THE SWOLLEN LEG OF DISTURBED VENOUS OUTFLOW

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Thrombophlebitis of the deep veins is so generally accepted as *the* cause of chronic oedema of the leg in those cases in which venous obstruction is evident, that the term of 'post-phlebitic syndrome' has become synonymous with the swollen leg of venous origin. Even those patients who develop a swollen leg without any history suggesting deep-vein thrombosis, are assumed to have had 'silent' thrombosis of the deep veins. This narrow outlook on the pathology of such a prevalent and morbid condition has led to a number of important fallacious concepts about its pathophysiology and treatment.

In actual fact, it is very doubtful whether obstruction of the deep veins of the leg by thrombus is *ever* the cause of the chronic swollen leg. Although it is quite clear that acute thrombophlebitis, especially when in the ilio-femoral veins and when massive, can, and usually does, cause swelling of the leg, it is equally clear that this acute swelling clears up in the great majority of cases, to be followed, in about 75% of cases, by a slow progressive swelling of the leg, which appears a few years after the acute episode.

The mere finding of thrombi in the deep veins at post-mortem examination is no proof that they are the cause of the swelling of the leg. In 2 out of every 3 cases in which occluding thrombi are found at postmortem, there are no signs of swelling or other abnormalities caused by these thrombi.[1] Similarly, McLachlin and Paterson[2] found clinical signs in only 5 cases out of 34 in whom they found thrombi at autopsy and in all 5 the histological features indicated an acute thrombophlebitis.

In phlebographic studies of 62 cases with oedema and ulceration of the leg, Dow[3] found that in no case were the deep veins actually blocked. It is not the thrombophlebitis *per se* which is the cause of the swollen leg, but the destruction of the venous valves by the process of organization and recanalization of the thrombus.[4] Depending on the length of vein which is involved, the valvular incompetence results in a dynamic obstruction to the venous outflow and this is the important factor in the pathogenesis of the swollen leg following thrombophlebitis. The realization of this basic fault in the venous outflow opens the way for a more aggressive and positive therapeutic approach to these patients.

The proportion of patients with swollen legs of venous origin which give a history of past deep-vein thrombosis, varies considerably from 80%[5] to 30%.[6] Whatever the incidence may be, the fact remains that there are a considerable number of cases in which no history suggesting deep-vein thrombosis exists. Merely to assume that they have had 'silent' thrombosis is to my mind not only unwarranted but also illogical. Even a 'silent' thrombosis must involve a considerable length of vein in order to produce an ultimate incompetency, sufficient in extent to cause chronic oedema of the leg. Thrombosis of such an extent must make its presence known during the acute phase. These cases present with exactly the same picture of incompetence of the deep veins but in their case some other factor(s) have caused the incompetence.

These cases of 'idiopathic' incompetent deep veins, which are exceedingly common according to Morrell,[7] are relegated to the obscurity of ignorance because the possibility of the repair of incompetent veins has never been seriously considered. This lack of enterprise in this age of cardiac and arterial surgery is truly amazing and can only be ascribed to the concept that thrombophlebitis is the only cause of the venous incompetence and that its progressive course is inevitable and unchangeable. This defeatist attitude has had a stifling effect on research, resulting in a paucity of precise physiological and pathological data on venous disease.

In order to evaluate the role of disturbed haemodynamics of the venous outflow on the genesis of the various pathological states giving rise to a chronic swollen leg, a short review of the known physiological data and of the anatomy of the veins constituting the outflow tract from the leg, is necessary. With the aid of illustrative cases, the salient features of disturbed venous outflow from the leg will be discussed, with special emphasis on the aetiology of the idiopathic group and on the surgical treatment of both the early and the late cases.

ANATOMY OF THE OUTFLOW TRACT OF THE LEG

The main outflow tract of the leg consists of the ilio-femoral veins, which join to form the inferior vena cava. This tract begins in the popliteal fossa with the junction of the posterior and the anterior tibial veins and it ends at the tricuspid valve. The femoral and popliteal channels are variable in their make-up. Instead of one large ascending vein it is not uncommon to find reduplication of venous channels. Throughout its course the femoral vein receives muscular tributaries, the most important of these being the profunda femoris vein which joins its posterior surface in Hunter's canal, 2 inches below the inguinal ligament. Slightly above this level the internal saphenous vein joins its anterior surface. The iliac veins may be compressed by either the inguinal ligament or the internal iliac artery, where it crosses over the external iliac vein. The former is demonstrated by venography when the hip is flexed and the latter is suggested by the frequency with which thrombophlebitis of the ilio-femoral vein starts at precisely this point.[8] The left iliac vein is longer and follows a more oblique course than the right iliac vein. It therefore makes a sharp bend medially and forwards in order to climb over the sacral promontory to reach the inferior vena cava on the right side of the vertebral column. This difference between the left and right iliac veins is important in view of the prevalence of ilio-femoral thrombosis on the left side.

Venous Circles

Apart from the main outflow tract described, there are important venous circles which may effect not only an important collateral pathway but also a potential route for the return of retrograde flow down the ilio-femoral veins. These venous circles have been described by Edwards and Robuck[9] after their dissections of 61 extremities. They comprise a deep and a superficial system:

1. The deep venous circles are formed via the medial and lateral circumflex femoral veins. The most important one is formed by the junction of the medial circumflex femoral vein with the obturator and inferior gluteal veins. The valves in these venous circles are arranged in such a way that, under normal circumstances, centripetal flow through them is not possible. Centripetal flow may occur when these valves are rendered incompetent as a result of dilation of the veins or when valves are absent in the medial circumflex iliac vein.

2. Superficial venous circles are formed from the femoral vein via branches of the internal saphenous vein to superficial veins of the body and the axilla.

Venous valves occur at intervals throughout the deep veins of the leg. The popliteal vein has from 1 to 4 valves; the superficial femoral vein has 1 - 5; the common femoral and the external iliac veins each usually have only 1, while no functioning valves are present in the common iliac vein or the inferior vena cava. One of the most constant is the valve in the common femoral vein which is situated at the level of the inguinal ligament. The venous valve is a pouch-like fold of the tunica intima. These valves are usually bicuspid, though occasionally tricuspid. The free border of the valve is lunate. On the heart side of the valve the vein wall is usually thinned, allowing a certain amount of dilation. This gives a somewhat nodular appearance to the vein when it is full of blood. There is a tendency for the number of valves to diminish with advancing age. The human embryo demonstrates the presence of venous valves at the stage of 4 months' development. Congenital areas of constriction have been described in the iliac veins.

HAEMODYNAMICS OF THE VENOUS OUTFLOW

The Capacity and Distensibility of Leg Veins

Dilation of the veins of the leg may render their valves incompetent and, because of the importance of incompetent veins in the genesis of the swollen leg, a knowledge of the factors influencing the capacity and distensibility of these veins is essential. The relevant aspects of our present knowledge of these basic physiological factors have recently been reviewed by Shepherd.[10] Various experimental studies have shown that the veins can reduce their capacity in response to local, humoral and nervous stimuli, which also increase the tone of the arterioles; however, in the comfortable warm subject, the veins, unlike the arterioles, are normally at their maximal distensibility, and the amount of blood they contain is determined by the transmural pressure acting on their walls, and not by the reactivity of the walls. The calibre of a bloodvessel depends upon the balance between the transmural pressure tending to dilate it, and the tension in the wall tending to constrict it. From this it follows that, in the comfortable warm subject, the pressure and distension of the veins will depend on the degree of arteriolar dilation by which the arterial pressure is more directly transmitted to the veins. Thus, factors like exercise which causes arteriolar dilatation will cause distension and increased pressure in the veins.

Lowering a leg from the horizontal to the passive dependent position, causes an increase in bloodflow to both the skin and the muscles as shown by an increase of heat elimination of the toes[11] and by a raised oxygen saturation of the venous blood.[12]

The Nature of Bloodflow

Liquids flowing through conduits such as the vascular system may behave basically in two fashions, viz. laminar flow or turbulent flow.

1. Laminar flow. In laminar or streamlined flow the liquid may be visualized as moving in very thin layers parallel to the wall of the containing vessel.

2. Turbulent flow. In this type of flow the fluid particles move in irregular paths at different angles to the main direction of flow. The higher the flow velocity and the bigger the radius of the bloodvessel, the more likely will turbulent flow be. Turbulence can also be influenced or created by sudden changes in the size or direction of the bloodvessel and also by irregularities of the inner surface of the vessel. In this way turbulent flow may arise on the proximal side of the venous valves where small fibrinous thrombi can be precipitated from the blood as the result of the localized area of turbulence, this being analogous to the familiar process of defibrinating blood by agitating it with some stirring device.

The Role of Arteriovenous Shunts

In many tissues the arterioles and venules are directly connected by channels with thick muscular walls, richly supplied by vasomotor nerves. Through these arteriovenous anastomoses the blood can be short-circuited and the capillary bed bypassed. They are particularly abundant in the skin where they assist in temperature regulation. That similar anastomoses exist in skeletal muscle is indicated by the work of Walder,[13] who found that intravenous administration of epinephrine did not affect the clearance rate of ^{24}Na from human skeletal muscle, although the total flow through the muscle had about doubled.

That these arteriovenous shunts may become more extensive in pathological conditions and play an important role in the causation of venous distension and valvular incompetence is indicated by the following observations as summarized by King[14] on the early stages of varicose veins: the centripetal pulsations of the veins, the high oxygen saturation of the venous blood, the dilation just *distal* to the valve, and the localization of the condition with normal vessels to be seen proximal to the affected area. The prevalence of varicose veins during the early stage of pregnancy and the aggravation of venous distension in the premenstrual phase suggest that one of the stimuli causing these shunts to open up may be hormonal.

Pressure Changes in the Veins of the Leg

The pressure changes in the superficial veins at the ankle were studied by Pollack *et al.*[15] In normal subjects the pressure was 59 mm.Hg when they were sitting, 90 mm.Hg when standing, and it fell to 18 mm.Hg when they were walking.

Walker and Longland[16] found that patients with thrombosis or incompetent femoral veins had little or no fall in pressure on exercise. They confirmed the report of others that the venous pressure in the foot at rest was equal to that exerted by a column of blood as high as the patient's heart above his foot. This latter finding shows that, with the subject standing still, the venous valves become incompetent, with the result that an unbroken column of blood exists from the foot to the heart. The fall of pressure in the superficial veins on exercise demonstrates the efficiency of the muscle pump in drawing the blood from the superficial veins into the deep system.

The pressure changes on exercise have also been studied in the deep veins of the thigh and popliteal region. In both normal subjects[17] and those with varicose veins or postphlebitic syndrome[18] no change in the main pressure was found with exercise. A constant pressure in the deep veins of the thigh is quite compatible with normal function, for it has been shown by plethysmographic methods that the muscle pump of the lower leg can work efficiently against a pressure as high as 90 mm.Hg.[19]

Pressure studies done recently[20] in the femoral veins of patients undergoing intra-abdominal operations have shown marked increases in pressure during the normal amount of manipulation and traction on the gallbladder during cholecystectomy.

The Venous Circulation of the Leg

When the veins of the leg are healthy the blood flows from the leg towards the heart. In the erect position this flow has to overcome the force of gravity which is considerable when one realizes that for many hours the blood has to travel upwards through a vertical channel some 1½ metres in length. Elevation of the leg towards the horizontal position causes a decrease in venous pressure and an increase in the velocity of the venous outflow. The driving force causing the return of blood to the heart has been determined by innumerable physiological experiments. It consists of the following components:

1. The muscle pump. The contractions of the muscles of the calf in conjunction with competent valves cause the blood to ascend the leg by the process of 'relay pumping'. The power of this muscle pump and the velocity of the bloodflow in the iliac veins caused by it, are dramatically visualized by cinevenography. During the diastole of the calf muscles blood from the superficial veins flows into the emptied deep veins via competent communicating veins. Muscle tone and involuntary contractions in the vertical statis posture are enough to keep the veins fairly empty. In spite of this, in a healthy man it has been shown that half a litre of blood may be added to the legs after standing for an hour. This addition will disappear on walking or elevation of the leg.[21]

2. Transmitted arterial pressure — vis a tergo. The pressure transmitted through the capillary bed is too small to be of any significance; the role which the arteriovenous shunts may play

could, however, be considerable. To what extent this mechanism is a factor in the ascent of the blood under normal circumstances is unknown.

3. Cardio-thoracic 'aspiration'. Although the speed of venous flow is generally increased by the movements of respiration, evidence has accumulated that, while quiet breathing has no effect on the venous pressure in the leg, deep respiration impedes the flow from the legs.

This concept of the normal haemodynamics of the outflow of blood from the legs is probably over-simplified. For one thing, it is quite clear from cine-venographic studies and from pressure studies that retrograde flow down the ilio-femoral veins is a frequent occurrence during any straining (e.g. Valsalva manoeuvre, coughing, defaecation, etc.). It is likely that retrograde flow during such short-lived periods of straining and in the presence of competent valves, is confined to the iliac and the common femoral veins, and that the blood is returned along the venous circles demonstrated by Edwards. In the erect position retrograde flow is probably more extensive because of the distension of the veins and the resultant temporary valvular incompetence. This retrograde flow in normal subjects in the erect position has been clearly demonstrated with cine-venography.[22]

Morrell[23] points out various aspects of pathology of the veins of the leg which cannot be explained in the light of the current views of the haemodynamics of the circulation in the limbs. He postulates the existence of a series of U-shaped loops including arteriovenous shunts, which exhibit countercurrent activity, similar in nature to the current hypothesis for urinary concentrating mechanisms. In this way he visualizes an integrated chemical haemodynamic factor or set of factors which may be altered by disease, stress or trauma.

DISTURBED VENOUS OUTFLOW AND THE SWOLLEN LEG

1. Obstruction of the Venous Outflow

(a) *Thrombophlebitis*

The salient features of acute ilio-femoral thrombophlebitis have been discussed previously.[8] Since that publication, 8 additional cases have come under my care, making a total of 11. All of these were treated by venous thrombectomy and they have confirmed my previous conclusions and emphasized the following aspects:

(i) *Thrombectomy,* if complete, will prevent the resulting incompetence of the deep veins following valvular destruction during recanalization of the thrombus, and is therefore the treatment of choice in all cases of acute ilio-femoral thrombosis. The argument that a lot of these operations will be performed unnecessarily because a great majority of these cases will clear up on conservative treatment, is no longer to be countenanced. It is quite clear that, in spite of the apparent success of conservative treatment, the great majority of such treated cases develop signs of deep venous incompetence with its progressive course towards eventual chronic swelling, with or without ulceration, within 2 or 3 years.

(ii) *Ilio-femoral thrombophlebitis* should be suspected in all cases with pain and swelling of the leg in whom there is but the slightest sign of swelling of the thigh, which can frequently only be detected by measurement and tenderness over the femoral vein in Scarpa's triangle.

(iii) *The left leg* was the one affected in all these patients. In view of this prevalence of thrombosis on the left side, the particular anatomy of the left iliac veins which may cause altered venous outflow from the left leg, assumes greater importance as the likely initiating cause of ilio-femoral thrombophlebitis.

(iv) *The acute inflammatory reaction* in the wall of the affected segment of vein is not the result of bacterial infection. The clot and the vein wall yielded no growth on both aerobic and anaerobic culture in three of these cases.

(v) *Associated spasm* of the femoral artery occurs frequently and clears up dramatically on removal of the venous thrombus.

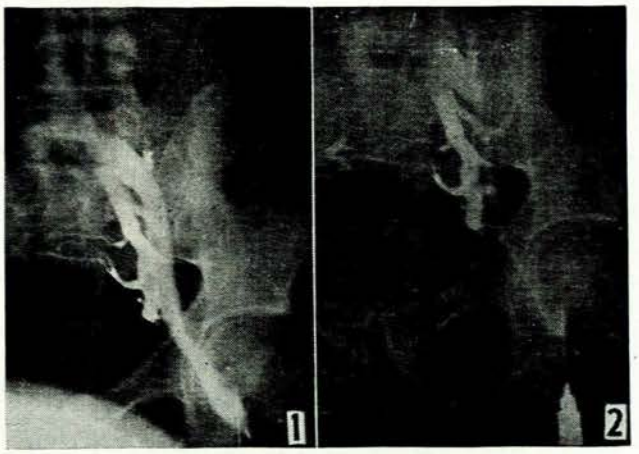
(vi) These patients are definitely *prone to recurrence* of the thrombus and they should be treated with heparin postoperatively. Two of the above cases had to be re-operated because of recurrent thrombus within a week of the first thrombectomy.

(vii) *The 'classical' signs of thrombosis of the calf veins,* viz. tender induration of these muscles and a positive Homan's sign, may also be caused by the venous back-pressure and congestion due to ilio-femoral thrombophlebitis. This was very vividly illustrated by a middle-aged Coloured woman, in whom femoral venotomy with removal of a limited thrombus and the escape of very dark blood under high pressure from the distal part of the vein, caused a marked softening of the calf while she was still on the operating table, with a complete disappearance of pain on coming round from the anaesthetic.

The following case is presented because the patient showed some unusual and interesting features.

Case 1. V.T., a White male aged 35 years, had been a professional boxer until a few years ago. During his boxing career he had to bring down his weight on several occasions by using diuretics, to such an extent that his eyesight became affected. He was still in training at the time of his present illness and he experienced fairly severe aching pain in his calf muscles while cycling, which he did as part of his training.

On admission he complained of pain and swelling of the left leg for one day. His whole left leg was found to be swollen with pitting oedema of the lower leg. The left calf was tender and Homan's sign was positive. Mild tenderness on deep pressure in the left lower abdomen was the only other abnormal finding. After 24 hours in bed with the foot of the bed elevated, there was practically no improvement in the swelling or the pain. A venogram was therefore performed by direct puncture and injection of contrast medium into the left femoral vein with the patient supine (Figs. 1 and 2). The venogram showed



Case 1. *Fig. 1.* Venogram demonstrating a filling defect in the left common iliac vein and early filling of the collaterals via the internal iliac veins. *Fig. 2.* Venogram taken 5 seconds after the injection of contrast medium.

a filling defect in the left common iliac vein, where it crosses in front of the sacral promontory. This filling defect was rather ill-defined, and of more significance was the rapid flow into the

collateral veins which conducted the contrast medium along branches of the internal iliac veins to the right common iliac vein.

Laparotomy through a left paramedian incision disclosed dense adhesions between omentum, caecum and the peritoneal scar of a previous appendectomy. Similar adhesions were present between the sigmoid colon and the postero-lateral parietal peritoneum. The posterior peritoneum was incised in the midline to expose the bifurcation of the aorta and the left common iliac vein. After mobilizing the vein a venotomy was made, between 2 controlling tapes, at its distal end. Bleeding from the distal side was immediate and very brisk but from the proximal side practically no bleeding occurred. A polyvinyl catheter, attached to strong suction, was introduced through the venotomy and slowly advanced in a proximal direction. A mere trickle of dark blood was aspirated through the advancing catheter until it became blocked completely, about 5 cm. proximal to the venotomy. On withdrawal of the blocked catheter, retrograde bleeding was brisk and a soft dark-red clot was suddenly and very rapidly sucked through the catheter. The venotomy was closed with 6-0 arterial silk and a Penrose drain was used to drain the retroperitoneal area.

The postoperative course was uneventful. The patient was started on oral anticoagulants on the second postoperative day and became ambulant on the third day with crepe bandage support for the leg. Ten days after the operation he was discharged from hospital with a completely normal leg and when last seen, 4 months after the operation, he had no complaints and the leg showed no sign of oedema.

Comments. This case differs markedly from the usual case of ilio-femoral thrombosis where there is almost invariably a preceding period of bedrest due to labour, abortion, or surgical or medical disease; where the thrombus consists of a pale, firm and adherent proximal 'head', situated about 15 cm. proximal to the inguinal ligament, and a soft, red, glistening 'tail' lying freely in the lumen and extending for a variable distance distally down the ilio-femoral vein; and where the vein wall in relation to the thrombus shows acute inflammatory changes. The reason why a perfectly healthy young man should develop thrombosis out of the blue is completely unknown. One could speculate about the effect of repeated blunt trauma to the abdomen during boxing. This may give rise to small retroperitoneal haemorrhages and later result in fibrosis which may cause compression of the left common iliac vein where it stretches over the sacral promontory. The periods of severe dehydration causing increased viscosity may have contributed towards thrombosis in this compressed segment, resulting in incomplete obstruction. This could have been the cause of his complaint of pain in his calves on exercise.

(b) *Traumatic Interruption of Continuity*

Vascular complications of laminectomy for removal of intervertebral disc material are well recognized. Serious injury to one of the major bloodvessels lying in front of the vertebral column, and especially to one of the iliac veins, is less well known. The following case gives a very vivid impression of the serious consequences that such an injury to the left common iliac vessel may have.

Case 2. E.v.R., a 17-year-old White girl, developed acute pain in the left iliac fossa, the left loin and the left thigh 12 days after a laminectomy for a disc lesion. Since the onset of the pain, 12 hours previously, she developed rigors. Her temperature was 101°F, her pulse rate was 100/min. and her respiration 22/min. The legs appeared normal with no oedema and no difference on measuring them. She was acutely tender in the left iliac fossa, the left groin and over the whole length of the thigh, along the course of the femoral vessels. There was no tenderness of the calf and Homan's sign was negative. The left femoral pulse could not be felt, but all the other pulses were present and normal.

It was concluded that she had an ilio-femoral thrombosis. The absence of swelling of the leg was thought to be due to the fact that she had been recumbent and not up and about. The left common femoral vein was exposed and opened. It was found to be completely occluded by soft red clot. Distal thrombectomy with catheter-suction was accomplished with ease, but proximally the catheter refused to go further than about 20 cm. proximal to the inguinal ligament. Here it met with an obstruction which defeated all attempts to manoeuvre the catheter past it. For a long time bits and pieces of white and red clot were aspirated from this area before a fair amount of retrograde bleeding appeared. This bleeding was too slight to convince one that a complete proximal thrombectomy had been achieved but, since it appeared there was a primary thrombus too adherent to the vein wall to be removed, further attempts were abandoned.

The day after the operation she felt fine and had no pain. On the next day, however, the pain in the leg returned and became progressively worse. It was now maximal in the groin, and the calf had also become painful. She looked pale and toxic and had a temperature of 103°F and a pulse rate of 120/min.

This time the iliac vessels were exposed through an extraperitoneal approach and a venotomy was made in the external iliac vein. The thrombosis had recurred and the vessel wall was oedematous and pale with thick fibrin around it. After the suction-catheter had been introduced proximally into the vein it was again prevented from advancing further than somewhere in the neighbourhood of the sacral promontory. Injection of saline into the catheter demonstrated the presence of a leak in the vein somewhere more proximally and out of view. The incision was extended and the left common iliac vein and the beginning of the inferior vena cava were exposed transperitoneally. After mobilization of these vessels a large posterior defect in the terminal part of the left common iliac vein, as well as the ragged hole between the fourth and fifth lumbar vertebrae, through which the instrument causing the damage to the vein during the laminectomy had passed, were demonstrated. The defect in the vein was about 3 cm. in length, while in width it comprised almost half of the circumference of the vein. There was no thrombosis in the inferior vena cava, and the complete absence of any sign of bleeding in the retroperitoneal tissues in the neighbourhood of the common iliac vein was very striking. The defect in the iliac vein was repaired with a teflon patch and, after distal thrombectomy had been performed, the venotomy and the abdominal incision were closed.

The day after this operation her temperature was back to normal and the leg was not painful or tender although the calf felt slightly indurated. She was put on oral anticoagulants and made steady progress until she was discharged from hospital 25 days later with the leg free from pain or swelling.

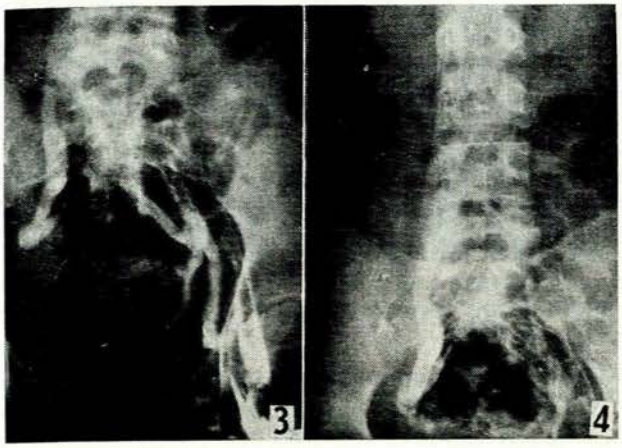
Less than a week later she was back in hospital with the same clinical picture as before her previous operation. A venogram (Figs. 3 and 4) showed obstruction of the segment of the left common iliac vein in which the teflon patch had been inserted. It also demonstrated a very well-developed collateral flow to the right side. Because of this, the teflon patch was removed and the common iliac vein was tied off. The left common iliac vein was stretched so tightly over the sacral promontory and bound down so firmly by young fibrous tissue that its occlusion was not surprising.

Her troubles were not over yet. She developed intestinal obstruction because of adherence of small bowel to the scar in the posterior peritoneum. After a few episodes of swelling of both legs which cleared up on conservative treatment, she was finally and at long last able to leave hospital and, to my knowledge, she has recovered good health.

Comments

1. Retrograde spread of thrombosis, against the direction of bloodflow, from the common iliac vein right down to the origin of the femoral vein, is clearly demonstrated in

this case. It confirms the finding in other cases of ilio-femoral thrombosis that the pale, firm, primary thrombus is situated proximal, i.e. closest, to the heart, and that the secondary red clot forms distal to this. This is contrary to



Case 2. *Fig. 3.* Venogram demonstrating thrombotic occlusions of the left iliac vein and collateral flow along the medial circumflex femoral, the obturator and the gluteal veins to the internal iliac vein on the right. *Fig. 4.* Venogram at a later phase demonstrating the extensive flow in the right internal iliac, the common iliac vein and the lower portion of the inferior vena cava.

the usual description of the process of thrombosis, where the secondary clot is formed in the stagnant column of blood behind the primary occluding thrombus (behind, according to the direction of flow), or in front of it, where the blood will also be stagnant up to where the first collateral vessel branches off. This retrograde spread of the thrombosis demands an explanation. One possibility is that retrograde flow of blood down the ilio-femoral veins occurs after proximal occlusion. That this can occur down to the level of the circumflex femoral veins, through which the blood may be returned along the venous circles of Edwards, must be conceded. Since the thrombosis has spread right down to the origin of the femoral vein, retrograde flow, to this extent, especially in the absence of any oedema, can hardly be visualized. Another possible explanation may be that the extent of the stagnant column of blood in front of the primary occluding thrombus is much more extensive in veins than in arteries. This would be possible only if the venous collaterals, which are potentially very extensive, did not open up after proximal occlusion of the main stem. Only in this way can a stagnant column of blood right down to the popliteal fossa be visualized as occurring. Experience with venous collaterals, however, makes this explanation completely unacceptable. Whichever way we look at this problem, *it must be concluded that retrograde spread of thrombosis cannot be explained on the basis of mechanical hydrostatic factors.*

2. *The cause of oedema in acute thrombosis* is a problem which this case raises. At her first operation extensive occlusion by thrombosis of the whole ilio-femoral vein was found and yet this did not cause the slightest degree of oedema. This contrasts markedly with the very extensive oedema seen in other cases of much more limited thrombosis, where the patient may develop the oedema while also recumbent; and also with the pronounced oedema of our patient before her subsequent operations, in spite of the fact that the extent of the thrombosis was much reduced and in spite of the excellent collateral circulation as illustrated by the venogram (Fig. 2). Pertinent to this problem is the difference in appearance of the obstructed vein between the first and the second exploration. In the first the vein wall was still blue and only slightly thickened, while in the second it was pale and extremely oedematous with gross oedema and fibrin in the tissues surrounding both the vein and the artery.

An increase of as little as 2 - 3 mm.Hg at the venous end of the capillary prevents normal absorption of tissue fluid by the capillary, and under these conditions the lymphatics help in the absorption and removal of the excess of tissue fluid. While it is obvious that the venous pressure could not have been more at the time of our patient's second operation, it must be concluded that *the oedema must have appeared when obstruction to the main lymph vessels of the limb occurred owing to the perivenous inflammation.* It has been shown by Oganesion[24] (quoted by Morrell) that in venous thrombosis there is a concomitant dilation of lymphatic vessels and that lymph flow is markedly increased. This is also demonstrated experimentally by occluding major limb vessels. After producing experimental acute thrombophlebitis, lymphangiograms at the height of the resulting oedema showed narrowing of the neighbouring lymph vessels, while histologic studies showed thickened lymphatic walls with some desquamation of the endothelial lining.[25] These findings tend to confirm the above clinical observation that *in acute thrombophlebitis, the lymphatic system may be altered and fail in its reserve function, with the combination of venous and lymphatic occlusion resulting in regional oedema.*

3. *Thrombophlebitis is merely a later phase of phlebothrombosis.* I think that this is very well illustrated by case 2. The injury to the common iliac vein took place 12 days before any symptoms appeared. The nature of the injury was such that thrombosis must have started immediately afterwards. Only by this belief can the absence of any sign of retroperitoneal bleeding be explained. For 12 days she had 'silent' thrombosis, or phlebothrombosis; then she developed proximal pain in the leg with no oedema, and in spite of her raised temperature and rigors, the gross appearance of the vein was that of a mild inflammatory reaction; 48 hours later she had the full-blown clinical picture of thrombophlebitis and the vein showed extensive inflammatory reaction, both in the vein wall and in the surrounding tissue. This same sequence of events is seen in most cases of ilio-femoral thrombosis, where the initial symptom is pain in the groin, lower abdomen or buttock, which subsequently spreads in the course of a few days down the leg, to be followed soon by a similarly progressive downwards-spreading oedema.[8] Since the majority of cases of ilio-femoral thrombosis develop symptoms a week or more after the precipitating period of bedrest or operation or labour, and because the primary thrombi are usually fairly firmly adherent by that time, it seems as if in all these cases the episode of clinical thrombophlebitis was preceded by a period of phlebothrombosis. This concept, that thrombophlebitis and phlebothrombosis are merely different stages of one process, is further strengthened by the failure to culture any organisms from the thrombus

and from the vein wall (except in cases of septic thrombophlebitis, of course). The inflammatory reaction appears to be a response of the tissues to the presence of the clotted blood, possibly in the same way in which a large haematoma in the tissues can cause such a severe local and systemic inflammatory reaction that it is difficult to distinguish it from an abscess.

4. *The value of thrombectomy is emphasized* by the fact that the femoral vein remained patent after thrombectomy and that, in spite of the recurrence of obstruction of the common iliac vein, it enabled the blood from the leg to bypass the obstruction along the venous circles, as is shown by the venogram (Figs. 3 and 4) which was taken 30 days after the thrombectomy.

5. *Tying off the common iliac vein* at the time of the second operation would have been a better form of treatment than the teflon graft insertion.

(c) *Other Causes of Obstruction of the Venous Outflow*

Compression by tumour and by post-radiation fibrosis and traumatic arteriovenous fistulae are a few examples. Hardin[26] has achieved impressive success with bypass saphenous grafts in 5 cases—1 as a result of operative trauma, 1 owing to traumatic thrombosis and 3 with obstruction due to carcinoma and radiation. This use of venous bypass grafts for the relief of venous obstruction has been much neglected, probably because of its failure owing to occlusion in cases of obstruction to the superior vena cava. In the lower limb and especially in its proximal veins with their fairly large calibre the venous pressure is higher and there will be less likelihood of occlusion of the graft.

2. Incompetence of the Venous Outflow Tract

Depending on the length of the outflow tract involved, the valvular incompetence results in a dynamic obstruction to the venous outflow from the leg. Once incompetence in the outflow tract is established it tends to spread progressively down the leg. The venous pressure increases concomitantly with the extent of the incompetence. When it reaches the calf, the communicating veins also become incompetent as a result of dilation, so that the final stage of venous ulceration is reached.

(a) *Incompetence Secondary to Thrombophlebitis*

The pathogenesis of incompetence as a result of organization and recanalization of the thrombus is well known. Boyd *et al.*[27] were the first to show by venograms that within 1 or 2 years deep veins blocked by thrombosis were effectively recanalized. My impression is that by the time that the postphlebitic syndrome has developed, most, if not all, thrombosed veins have either recanalized and become incompetent or they have been replaced by collateral veins. This impression tends to be confirmed by the findings of Dow[3] with venographic investigation in 62 patients with oedema and ulceration of the leg. In no case were the deep veins actually blocked. Studies of the major lymphatics of the leg in cases of postphlebitic syndrome[28] as well as experimental examination of the lymph vessels late after the production of thrombophlebitis, show convincingly that lymphatic stasis is not involved as a causative factor in the oedema of these cases.

It is my firm conviction that the prevailing conservative treatment of acute ilio-femoral thrombosis with bedrest and anticoagulants does not prevent the postphlebitic syndrome. At the time that symptoms appear in the acute case, the thrombosis is already established and anticoagulants will not melt it away. The only value that anticoagulants may have in the prevention of the postphlebitic syndrome is in those cases of limited thrombosis where they will prevent further extension of the clot. Such a case may clear up without any sequelae because the resulting incompetent segment of vein is short. *The only effective prevention of the postphlebitic syndrome is thrombectomy of the acute case.* This view is very slow in being generally accepted because the majority of cases with acute ilio-femoral thrombosis clear up on conservative treatment, and it is not always realized that the sorry aftermath of the acute case only becomes evident after a latent period of 2 or 3 years.

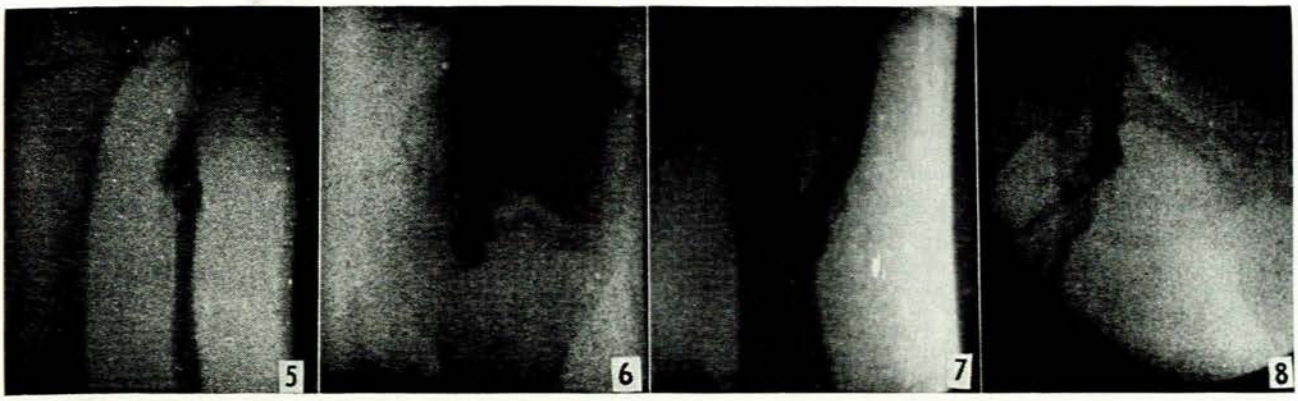
(b) *Incompetence following Rupture of a Valve*

As far as I know this has not been described before and its incidence must be completely unknown, because it will only be recognized when the vein is explored and opened.

Case 3. V.V., a White woman aged 51, complained of pain and swelling of the left lower-leg which became specially noticeable towards the afternoon after she had been standing a lot. The right leg was also inclined to swell slightly, especially in summer, but she had no pain in it. Her left leg had been put in a plaster-of-paris cast for a fractured patella 5 years previously. After the plaster cast was removed she developed calf-vein thrombosis. This was treated and cleared up, but since that time the swelling of the lower leg had become progressively worse and she had repeated bouts of pain in the upper part of the left calf, just below the popliteal fossa. The last of these attacks then occurred 2 weeks previously and she was treated for a week in hospital as suffering a deep calf-vein thrombosis.

On examination the left leg showed marked pitting oedema and slight tenderness on pressure in the lower part of the popliteal fossa. The right leg showed very slight pre-tibial pitting. She looked anaemic and showed a haemoglobin of 10 G/100 ml. of blood. The urine showed no albumin. The rest of her systemic examination was essentially normal. Cinevenography showed complete incompetence of the left femoral vein down to the level of the lower part of the popliteal fossa where a competent valve held up the long column of blood. Figs. 5 - 8 are stills made from the cine film taken directly after injection of the contrast medium into the femoral vein with the patient tilted about 30° short of the vertical leg-down position. Fig. 5 shows the contrast medium falling down the femoral vein, while Fig. 6 demonstrates the broad column where it is stopped by the competent valve in the popliteal vein, causing a prominent bulge. After watching this valve for a while in the cine-venogram a drop of blood can distinctly be seen leaking through the valve. Fig. 7 demonstrates the valve in Hunter's canal while contractions of the calf muscles pump the blood rapidly up the femoral vein. The normal structure of the valve can be seen clearly, showing that it has not been rendered incompetent by previous thrombosis. The cine-venogram suggested a partial thrombotic occlusion of the popliteal and tibial veins. There was no demonstrable incompetence of either saphenous veins and the iliac vein and inferior vena cava were patent. Cine-venography of the right leg showed no abnormality.

Operative exploration of the left popliteal vein showed no abnormality and bleeding from both ends of a venotomy in it showed brisk bleeding. On opening the left common femoral vein, a longitudinal tear in the intima of its posterior wall, about 3 cm. in length, was found. The tear went through the valve in the common femoral vein which was situated just proximal to the junction of the superficial and the deep femoral veins and which was guarding both these openings. The



Case 3. *Fig. 5.* Cine-venogram with the patient semi-erect, showing retrograde flow down an incompetent femoral vein. *Fig. 6.* Competent popliteal valve shown lower down. *Fig. 7.* Structurally normal valve in Hunter's canal is shown during contractions of calf muscles. *Fig. 8.* After operative repair of the femoral valve.

cusps of the valve were atrophic and the gap, caused by the intimal tear between them, was so wide that it rendered the valve obviously incompetent. The edges of the tear, although level with the rest of the intima, were elevated slightly above the floor of the gap. The floor of the gap was red in colour in comparison with the paler intima. The edges of the tear, and with them the valve cusps, were drawn together with 3 sutures of 6 - 0 arterial silk, introduced and tied on the outside of the posterior wall of the femoral vein. The patient still complained of pain in the upper part of the calf postoperatively, but this and the swelling cleared up completely after a few weeks. Fig. 8 demonstrates the postoperative appearance of the femoral vein and the repaired valve during cine-venography done in the same way as with Fig. 5. The valve is now competent and filling of a few collateral veins can be seen.

Comments

1. Although thrombophlebitis in the venous pools of the soleus muscle might have occurred and may be the explanation of her pain, backpressure and bulging at the competent valve in the popliteal vein can cause similar symptoms. The tear in the intima, however, appeared too recent to be incriminated in her previous attacks.

2. The tear in the intima was unaccompanied by any pain when it occurred. The mechanism by which such a tear may occur can be visualized as a sudden, sharp increase in intra-abdominal pressure being transmitted by the valveless iliac vessels to the column of blood resting against the closed femoral valve, and this pressure wave, if big enough, may cause a tear in the valve.

3. Her anaemia probably played a subsidiary role in the causation of the oedema, thus explaining the swelling of the right leg which cleared up after correction of the anaemia.

4. Cine-venography is extremely valuable in studying venous haemodynamics and in the diagnosis of its abnormalities.

(c) *Idiopathic Incompetence*

Case 3 demonstrated very clearly how venous hypertension can cause incompetence of a structurally normal valve by distension of the vein. Most of those cases in which no history of previous deep-vein thrombosis is present and who present with a chronic swollen leg, can be included under this category. Although the aetiology is unknown, increased venous pressure, due to either increased proximal backpressure or distal increased bloodflow as a result of, e.g., the opening up of arteriovenous fistulae possibly under hormonal influence, is probably the initiating mechanism. Valvular incompetence will then be caused by the venous distension produced by the raised transmural pressure.

SURGICAL TREATMENT OF INCOMPETENT DEEP VEINS

The rationale for the surgical repair of incompetent deep veins is that if the highest incompetent valve in the leg can be rendered competent, the more distal valves will be protected from the high hydrostatic pressure. The vein may regain its tone, and diminish in calibre with resulting competence of its valves. Even if the more distal valves have been destroyed, the proximal competent valve may still be enough for the return of normal haemodynamics of the venous outflow from the leg. It is accepted that incompetent communicating veins in the lower leg will have to be treated in the usual way. If the factors producing distal venous hypertension persist, it is obvious that the repaired valve will again become incompetent. It is therefore imperative that these factors should be elucidated.

Restoration of venous-valve function might be attempted in various ways, including reconstructive venoplastic procedures, substitution with synthetic valve prostheses or by transplantation of short valve-bearing vein segments. The latter is the simplest approach and its use is encouraged by the recent experimental use of this method by Waddell *et al.*[29] They found that four-fifths of autographs in 30 dogs showed valve function with no thrombosis and with gross and microscopic recognition of valve cusps 2=12 months postoperatively.

CONCLUSION AND SUMMARY

1. The title of this paper was chosen for the purpose of emphasizing the fact that the chronic swollen leg of venous stasis is due not only to deep-vein thrombosis with consequent venous incompetence but that it is also the result of disordered haemodynamics of the venous outflow of the leg, owing to a variety of causes.

2. Review of the anatomy and the haemodynamics of the veins of the leg revealed that (*a*) the venous circles of Edwards are potential pathways for the return of blood after its retrograde flow down the ilio-femoral vein, (*b*) the peculiarities in the course of the left iliac vein may contribute to the marked prevalence of ilio-femoral thrombosis on the left side, (*c*) distension of veins is the result of raised intramural pressure and not of the reactivity of the vein wall, and that (*d*) raised intramural pressure is caused by increased inflow through the capillary bed and through the arteriovenous fistulae (more important) or by decreased outflow as a result of backpressure due to obstruction or incompetence of the major outflow tract. There are indications that the arteriovenous shunts play a more

important role in health and disease than has been suspected up to now.

3. The major causes of disturbed venous outflow from the leg are:

(a) *Obstruction* — (*i*) Thrombophlebitis
(*ii*) Disruption of continuity
(*iii*) Occlusion by tumour or fibrosis
(b) *Incompetence* — (*i*) Postphlebitic
(*ii*) Traumatic with valve rupture
(*iii*) Idiopathic

4. The retrograde thrombosis, which is a common occurrence in the proximal veins of the leg, cannot be explained by the prevailing concept of mechanical hydrostatic factors.

5. In *acute* thrombophlebitis, the major lymph vessels of the leg are involved in the peri-venous inflammation and, with the combination of venous and lymphatic obstruction, oedema results. Venous obstruction by itself does not usually cause oedema.

6. Thrombophlebitis is but a later phase of phlebothrombosis.

7. *Treatment*: (*a*) Acute (ilio-femoral) thrombophlebitis — thrombectomy; (*b*) Chronic obstruction — bypass venous graft; (*c*) Incompetence — restoration of valvular competence to the most proximal valve in the leg by replacement with autogenous valve-bearing vein segments or repair of valvular tears.

GEVOLGTREKKINGS EN OPSOMMING

1. Die opskrif van hierdie artikel is gekies om nadruk te lê op die feit dat die chroniese dik been van veneuse stase nie die gevolg van diep veneuse trombose met gevolglike inkompetensie alleen is nie, maar eerder die gevolg van versteurde hemodinamika van die been se veneuse uitvloei, a.g.v. 'n verskeidenheid van oorsake.

2. 'n Oorsig van die anatomie en hemodinamika van die venes van die been toon dat (*a*) Edwards se veneuse sirkels potensiële bane vorm vir die terugvloei van retrograde vloei in die ilio-femorale vene, (*b*) die besondere anatomie van die linker iliaka venes mag bydra tot die oorwegende voorkoms van ilio-femorale trombose aan die linker kant, (*c*) distensie van venes die gevolg is van verhoogde transmurale druk, en nie van die reaktiwiteit van die wand van die vene nie, en dat (*d*) verhoogde transmurale druk veroorsaak word deur verhoogde invloei vanaf die kapillêre bed en vanaf arterio-veneuse kortsluitings (van meer belang) of deur verminderde uitvloei weens obstruksie en inkompetensie. Daar is aanduidings dat die arterio-veneuse anastomoses 'n groter rol speel as wat tot nog toe vermoed is.

3. Die belangrikste oorsake van verstoorde veneuse uitvloei van die been is:

(a) *Obstruksie* — (*i*) Tromboflebitis (ilio-femoraal)
(*ii*) Verbreking van kontinuiteit van die vene
(*iii*) Toedrukking deur tumorweefsel of fibrose
(b) *Inkompetensie* — (*i*) Post-flebities
(*ii*) Troumaties met skeur van die klep
(*iii*) Idiopaties

4. Die retrograde trombose wat die gewone soort in die proksimale venes van die been is, kan nie verklaar word op grond van die aanvaarde meganiese hidrostatiese faktore nie.

5. In akute tromboflebitis word die hoof limfbane van die been betrek in die peri-veneuse inflammatoriese proses en die kombinasie van veneuse en limfatiese obstruksie veroorsaak edeem. Veneuse obstruksie veroorsaak op sigself gewoonlik nie edeem nie.

6. Tromboflebitis is slegs 'n later fase van flebotrombose.

7. *Behandeling*: (*a*) Akute ilio-femorale tromboflebitis — trombektomie; (*b*) Chroniese obstruksie — veneuse omleidingstransplantaat; (*c*) Inkompetensie — herstel van kompetensie van die klep wat mees proksimaal in die been is deur verplasing met outogene klep-draende segmente van vene of deur herstel van klep-skeure.

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