniiodide of Mercury. The technique was to inject $\frac{1}{2}$ cc. at first to see if any

reaction occurred, if not, then 2-3 cc. thereafter. He states there were no immediate effects if vein was big but if small then they would get swelling and mild burning sensation. The later effects constituted aching and tenderness over injected area. Itching and discoloration would also manifest itself. The constitutional reactions were rare and if it did show up there would be a tinnitis due to quinine, fainting, giddiness and aching in affected limb which is the most common complaint. Faxon (20)

de Takat's (105), McPheeters (78), by pathological slides have proven their original theory of sclerosing solutions. Any hypertonic solution injures the endothelium by dehydration while the chemical substance acts upon the cell directly. When the solution is injected into the lumen of the vein there is an inflammatory reaction upon the vein wall which pours out an exudate which is the ground work whereby the organized clot (thrombus) and the vessel walls are adherent. This thrombus, according to Aschoff (McPheeters 76) is formed by stagnation of the blood stream, destruction of lumen wall, reaction on blood constituents. Solutions should be controlled by strengthening or weakening the solution and by localization as the case may be. The chemical

irritation (Silverman 104) of the intima with a destruction of endothelium and a round cell infiltration tends to cause a fibroblastic proliferation. This will lead to a gradual sclerosis of media and adventitia even including the perivenous connective tissue. The blood clot forms only after endothelial lining of intima is destroyed and layers of fibrin are deposited on wall of vein.

de Takats (105) thought that most of the failures at this time were due to improper technique and failure to adhere to systematic follow up treatment of recurrences.

McPheeters (77) suggested the use of the occluder as a possible means of improving the technique of that day. The apparatus was of nickel composition that could be moulded in any form whatsoever. It was in the shape of a ring with a diameter of 3-4 inches. In this way a certain area could be excluded from the rest of the circulation and the vein made visible. Another advantage would be that the sclerosing solution is kept in that area so as to act upon the intimal lining.

In 1919 Rogers and Merkegge had worked out a solution called Sodium Morrhuate for the treatment of

leprosy and later Higgins and Kittel (33) used it for the first time for varicosities. They found it less toxic and reported very good success.

Ochsner (37) used Sodium Chloride 20-25%. Also Sodium Salicylate 20,25,30,40%. He found them good sclerosing agents but thought the injection of these substances caused severe pain and if made perivenous a slough. Dextrose was added to Sodium Chloride to decrease the irritation and toxicity and improved solution. (Kern and Angle 47, Ferguson 23, Logefeil 58,59, de Takats and Quint 105). Those investigators maintain that in this way the toxicity is decreased without interfering with the efficiency of the sclerosing properties. An added disadvantage of salicylate solution is that frequently patients have a idiosyncrasy to salicylates and the injection which must be made intravenously may produce a severe systemic reaction. The relative value of hypertonic Sodium Salicylate and Sodium Chloride solution are shown by the experimental investigation of Ochsner and Garside (84). It was found that normal veins injected from $\frac{1}{2}$ hour to 8 weeks previously showed an average destruction of endothelium and average thrombus incidence of 76% and 47% respectively,

When injection is made with Sodium Salicylate (40). Whereas when injected with Sodium Chloride (25%), the respective values were 66% and 23%. The clinical efficacy of these solutions is shown by the investigation of Lewis (57) who found that the injection of varicosities with 30-40 solution of Sodium Salicylate produced a thrombosis in 95.5% of instances whereas 20% Sodium Chloride produced thrombosis in 90%. This difference between the experimental and clinical results is undoubtedly due to the fact that in varicosities not only is the vein wall altered but the flow of blood is slowed, both of which favor thrombosis. Because of the disadvantages and because equally as good or better results (Ochsner and Mahorner 83,86,87) can be obtained by other sclerosing agents, the use of hypertonic Sodium Chloride and Sodium Salicylates have been used to a lesser extent.

Dutton (18) suggested that most reactions were due to poor technique and devised a needle whereby the principle of the trocar was used, but a needle was in place of stylet. The instrument consisted of two needles, the point of the outer one being ground off and carefully bevelled. The inner needle fits snugly into the outer one, its short medium bevelled point just protruding at end. The needles are inserted

into the vein and the blood is drawn by syringe, then inner needle is removed and another syringe is attached to the needle. Blood is again withdrawn and needle is then boldly pushed up vein because of blunt point. This is quite an improvement on technique and the medium bevelled needle does not allow the instrument to go through other wall. It allows the operator more confidence.

Ochsner and Garside (83,84,86) finished up the experimental work on evaluating the different sclerosing solutions.

Dextrose 50% and invertose 50-75% solution (Ochsner 87) have been popular in the treatment of varicose veins because they produce few local and systemic reactions as the endothelial injury is much less marked than that produced by hypertonic salt solutions, it is necessary that larger quantities be used. The sugar solutions in these high concentrations are viscid and are difficult to inject, requiring the use of a large needle. This causes a large opening in the vein through which the injected substance may leak into the perivascular tissue. The intravenous injection of Hypertonic sugar solution, however, produces little or no discomfort and is

therefore much more welcomed to the patient. Also if an accidental perivenous injection is made, the danger of necrosis and subsequent sloughing is less.

Sodium Morrhuate produced thrombosis in 71.4% cases.

Sodium Gynocardate 5% produced thrombosis in 50% cases.

Sodium Gynocardate 3% produced thrombosis in 50% cases.

Sodium Gynocardate 2% produced thrombosis in 44% cases.

Sodium Hydnocarpate 5% produced thrombosis in 37.5% cases.

Sodium Morrhuate over 5% produced thrombosis in 33% cases.

Sodium Hydnocarpate 3% produced thrombosis in 33% cases.

Sodium Morrhuate 10% produced thrombosis in 19% cases.

Sodium Hydnocarpate 2% produced thrombosis in 12.5% cases.

According to the results the grade and incidence of endothelial destruction closely parallel the incidence of thrombosis.

Faxon (20) used 13% Quinine Hydrochloride and Urethane 6% and in 314 cases found these results:

Group I Good result no further treatment 27.3%

Group II Fair results further treatment
not urgent 53.3%

Group III Poor results further treatment
urgent 2.0%

Group IV Poor results Saphenous vein ligation
17.4%.

Ochsner and Garside (86,87) in their experiment investigation found that of the sugar solution, invertose 75% combined with saccharose 5% was the most efficient sclerosing agent. It produced an average endothelial destruction and average incidence of thrombosis of 55% and 23%. Various combinations of hypertonic sugar solutions have been used. Kern and Angle (47), Ferguson and Loefford (23), Logefeil (59) de Takats (106) and Quint, Lewis (57) have advocated combinations of Sodium Chloride and dextrose. Lewis (57) reports that in 92.1%, thrombosis occurred in Varicosities injected with dextrose 50% and Sodium Chloride 30%. Combinations of invert sugar and Sodium Salicylate have been suggested by Logefeil. Ochsner and Garside (87) found that injection of invertose 75% and Sodium Salicylate 20% into veins of normal animals resulted in an average endothelial

destruction and incidence of thrombi.

Since the recommendation by Genevrier (25) in 1921 that Quinine Hydrochloride and urethane solution be used in the treatment of varicose veins, this substance has become quite popular. The sclerosing property of Quinine Hydrochloride and urethane are exemplified by experimental researches of Ochsner and Garside, and clinical investigation of few. Ochsner and Garside (83,86,87) found that Quinine Hydrochloride and Urethane was second only to Sodium Salicylate (40%) in its destructive action on venous endothelium and its ability to produce thrombi. They found that in 66% of their observations there was endothelial destruction and in 28% thrombi was produced. Lewis (57) found that in 94.8% of varicosities injected with Quinine Hydrochloride and Urethane a thrombus developed. The other apparent advantages of Quinine Hydrochloride and Urethane are, just small amounts needed for sclerosing, the solution is not viscid and therefore the danger of perivenous infiltration is slight and if the perivenous tissue becomes involved the sloughing and necrosis is less. This solution has to be used cautiously because the patient might have an idiosyncrasy toward the Quinine.

The most recent additions to the group of sclerosing agents is Sodium Morrhuate which is the Sodium Salt of a fatty acid derived from cod liver oil (Rogers 96). He originally used Sodium Morrhuate in India in the treatment of leprosy and tuberculosis and observed if concentrations higher than 3% were used, a localized mild inflammation of the vein occurred, which resulted in the occlusion of the vessel by an organized thrombus. He recommended the use of 5% Sodium Morrhuate in treatment of varicose veins. Excellent results have been reported by Rogers (97), Winchester (114), Higgins and Kittel (33), McPheeters (73), Tunich and Nash (112), Ochsner and Mahorner (63,64,87), Shelley (100), Dale (14) reports that it has proved to be not only the most efficient sclerosing agent but also one which has as few disadvantages as any of the other substances. The relative efficiency of Sodium Morrhuate 5% is demonstrated by Ochsner and Mahorner (64,88) in which it was found that the average destruction of endothelium 82.5% and the average percentage of thrombi (82.5%) were considerably greater than those obtained following the injection of any other sclerosing agents. Sodium Morrhuate is not

toxic (Rogers 97, Higgins and Kittel 33) produces practically no discomfort and causes little or no reaction when a perivenous injection is made.

Ochsner and Mahorner (88) have shown that even though clinical manifestations following perivenous injections of Sodium Morrhuate solution are slight, such injections cause a coagulation necrosis of the cells which can be demonstrated microscopically. In fact, a similar change occurs frequently in the cells of the media when an intravenous injection is made. It is possible that this extensive change is responsible for the high incidence of thrombosis in varicosities injected with Sodium Morrhuate solution.

Even though the enthusiasm for Sodium Morrhuate is great it does not go without saying that one has to be careful because of reported side actions and fatalities (Hawkes and Borsher 31). Barber (5) reports failings and recurrences. But from his report his treatment is haphazard and does not follow up his patients very well.

Works on hypothesis that sclerosing solution should be as near like body humors as possible, compares four new substances. Biegeleisen (7) compares the more recent sclerosing agents. Morrhuate-Quinine A consists of Sodium Morrhuate 5%, Quinine

Hydrochloride 2% and Benzyl Alcohol. Morrhuate and Quinine B contains Sodium Morrhuate 10%, Quinine Hydrochloride 2% and Benzyl Alcohol 2%. The above two fills a useful niche midway between the weaker and stronger members of the endothelial irritants. Another sclerosing agent that he brings out is Oleate Quinine. It consists of Potassium Oleate 5%, 2% of Quinine Hydrochloride, 2% benzyl alcohol in H₂O. He considers this solution very good except that he gets some very marked pigmentation. The sclerosing efficiency of this solution is slightly less than Quinine Hydrochloride and Urethane.

Sylvasol (known as Sodium Psylliate) is the Sodium salt of fatty acid derived from the psyllium seed. Less irritation than Sodium Morrhuate and gives no dermatosis like Sodium Morrhuate.

Monoethanolamine Oleate: organic base combined with oleic acid. This is a definite chemical compound. Uses Quinine Hydrochloride and Urethane and Monolate used together in 5 cc. proves to be better than Sodium Morrhuate.

According to Glasser (28) Monolate is a definite compound and is stable with no side reactions. With Sodium Morrhuate one is apt to get allergic reactions.

(Kilbourne 51,52, Hawkes and Borsher 31, Dale 14, Holland 34) Besides there is a decided lack of standardization of this compound which makes it less efficient in its use.

Sodium Ricinoleate which was used and discovered by Froehlich and Hendrickson (24) is receiving good support. Pratt (93) uses $3\frac{1}{2}\%$ solution and goes by the formula of $\frac{\text{diameter of Saphenous Vein} \times \text{length of Saphenous Vein}}{3} = \text{amount of } 3\frac{1}{2}\% \text{ Sodium Ricinoleate}$. This writer has improved arterial flow by clearing up arterial occlusion and also one with marked arterial sclerosis. Postlethwaite has good results with Koleate and Potassium Oleate.

In undertaking the treatment of varicose veins one must take into consideration the amount of venous involvement, a history of or the presence of phlebitis in the deep or superficial veins, history of or presence of thrombosis in the deep or superficial veins, acute or chronic ulceration and edema of lower extremities.

It will be found that the modern method of treatment may be divided into:

1. Type that needs injection only
2. Type that needs ligation alone

3. Type that needs combination of both
4. Some individuals still use the stripping method in combination with the other methods.(Horgan 36)

McPheeters (73) takes in the size of the varicosities and determines the type, amount and course of the injections. Of course previous to this he has gone very well over the patient's history to find if any contraindications present themselves. If the size of the varicosities are small and just a few in number they may be injected at one sitting. The general technic used by most individuals (Mahorner and Ochsner 63, McPheeters 73, Rakov 94, Clarkson 11) for injection at one sitting. (Some Ak1 3, Dawkins 15, McPheeters 73, like to inject into a full vein,) depending upon whether the varices slightly larger than size one. Varices noticed and located. Spot marked for injection. The patient is allowed to lie on table and the area cleaned aseptically. Then patient swings leg over side of table if varices are small. If larger at level of table. McPheeters and Anderson (73) like to place three tourniquets, one at heel, one below lowest varix and the other at

groin; while, in the University of Nebraska Surgery dispensary for size one-half to one they did not use any tourniquet. Raise leg. Whatever solution is used 2-3-5% Sodium Monolate (McPheeters 73), Postlethwaite (92) use Sodium Ricinoleate, $3\frac{1}{2}$ Sodium Monolate used by Biegelseisen 6,7) extreme care must be taken to be sure needle is in lumen of vein, (McPheeters 73, Rakov 94, Dutton 18) by aspirating blood into syringe. As injection is being made then leg is lowered to level of table so as to hold solution in area. As injection is finished the syringe and needle are taken out and compress placed over point of injection so that no solution will come back out and get into subcutaneous tissue. Patient can then go about his work. It is important to follow up cases to see if repeated injections are the type that needs more than one sitting for injection--bandage up to knee and lower at each time of injection, (Mahorner and Ochsner 63,88, MCPheeters 73), are needed. The greatest problem in connection with the injection treatment is not that of curing varicose veins but rather that of preventing recurrences after treatment. No matter how large or numerous the varicosities they

can all be made to disappear, with a concomitant subsiding of symptoms and with little or no disability during course of treatment. (Rakov 94, Cooper 9). The problem of recurrence, however, presents certain difficulties. By the term recurrence (McPheeters and Anderson 73, de Takats 105,107, Rakov 94, Mahorner and Ochsner 63, MCPheeters and Lunblad 78) it is meant that those varicosities which have been injected and which at a later date reappear; secondly those varicosities, which were too small to be injected at the time of treatment and which later attained greater size (de Takats 105, Siegler 103, Dawkins 15, MCPheeters and Anderson 73) and third those varicosities that developed from normal veins, following injection of the varicosities present at the time of treatment.

To prevent recurrences (de Takats 105, MCPheeters and Lunblad 78, Clarkson 11, Kitchen 53) requires a careful, diligent search for all varicosities present. There are some of these that are not large and are so imbedded in fat that they are difficult to palpate (Rakov 94). By patience and diligence and experience one can by time detect and become adept at picking up the smaller

varicosities. Another thing that helps in the prevention of recurrences is the checkup of the patient ever so often and injection can be made while still small. In cases in which there is marked swelling of the legs, the preliminary use of an elastic support will often reduce the swelling to the extent that varicosities that could not be detected before will now become palpable. Where there are extremely large varicosities, elastic bandages should be worn during the course of treatment.

By the use of the tests, as given previously, one can determine the type of varicosities and the competency of valves, what type of surgical treatment is needed, types of ligations, high, low, ligation of small or external saphenous vein.

1. High saphenous ligation: When the saphenous vein is incompetent the veins below are subjected to increased pressure (McPheeters and Rice 74, Bernstein 116, Trendelenburg) by blood above varicosities, from the large intra-abdominal veins and higher. Therefore, any increased pressure is directly transferred down the saphenous veins into the varicosities, or if injection has been

done previously, against the thrombus that will be present. If the valve at the sapheno-femoral juncture is incompetent, some of the blood that is being pumped upward in the femoral vein will regurgitate or spill back through this leaky valve down into the saphenous veins. In a relatively short time the effect of this pressure is to recanalize the thrombus, forming a channel that eventually dilates and becomes varicosed. Isaak (41)

The technic of the operation is to have facilities of an operating room with patient on their back, (Rakov 94) or standing (Kitchen 53). The surgical field is made as sterile as possible. The field around is kept the same way. The operation is done under local anesthesia. A transverse incision (McPheeters and Anderson 73) or a longitudinal incision (Mahorner and Ochsner 63) is made about one inch below Poupart's ligament. (McPheeters and Anderson 73). The saphenous vein is exposed and the proximal end is ligated above its three tributaries (superficial circumflex iliac, superficial epigastric and superficial external pudendal. This, or ligation of these with section of saphenous removed. (Edwards 19, Kitchen 53).

The distal end has a ligature placed around it but not tied. (McPheeters and Anderson 73) The canula or syringe with sclerosing solution is then introduced into distal segment and the ligature tied firmly about it. The leg being treated is then elevated high to 45° for one minute. This allows the blood to drain out of the saphenous through the communicating veins. The table is then tilted into reverse Trendelenburg and the leg is lowered to the table. The sclerosing solution is injected as the leg is lowered. With the great saphenous vein empty the force of gravity will often times carry the injected solution down to the knee and at times to the ankle. The deeper layers of the wound are closed with two or three interrupted chromic sutures and the skin with the dermal. With careful hemostasis no drain is needed.

When varicosities are large and extended it is good therapy to strap the lower leg with wide bandages (Ace) previous to operation so as to limit the sclerosing solution.

If the whole saphenous vein is sclerosed the patient might get some reactions and so after the

operation patient is allowed to move around at will. When pain starts the patient should be given some light sedatives and warm Magnesium Sulfate packs on whole leg (Kitchen 53) below site of operation. This procedure frequently scleroses all varices distal to the site of ligation if they are not extremely large. If this does not occur injection treatment of any few remaining varices may be carried out in the usual manner.

II Low Saphenous ligation: There are three sets of perforating veins in the thigh, that is, veins connecting the deep or femoral veins with the superficially situated saphenous vein. If the valves in these veins are incompetent, allowing a reflux or spilling of blood from the femoral into the saphenous, it becomes necessary to perform ligation at a low level in addition to a high ligation at the saphenous-femoral junction. By tests previously mentioned adequacy of valves can be determined. Just as recurrence is inevitable if a high ligation is not performed in the presence of an incompetent saphenous vein, so also is it inevitable if a low ligation is not performed

in the presence of incompetent perforating veins in the thigh.

III. Ligation of small or External Saphenous Vein. When, as indicated by tests, the valves of the small saphenous vein are incompetent, it becomes necessary to ligate this vein in the popliteal region just before it joins the popliteal vein. A sclerosing agent is injected distally as in high saphenous ligation.

SUMMARY

1. When no contraindications present varicosities may be injected up to size 1, if few in number, at one sitting.
2. Sodium Morrhuate, Searle 5%. Sodium Ricinoleate 3 $\frac{1}{2}$ %, Sodium Linsolate 1/2-10%, Monoleate 1/2-10% are the best solutions to use.
3. Never over treat with any solution. Rather reinject.
4. Ligation. High or low with injection have proven very successful and is the modern up to date treatment.
5. Reactions have been reported. Treatment must be maintained, not stopped, and then continued after an interval of time, unless indications are such that it is advisable to stop treatment.

COMPLICATION

The complications of any treatment of varicosities presents several distinct problems.

Pulmonary Embolism. In this instance McCausland 70,71, McPheeters and Anderson 73) reports a mortality of 0.00754 in his series review of the literature for the injection treatment and 0.53% following operative treatment. (McPheeters and Anderson 73) states that the possibility of embolus is very rare due to two reasons. One is that the sclerosing solution when placed in the veins becomes organized and does not break up. The second reason is that because of the direction of the venous flow in varicose veins, demonstrated by lipiodol injection (McPheeters 75) emboli from segments of the thrombus breaking loose and spreading into general circulation are very rare. Logically this can be seen because of the emboli being forced down into the smaller varices and veins and becoming lodged. When an acute infectious thrombophlebitis develops associated with the injected treatment there is a possibility of getting an embolus (McPheeters 75, McCausland 70,71, Taylor 108,

Silverman 104).

McCausland (70) divides phlebitis into

(1) Infectious, which may be due to poor technique or to resting infections (de Takats 105) present in the vein. The treatment would be supportive, combined with as much heat as possible. Of course, the best treatment is prophylaxis. If an acute infectious thrombophlebitis is present then bed rest is immediately instituted.

(2) Chemical. McCausland 70, McPheeters 75, deem this not serious.

Sloughs. This condition may be readily attributed to poor technique, (McPheeters 75, de Takats 105-107, McCausland 70,71, Ochsner 85) both in the injection treatment and operative treatment

The most serious complication of varicose veins is varicose ulcers. The cause of varicose ulcer is the stagnation of blood within the capillaries and the loading of the tissues spaces outside of the capillaries with a poorly oxygenated serum (Tunich 111). One might say the tissues are waterlogged (Rakov 94). The skin is poorly nourished, its resistance is lowered and an otherwise trivial injury produces an ulcer. If the

ulcer becomes infected, there occurs a varying degree of cellulitis. As a result of recurrences, scar tissue is formed that brings about an actual blocking of the lymphatics and a stasis of lymph.

If the patient is put to bed and the leg elevated the varicose ulcer in most cases will heal without further treatment. (Rakov 94, McPheeters 95). However, as soon as the patient walks, edema develops and the ulcer recurs. The ideal treatment is one which would not require bed rest or hospitalization and which attacks the cause of the condition, in addition to the secondary complication. All this is accomplished by the injection-ligation, together with elastic compression. Tunich (110,111), Akl (3), Barber (5).

It goes without saying that if varicose veins are present they must be treated, for they are the primary cause of the ulcer and recurrence is inevitable if the treatment of these varicosities is neglected. One method will not serve for all cases. (Rakov 94, Navat 82, Akl 3, Brown 8, Ines 40, Isaak 41).

(1) The ulcer complicated by little or no edema

or induration. For this type the injection-ligation treatment alone will produce a cure.

(2) The ulcer complicated by soft, pitting edema but little or no induration. For this type, in addition to injection ligation, elastic supports are used to relieve the already existing edema. (3) The ulcer complicated by induration localized in the vicinity of the ulcer. This type responds well to injections plus the use of McPheeters (79) so-called sponge heart. (4) The fourth type is the ulcer complicated by hard non-pitting, brownish induration of an extensive part of the leg or even of the whole leg. In this type of case, elasto-plast or zinc gelatine bandages are frequently used because the hard induration requires (Akl 3) tight compression in order to occlude the dilated veins, although the sponge heart and elastic bandage are sufficient. (McPheeters 79, Dawkins 15) states arterial disease and diabetes are contra-indicated with the above treatment. It is contended that pressure on leg tends to start gangrene. There is no question but that the deep system of veins is incompetent in this condition and its incompetency accounts for the tremendous

swelling. Iontophoresis, (McGarack and Samworth 72) has been highly recommended but Rakov (94) disagrees.

Individuals having undue pain with varicose ulcers the use of Vitamin B₁ (Thiamin and Betaxin) has some value (Ochsner and Smith 89).

Varicose Eczema - A frequent complication of varicose veins is eczema of the skin with or without pruritus. This condition responds very well to treatment of the varicose veins plus bland ointments and x-ray therapy.

SUMMARY

1. The lack of knowledge in regard to asepsis retarded the first treatment of varicose veins.
2. The invention of the syringe was a good step forward.
3. Many solutions have been tried. The first group was more or less blood coagulants and the incident of thrombus formation with emboli appearing was common. Reactions were greater.
4. The solution of Sodium Chloride with the sugars was the first effective sclerosing solution.
5. The combining of the ideas that the surgeons and medical men advocated was a great contribution.
6. The improvement of technique and knowledge of the pathology of the venous walls stimulated the perfection in treatment.
7. The discovery of Sodium Morrhuate, Sodium Ricinoleate, Monolate and the value in the high ligation proved to be the needed niche to make varicose veins amenable to satisfactory treatment.

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