INTERMITTENT CLAUDICATION
IN
ATHEROSCLEROTIC OCCLUSION OF THE ARTERIES
TO THE LOWER LIMB.

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" In the so-called segmental arteriosclerosis, only the occlusion is segmental, not the arteriosclerosis".

Martorell, 1950.

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CONTENTS.

LIST OF TABLES. Page.

CHAPTER ONE.

INTRODUCTION.

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INTRODUCATION.

The development of surgical methods in the treatment of arterial occlusion has constituted one of the outstanding contributions to the advancement of Surgery in the post-war years and with it has come an increased understanding of the underlying factors involved. The initial stimulus came from the development of safe methods of angiography which delineated the lesions producing symptoms and created a challenge to surgeons to deal with the mechanical factors responsible. In the field of peripheral vascular disease, which is largely, though far from exclusively, the effects of atherosclerosis on the abdominal aorta and its tributories, advance has been swift and much of the current literature is devoted to the assessment of surgical techniques and the results that can be obtained by utilising them.

The difficulty of obtaining reliable information as to the physiological disturbances induced by alteration in blood flow, and the relative absence of physicians actively interested in this field has tended to limit the scope of fundamental research, but much has been learned about the disease itself through

increased application of radiological and surgical techniques.

The natural history of atherosclerosis in the lower limb has been established by the monumental survey of 1476 untreated cases of claudication carried out by Boyd and his colleagues and reported by Boyd (1960) and by Bloor (1961). This has established a base line against which surgeons can assess the results of surgical treatment. Boyd and Bloor confirmed that intermittent claudication is in many cases a benign condition which does not progress, yet in others it presages a relentless deterioration to gangrene or death. Unfortunately, it is not yet possible to differentiate between the two types of claudication in the individual patient.

This thesis has been written in an attempt to correlate the information gained during an experience covering six years. A major portion of the material has been the analysis of 528 femoral arteriograms performed in the investigation of a comprehensive group of 264 male patients with intermittent claudication due to femoro-popliteal occlusion, and the analysis of 90 aortograms in patients with claudication due to aorto-iliac occlusion. The pattern of arterial occlusion has been elucidated and the material has been supplemented

by additional illustrations where necessary.

For convenience in reading, photographs and other figures have been bound separately so that illustrations and text can be read together, and tables have been included in the appropriate portions of the text.

CHAPTER TWO.

HISTORICAL.

CHAPTER TWO.

HISTORICAL ASPECTS OF INTERMITTENT CLAUDICATION.

The commonest symptom of arterial insufficiency in the lower limb is intermittent claudication, i.e. pain developing in certain groups of muscles during exercise which produces intermittent limping (Latin:- claudicare - to limp).

This pain is most commonly felt in the calf muscles and is described by patients as a cramping type of pain which develops after walking a variable distance. In its mildest form, it may be merely a feeling of tiredness in the leg which causes the patient to walk more slowly, but, as the condition develops, the pain increases in severity and he is forced to stop and rest. The pain passes off within a few minutes and he is then able to walk a similar distance before the pain recurs and compels him to halt once more.

The condition was first described in horses in 1831 by Boullay, a French veterinary surgeon, and it was later found to be associated with occlusion of the main artery of the leg. More than 20 years passed until Charcot, a Parisian surgeon, described the condition in man in 1858.

In 1931, Lewis published the results of his

studies on the mechanism of production of the pain and showed that it is due to accumulation of the products of muscle metabolism. In the presence of arterial occlusion proximal to the affected muscles, metabolites cannot be removed as quickly as they are formed and these soon reach a level at which the somatic nerve endings in muscle are stimulated and pain is produced. In 1934, Kissin showed that it was not necessary for the artery to be occluded by disease when he showed that a similar pain can be produced in a normal limb by muscular exercise when the arterial circulation is stopped by a pneumatic tourniquet.

Although the condition was initially described in the middle of the 19th century, opportunities for the study of claudication were few. Elucidation of certain aspects had to await the development of x-rays, the pioneer studies of R. dos Santos in aortography (1929) and the development of the modern contrast media.

Following Röntgen's announcement of the discovery of x-rays in Würzburg in 1895, attempts were made in various fields to utilise this new technique for the elucidation of disease. The demonstration of blood vessels in amputated limbs was achieved in 1896 by Haschek and Lindenthal in Germany and separately in the

same year by Destot and Berard in France.

John McIntyre, who founded in Glasgow the second oldest diagnostic x-ray department in the world, also experimented with the demonstration of blood vessels in amputated and autopsy material. At first he used chalk which had been used to produce the earliest pictures, but he also tried other radio-opaque materials such as calcium sulphate and compounds of bismuth and barium.

As the technique of radiology improved and exposure times became shorter, the search for improved opaque media was intensified and, in 1923, Sicard and Forestier of France discovered the value of iodised poppy-seed oil. Following experiments on dogs, they injected 4 ml. of lipiodol into an arm vein and followed the course of the dye as it flowed to the heart and pulmonary artery.

The first angiogram of a peripheral vessel was produced by Berberich and Hirsch of Germany in 1923 by injecting strontium bromide into the arteries and veins of the hand, and the first successful arteriogram of the leg in 1924 by Brooks of the United States of America using sodium iodide. His method was to expose the femoral artery in mid-thigh under local anaesthesia, then to inject 100 grammes of sodium iodide in 100 mls.

of water during a short period of general anaesthesia with nitrous oxide. This technique proved to be so successful that it remained the standard technique of femoral arteriography for many years.

At the same period, K. dos Santos of Portugal and his colleagues Lamas and Caldas were developing the technique of trans-lumbar aortography and they published their paper in 1929. They injected 20 ml. of 80-100% sodium iodide solution into the aorta through a needle inserted from the back using a compressed air machine. The essentials of this technique remain the basis of trans-lumbar aortography, but at that time a violent controversy arose concerning its value and its dangers.

In 1941, Farinas in the U.S.A. introduced the method of aortography by femoral catheterisation at open operation and Seldinger (1953) subsequently devised the method of percutaneous arterial catheterisation which is now universally known as the Seldinger technique. The accuracy with which the siting of the dye injection can be controlled by this method and its versatility of application to angiography of the renal, superior mesenteric, and coeliac axis vessels have established the method as a standard one.

The routine use of aortography by either

method was slow to develop and this, to a large extent, was due to the use of sodium iodide as a medium. However, in 1942, Nelson and separately Doss in the U.S.A. published accounts of the use of aortography in the investigation of renal disease and the method gained ground. Progress in this country remained slow and it was not until 1950 that Griffiths published the first British account of aortography in renal diagnosis.

Initially, aortography was utilised in the diagnosis of renal disease and, although occlusive arterial disease was occasionally demonstrated incidentally, there was no impetus to the routine use of either aortography or femoral angiography until surgical advances made it essential to have angiograms performed.

Contrast Media.

The development of the modern trimolecular organic iodide compounds has eliminated many of the hazards which prejudiced the establishment of angiography as a routine procedure.

The early development of contrast media was due to urological stimulus. The initial use of these was to outline the bladder and, later, the upper urinary tract, but, in 1923, Osborne and his colleagues administered sodium iodide intravenously or by mouth

and visualised the urinary tract in half the cases they examined.

The discovery of the modern organic compounds was made by Binz of Frankfort (1919) who was working on the Salvarsan group for use in syphilis and fortuitously produced a substance called Selectan which was later found to be excreted in bile and urine. Uroselectan was developed later, and in 1931 Lichtenberg produced Uroselectan - B, the original member of the Iodoxyl group. Discovery of Abrodil, the original member of the Diodone group, came later and eventually, in 1950, Sodium Acetrizoate was introduced. This was a trimolecular organic compound with a higher iodine content than either the Iodoxyl or Diodone groups.

This group of acetrizoate drugs has a high iodine content which gives better x-ray contrast yet has proved to be less toxic and more rapidly eliminated than older media. There are now standard media for intravenous use, e.g. Hypaque (Sodium and N-methyl-glucamine salts of 3:5 - diacetamido - 2:4:6 tri-iodobenzoic acid) and Urografin (A mixture of the sodium and methylglucamine salts of 3, 5, diacetylamino 2:4:6 triodo-benzoic acid). Triosil (a simple solution of sodium metrizoate: Sodium 3 - acetamido - 2:4:6 - tri-iodo- 5 - N - methyl acetamido-benzoate) is a more

recent introduction which is manufactured in this country and is less viscous and possibly more radio-opaque than the other two in equivalent concentrations.

Development of Vascular Surgery.

The progress of vascular surgery in the earlier part of this century was slow and was mainly concerned with the treatment of gangrene, the cure of aneurysm, and the use of indirect methods to produce vaso-dilatation. In 1913, Leriche introduced periarterial stripping to relieve vasospasm and induce vaso-dilatation in the peripheral arteries. The results of this procedure were variable and it was later realised that it did not produce an adequate sympathectomy owing to the existence of other pathways via the somatic nerves.

In 1924, Royle and Hunter performed sympathectomy for spastic paraplegia and noted that the limb on the operated side became warmer. Following this and other observations on the increased warmth of the foot following sympathectomy (Adson & Brown, 1925) the operation was applied to the ischaemic limb with varying degrees of success. Lumbar ganglionectomy is still performed but with a better appreciation of its application and limitations than formerly (Gillespie, 1960).

Direct arterial surgery may be said to have begun when Murphy (1897) described a method of suture for large arteries. Carrel, in 1902, described certain basic principles and a technique which he applied to experimental animals in 1908, and, in 1912, Lexer excised a popliteal aneurysm and replaced it with a functioning vein graft.

In 1913, Hogarth Pringle, who was on the staff of the Glasgow Royal Infirmary, successfully excised a popliteal aneurysm and restored continuity by a vein graft and also performed a similar successful replacement of a traumatic brachial aneurysm. In 1914, Subbotitch presented the first large series of reconstructive operations for traumatic aneurysm and arteriovenous fistula where the vessels were repaired by suture, and this formed the basis of treatment for many years.

The introduction of heparin by Murray of Toronto in 1939 when he reconstructed the popliteal artery with an autogenous vein graft increased the possibilities of success but arterial grafting played relatively little part in the injuries resulting from World War II (1939-1945) and the Korean War (1952).

In 1949, Gross, Bill and Peirce showed that homografts could be successfully used for aortic

replacement in man and this stimulated the use of homografts and the development of artery banks. Initially, grafts were refrigerated in saline nutrient media but rapid freezing and storage in solid carbon dioxide (Hufnagel and Eastcott, 1952.) and freeze-drying (Eastcott, 1953.) were later introduced to overcome the difficulties of storage. The difficulties of collection, storage, and handling were formidable and as subsequent reports of aneurysmal dilatation in homografts appeared and synthetic prostheses became popular, homografts were largely abandoned.

In 1952, Voorhees, Jaretzki, and Blakemore described the use of a cloth tube to replace the abdominal aorta in dogs and other materials were introduced and later discarded e.g. Ivalon. The introduction of Dacron (De Bakey and his colleagues, 1958.) and Teflon (Rob, 1957.) proved so successful in clinical use because of the ease of handling, storage, and sterilisation that they have now largely replaced homografts.

Although synthetic grafts have been successful in aorto-iliac surgery, they do not give good results in femoro-popliteal operations and other methods have been developed.

In spite of early success with vein grafts

(Lexer, Pringle.) and the report by Weglowski (1925) of long-term patency in 40 vein grafts inserted for traumatic aneurysm and arterio-venous fistula, these were seldom used until Kunlin (1951) reported on their use as long by-pass grafts in femoro-popliteal occlusion.

Similarly, attempts at thrombo-endarterectomy had been reported by Gask (1912) in a series of collected cases but this technique did not become standard until the work of J.C. dos Santos (1947). Subsequent developments led to the use of arterial strippers (Cannon and Barker, 1955.) and to the use of vein patches or strips to close the arteriotomy (Edwards, 1962.). Various combinations of these methods are now being assessed in current literature.

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Although Reconstructive Surgery is not the ideal treatment for chronic generalised obliterative arteritis as it does not prevent deterioration or induce regression of the disease itself, it is valuable in the alleviation of symptoms and in the salvage of limbs which might otherwise be amputated. Its introduction constitutes one of the recent advances in Surgery which is still in process of evolution and the final chapters of its history have yet to be written.

CHAPTER THREE.

ANGIOGRAPHY.

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In the study of arterial occlusion, several methods of investigation may be employed but the most important and informative is angiography. This has been made relatively easy and safe by the development of the modern contrast media and by the adoption of percutaneous techniques which have superceded operative exposure of the vessels.

Angiography is essential for the accurate assessment of the nature and the extent of the disease. It provides confirmation of the diagnosis of atherosclerosis, gives a graphic illustration of the site and extent of arterial occlusion, of the state of the vessels above and below the occluded segment, of the presence of other unsuspected lesions and an indication of the pattern of the collateral circulation. Incipient occlusions are revealed and the possibility of future occlusion can be evaluated.

It is, therefore, widely employed in the selection of occlusions suitable for reconstructive surgery.

The two principal methods used in the lower limb are aortography and femoral arteriography.

In the present series translumbar aortography

was employed where weakness or absence of one or both femoral pulses was noted. Where the femoral pulses appeared normal, bilateral femoral arteriography was performed.

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A. TRANSLUMBAR AORTOGRAPHY.

Since dos Santos (1929) published his account of the method of translumbar aortography to outline the aorta and its main branches, improvements in technique have been made but the method remains basically the same. Visualisation of the abdominal aorta and its branches has become an essential part of the investigation of patients with suspected occlusion of the aorta or iliac vessels.

In essence, the technique involves the forcible injection of radio-opaque dye into the aorta through a needle introduced from the left lumbar region posteriorly and the exposure of one or more radiographic films to record the outline of the vessels.

The level of the aortic puncture is usually varied according to the information required.

Where the investigation is urological requiring visualisation of the renal vessels, the needle

is directed upwards to enter the aorta at or about the level of the first lumbar vertebra. When one wishes to demonstrate the occlusive lesions of atherosclerosis, it is preferable to puncture the aorta below the level of the renal vessels and thus diminish the incidence of renal complications which can follow the rapid injection of a large volume of dye into the renal artery. In such cases, the aorta is entered at or about the level of the second lumbar vertebra near the origin of the inferior mesenteric artery. This procedure, often called 'low lumbar aortography', is usually satisfactory as the majority of aortic occlusions involve the region of the bifurcation.

Because of the high rate of blood flow in the aorta and the resistance to injection produced by the diameter of needle used, hand pressure injection does not give satisfactory results, and some method of high pressure injection is required. The motive power for rapid injection can be obtained by using a cylinder of compressed air or oxygen as was originally used by dos Santos.

Melick and Vitt in 1948 described the use of an injector whose motive power is produced by a strong spring and they claimed satisfactory results in practice.

In 1955, Stirling introduced an injector

which utilised the force which could be transmitted to the syringe piston by manual depression of a long lever. For many of the aortograms performed in this series, the original prototype of Stirling's machine was used. The apparatus is illustrated in Fig.1 where the syringe is seen to be supported on a platform by two clips screwed into position. The lever is hinged at one end and a cap overlies the head of the piston and transmits the pressure applied at the end of the lever with a mechanical advantage of about four. With this type of injector it is possible to inject 20 ml. dye in 2-3 secs. This displaces the equivalent volume of blood in the aorta producing a column of dye which outlines the aorta and main vessels when recorded radiologically.

Technique.

Materials : The injector machine (Fig.1) with a 20 ml. Luer-Lok syringe mounted and filled with dye. Luer-Lok fittings are essential as the pressure generated will cause disengagement of ordinary fittings.

Pressure rubber tubing (solid rubber tubing with an outer coating of woven cotton fibre) is required to obviate the dissipation of pressure which occurs with ordinary rubber tubing and this tubing has Luer-Lok fittings at each end.

The needle used is of No.18 gauge (0.55 mm. internal diameter) with Luer-Lok mounting. This gives satisfactory technical results and is less likely to damage the aorta than a needle of larger bore.

Improvement in the contrast media available for aortography has been rapid in recent years and 76% Urografin has been used for most aortograms. Recently, Triosil, which is manufactured in Britain and is less viscous than Urografin, has been used.

As the examination would produce pain and movement in the conscious patient, general anaesthesia is required and this has the advantage that controlled apnoea can be obtained by the use of modern anaesthetic drugs.

The patient is prepared for general anaesthesia by premedication of 50 mgms. pethidine and gr. 1/100 atropine. Prior to commencement, a test dose of 1 ml. 76% Urografin is given intravenously to detect possible cases of allergy to the dye.

Anaesthesia is induced in the supine position on the x-ray table with pentothal and scoline and intubation of the trachea is performed with a cuffed laryngeal tube. It is continued by the method favoured by the anaesthetist, e.g. cyclopropane and oxygen, nitrous oxide-oxygen-trilene, etc. and further scoline

is given as required to permit control of respiration and production of apnoea when required.

When anaesthesia is satisfactory, the patient is turned into the prone position (Fig.2) and preliminary radiographic exposures are made to check exposure factors for the patient and the siting of the film.

At the same time, the operator is preparing the injector machine and filling the syringe and tubing with dye. The 20 ml. Luer-Lok syringe is clipped in position and the controlling clips carefully tightened. Because 76% Urografin is viscous and the piston of the syringe tends to stick, it is desirable to wash out the syringe with saline before use, and to fill the syringe only when the patient is almost ready for injection. When Triosil is used this precaution can be omitted owing to its low viscosity. The pressure tubing is filled with dye to exclude air and attached to the syringe filled with 20 ml. dye. The lever of the injector is then depressed to test the apparatus for leaks and easy movement and the syringe is refilled ready for use.

A sharp needle 14 cms. long of No.18 gauge with a short bevel and stillette is then selected. The site of the needle puncture is prepared with methylated spirit and the area towelled off. Sterile gloves may be worn if desired, but greater sensitivity in locating

the aorta may be obtained without them. The needle is entered at a point below the left 12th rib about 12 cms. from the midline (Fig.3) and directed antero-medially in a slightly upwards direction to reach the side of the vertebral column. The needle is partly withdrawn and directed anteriorly towards the front of the vertebral body where the elastic resistance of the anterior longitudinal ligament is often felt.

At this stage, the final adjustment is made and the needle is partly withdrawn and inclined more anteriorly to reach the equivalent depth of the previous position. On further penetration, pulsation may be transmitted from aorta to needle if the siting is correct although pulsation is not always felt in atherosclerotic aortas and, on further pressure, the needle is felt to "give" suddenly as it passes through the aortic wall. The stillette is withdrawn and, if the puncture is successful, blood will flow from the needle. Using a No.18 needle, the blood does not spurt but gives a rapid pulsatile flow. Should no blood appear, the needle is carefully withdrawn as it may have passed into the aorta and penetrated the right aortic wall. If no blood is obtained during withdrawal, the stillette is replaced and exploration continued.

When aortic puncture has been accomplished,

the needle is rotated several times to ascertain that the blood flow is maintained in all positions of the needle. This is an essential precaution as it is possible to have a satisfactory flow in one axis and not in another. When the flow from the needle is satisfactory in all axes, the tubing is connected and the twist imparted during locking is allowed to unwind.

Preparation for injection is now complete. The anaesthetist produces apnoea and the radiologist is alerted. The pressure tubing is firmly held to prevent the rotation induced by the pressure of injection and an assistant depresses the lever of the injector machine steadily and forcibly. When the films have been taken, the tubing is disconnected and the flow of blood reassessed. If the flow is comparable with that previously obtained, it can be assumed that injection has been satisfactorily accomplished. While the first x-ray films are being developed, the syringe is refilled with dye in preparation for a second injection.

The procedure is terminated after a second injection of 20 ml. Urografin. Should either injection prove technically unsatisfactory, a third injection may be given but not a fourth. If the renal vessels have been outlined, or damage to the aorta is suggested by outlining of the adventitial sheath, a third injection

must not be undertaken and it is usually preferable to repeat the aortogram at a later date.

Radiological Considerations.

It is possible to obtain reasonably good films of the arterial tree as far as the popliteal bifurcation (Fig.4). The aorta and iliac arteries are exposed on a cassette placed in a tunnel under the x-ray table using the Potter-Bucky diaphragm for better definition and films of both femoral arteries are taken by placing unscreened films below the thighs of the patient (Fig.2). Because of the area to be covered, a long tube distance (50 inches) is used and the operator is protected by a lead apron. There is some spread of direct radiation using this technique but measurement of the skin dosage has shown that the level of radiation is acceptable in a procedure which is not performed too frequently.

The exposure is a compromise between that required for visualising the trunk on the upper film and that required for the thighs which do not require such a long exposure. The x-ray factors normally used are 200 KV at 0.2 sec. and are varied according to the obesity of the patient and the degree of penetration of the preliminary films. The timing, which is different for

each injection, must be reasonably accurate and an experienced radiologist or radiographer is an essential member of the team.

For the first injection, the thighs are covered by a shield or lead apron to prevent exposure of the thigh films and the cassette is exposed as the last 2-3ml. are being injected, thus obtaining an 'immediate' aortic film. The cassette is withdrawn from below the table as quickly as possible and the lead apron covering the thighs is removed. A second exposure, taken at 5-7 seconds after the completion of injection, will usually outline the femoral arteries.

If these films show satisfactory visualisation of the aorta, a second injection is made and the aortic and thigh films are exposed simultaneously approximately 3-5 sec. after injection is complete. A guide to the early or late exposure of the second injection is obtained from the appearances of the lesions seen in the first films.

With this technique, aortic films are obtained at 0 and 3 seconds after injection and femoral films at 3 and 7 seconds.

The first aortic film usually outlines the aorta and iliac arteries as far as the upper end of the occlusion and the second aortic film demonstrates the

lower end of the lesion by dye which has passed through the collateral vessels to the reconstituted main channel. The femoral arteries will usually be outlined on the 3 sec. film where the lesion is small and on the 7 sec. film where the occlusion is extensive. A complete aortic block usually does not permit visualisation of the femoral vessels by this technique as the reconstitution of the arteries is considerably delayed.

Fig.5 illustrates the different appearances obtained in the first and second aortic films. Neither gives a complete record of the extent of the occlusion but from the two films a composite picture of the site and extent of the occlusion can be obtained.

The quality of aortic definition is usually excellent but the quantity of dye reaching the femoral vessels is often insufficient to give films suitable for reproduction. However, they usually give sufficient information to indicate whether the femoral and popliteal arteries are patent or occluded.

DIFFICULTIES AND COMPLICATIONS OF TRANSLUMBAR AORTOGRAPHY.

1. Technical.

Most of the difficulties encountered in translumbar aortography are technical in nature and to a large extent can be overcome through experience and

practice.

The most important difficulty is to ensure proper siting of the needle in the aorta. This is sometimes difficult in atherosclerotic aortas which are frequently smaller in diameter than a normal aorta. Because of the distance of the aorta from the surface and the necessity for relying on the sense of tactile perception, an off-central position of the needle is very likely. Provided that the lumen of the tip of the needle lies wholly within the lumen of the aorta, a satisfactory injection can be made, but an intramural position of the needle is more frequently encountered than in femoral arteriography and may lead to extravasation.

The unavoidable rotation of the needle which occurs during attachment of the tubing, and possible movement of the needle during the high pressure injection combine to produce a small number of failures. It is to minimise the possibility of this that the needle is rotated several times after puncture to ascertain that the flow is maintained in all positions.

Occasionally the aorta is not located by puncture at the usual site and the possibility of a high aortic occlusion must be suspected. The needle should then be directed upwards in the direction of the first

lumbar vertebra to puncture the aorta above the level of the renal vessels and a longer needle may be required in such cases. Needles of 16 cms. length should be available for use when there is a high aortic occlusion or when the patient is obese.

In addition to difficulties encountered in positioning the needle, the investigation may be vitiated by faulty radiological timing. The exposure of the first aortic film as the last 2-3 ml. of dye are being injected is normally automatic but may not be so when another person is introduced during holidays or sick leave. The timing of the late aortic film is more difficult, as allowance has to be made for the dye to traverse the collateral vessels and reconstitute the artery distal to the occlusion. To aid this calculation, it is helpful to have the first film developed before proceeding to the second injection.

2. Anaesthesia.

Many of these patients with atherosclerotic occlusion of a major vessel are poor anaesthetic risks and the hazard of the investigation has to be weighed against the need for obtaining precise information as to the site and extent of occlusion. As aortography is only essential where surgery is likely to be feasible,

it is unreasonable to undertake it when operation is not contemplated. Approximately one-third of patients with clinical evidence of aortic or iliac occlusion are not x-rayed because they are too old or are unfit for surgery.

Deterling (1952) found a constant circulatory reaction to aortography in both man and the experimental animal. He injected dye into 25 dogs and 9 patients and found that each developed a short period of hypotension and bradycardia lasting 3-6 secs. followed by hypertension and tachycardia of 1-5 mins. duration. These circulatory disturbances are slight and are not dangerous when the patient is fit for general anaesthesia. Although many patients had a history of coronary artery thrombosis, evidence of ischaemia on electrocardiography or marked hypertension, no major catastrophe or death has occurred in more than 100 aortograms.

3. Haemorrhage.

Before aortography became an established and acceptable procedure, the danger of major haemorrhage from the site of puncture was foremost in the writings of many critics. However, in spite of early criticism, the method has become established and although there are hazards, haemorrhage from the aorta is an unimportant

one. In 1952, Smith, Rush, and Evans performed 70 aortic punctures in moribund patients 6-24 hours before death and at subsequent post-mortem in 36 cases, only six showed evidence of minor degrees of bleeding. Reported deaths in World Literature due to haemorrhage are rare but McAfee and Wilson in 1956 reported 5 periaortic haemorrhages in 150 aortograms with 2 deaths. However, one patient died from haemorrhage subsequent to four diagnostic punctures of an aortic aneurysm and the other was caused by changing the patient's position from prone to sitting with the needle in situ.

In my own experience, there have been no patients in the present series in whom haemorrhage after aortography has been suspected on clinical grounds. The aorta is occasionally punctured while performing a paravertebral block on the left side, but no untoward effects have been noted. In some patients who have been operated on within a few weeks of aortography, some retro-peritoneal staining has been found but never evidence of a haematoma. The aorta and iliac vessels have been punctured at operation with a No.18 needle and in each case the bleeding was minimal and the site of puncture sealed rapidly.

4. Extravasation.

Extravasation of dye during aortography is not uncommon due to improper siting of the needle or to movement of the needle during injection. The commonest cause appears to be puncture of the right aortic wall owing to a penetrating rotatory movement produced in the tubing by the forcible injection and transmitted to the needle. This can be prevented by holding the tubing firmly during injection.

Peri-aortic extravasation produces the appearances seen in Fig.6. Absorption of the extravasated dye appears to occur fairly rapidly as a pyelogram due to kidney excretion can usually be seen within a few minutes on subsequent films.

The more serious type of extravasation is that which occurs into the wall of the vessel giving the appearance shown in Fig.7. The aorta is outlined by a dense shadow which stops abruptly. This intra-mural extravasation may distort the image of the vessel, and appears to induce dense fibrous adhesion between the aorta and neighbouring structures.

It has also been the apparent cause of further thrombosis in one patient.

A.McD., a female aged 44 years, was seen in 1959 when she complained of claudication of the right

leg at 100 yards distance. Both femoral pulses were normal at that time, but in 1960 her claudication distance deteriorated to 50 yards and reduction in volume of both femoral pulses was noted, suggesting an aortic bifurcation lesion. Aortography was performed in September, 1960 (Fig.7). This shows extravasation into the adventitial sheath with suggestive evidence of possible occlusion on the left side of the aorta just below the origin of the inferior mesenteric artery. She complained of severe pain on the following day and was able to walk only 10-15 yards before halted by claudication in both buttocks. Her condition improved a little thereafter and a second aortogram performed some weeks later (Fig.8), showed aortic occlusion from the origin of the inferior mesenteric artery to just above the bifurcation. This was confirmed when thrombo-endarterectomy was undertaken.

In retrospect, it seemed that the extravasation occurring during the first aortogram precipitated aortic thrombosis.

When extravasation of either type has occurred, the examination should be discontinued. Further injection is undesirable as this could lead to more damage and the procedure can easily be repeated at a

later date.

This precaution has usually been adopted and no serious ill-effects have been found to follow extravasation. These patients, however, appear to have more severe backache than is found after a straightforward aortogram and it is probably desirable to detain them for 48 hours instead of the usual 24 hours.

If there is doubt as to the siting of the needle it is desirable to give a small preliminary injection of 5 ml. dye and take an immediate exposure to determine the exact relation of the needle to the aortic wall. This precaution may be used routinely but it does not obviate the possibility of extravasation due to movement of the needle during attachment of the tubing or during injection.

5. Renal Complications.

Renal complications following aortography are much less common with low lumbar injection than in urological investigation where the aortic puncture has to be made above the level of the renal arteries. In the low lumbar injection, only a proportion of the dye is likely to pass into the renal arteries except in cases where the puncture is made at a higher level than intended. Puncture of the aorta above the level of the

renal vessels is, however, unavoidable in cases with high aortic occlusion. The forcible injection of 20 ml. of dye is most likely to give rise to renal lesions when the point of the needle is opposite the opening of the right renal artery and the right kidney may suffer severe damage under such circumstances.

Fig.9 illustrates the possibility of cannulating the renal artery, or, in this case, an aberrent one and the operator, who believes the needle to be sited in the aorta because of the satisfactory reflux, injects the dye directly into a renal vessel.

There have been no cases in this series with major renal damage. A few patients have shown evidence of haematuria on microscopic examination of the urine and the patient, in whom the aberrent renal artery was cannulated, produced a few casts.

Crawford and his associates (1957) reported 31 cases of renal damage in a series of 300 aortograms. The mortality rate in those developing renal damage was 20% and the lesions produced were bilateral. At post-mortem, they found disorganisation of glomeruli and tubular necrosis. They also tabulated the cases previously reported in the literature and noted a definite association between the presence of renal damage and the use of sodium iodide or large quantities

of organic iodine compounds. For this and other reasons, they advocated that the use of sodium iodide be discontinued and the repeated injection of large volumes of dye should be avoided. These precautions are generally accepted today.

6. Iodism and Allergic Reactions.

Iodism used to be a hazard in the earlier days of aortography when sodium iodide was used (Legueu, 1931), but with modern organic triodide compounds it has virtually been eliminated.

Allergic reactions, however, do occur and may occasionally prove fatal. Pendergrass and his colleagues (1958) have collected reports of fatal reactions in the United States and estimate the fatality rate as 1: 120,000 injections. Death has occurred in spite of preliminary testing for allergy, and Pendergrass considers that these tests are of no value.

It is a routine practice in the Unit to inject a test dose of 1 ml. dye intravenously a few minutes before aortography and no evidence of iodism or allergy has yet been encountered.

7. Damage to other Organs.

There have been various reports of paraplegia, mesenteric thrombosis and death owing to injection directly into the lumbar or mesenteric arteries. None of these complications has been encountered in the present series.

Crawford and his associates (1957) collected 13 reported cases of paraplegia following aortography but did not have any in their own series. This is a possible complication should the dye be injected in high concentration into a lumbar artery but again appears to have been most commonly associated with sodium iodide. Crawford and his colleagues reported four cases of superior mesenteric artery thrombosis following the use of sodium iodide and all four cases died. They also reported two cases of embolism with one death. Such complications are most liable to occur when sodium iodide is used. They are unlikely to occur when the modern contrast agents are used and when injection is performed below the level of the renal arteries.

Crawford (1957) reported one case in which the inferior mesenteric artery was found to be subsequently thrombosed at operation. No untoward effects on the bowel were noted and this accords with the observation that operative damage to the artery or its

ligation during excision of an abdominal aneurysm does not necessarily produce gangrene of the colon. The blood supply is maintained through the marginal artery which derives an adequate arterial supply from the middle colic branch of the superior mesenteric artery (Fig.88) and the middle and inferior haemorrhoidal arteries.

DESCRIPTION OF THE NORMAL AORTOGRAM.

With the technique described above, the arterial tree can be visualised from the lower abdominal aorta to the popliteal bifurcation. Opportunities for study of the normal aortogram have not been frequent as most examinations have been performed where occlusion of one of the major vessels was present.

Fig.10 is an illustration of an aortic film where the vessels were relatively normal in appearance. The aorta and iliac arteries are larger than most of the atherosclerotic vessels encountered and although some of the major branches are visible there are no anastomotic ramifications as in cases with arterial occlusion. In this figure, most of the dye has left the aorta and the kidney shadows are seen at the level of the needle. A denser area at the tip of the needle indicates a small patch of extravasated dye in the

aortic wall. On the right, there are two lumbar arteries and on the left, the inferior mesenteric artery crosses a prominent left lumbar artery. A faint outline of the marginal artery of the colon can be seen with terminal branches running from it. The middle sacral artery is not seen. Both common iliac arteries are of large diameter and the internal arteries can be seen although the right one takes origin at a high common iliac bifurcation and is partly hidden by the external iliac artery.

The course and pattern of the branches of the aorta and iliac arteries are best seen in the abnormal aortogram where occlusion is present and the remaining patent branches are hypertrophied and ramify extensively to form anastomoses. Fig.7 gives an excellent picture of the inferior mesenteric artery and the terminal branches from the arcades to a colon containing faeces. Another aortogram in the same patient (Fig.8) illustrates the anastomosis between the ilio-lumbar branch of the internal iliac artery and a right lumbar artery, also the anastomosis between the terminal branches of the superior haemorrhoidal and the middle and inferior haemorrhoidal arteries.

The aorta bifurcates about the level of the fourth lumbar vertebra (Gray,1962) although this may

take place at a higher level in some patients and give rise to technical difficulties of access at operation. The common iliac artery courses downwards and laterally along the brim of the pelvis without any named branches although occasionally small twigs are found at operation, and terminates in front of the sacroiliac joint at the level of the lumbo-sacral disc by dividing into external and internal iliac arteries.

The external iliac artery continues the line of the common iliac artery and courses along the pelvic brim to pass behind the inguinal ligament into the femoral canal where it becomes the common femoral artery. Before its termination, it gives off the deep circumflex iliac and deep epigastric arteries (Fig.93).

The internal iliac artery descends into the pelvis to supply the pelvic wall and viscera and makes important anastomoses with the lumbar arteries and inferior mesenteric artery, with branches of the femoral artery, and with the internal iliac artery of the opposite side.

B. FEMORAL ARTERIOGRAPHY.

The technique of performing this investigation has been developed and simplified since arteriography was first employed by Brooks (1924). The development of the less irritant organic group of contrast agents permitted a further advance in technique, which allows the whole procedure to be carried out under local anaesthesia. As experience of arteriography developed, percutaneous puncture of the femoral artery was adopted and this is now performed either by injecting dye through a needle introduced into the femoral artery or through a cannula fitted around a needle which is withdrawn after puncture is achieved. Percutaneous puncture with injection through a needle has been the method employed in a personal series of several hundred arteriograms with satisfactory results.

Materials. (Fig.11).

A short bevelled No.1 serum needle of 0.75mm. internal diameter is connected to a 12 inch length of polythene tubing using Luer-Lok connections.

A 20 ml. interchangeable "Summit" syringe with Luer-Lok fittings is filled with 20 ml. of 60% Urografin.

A 10 ml. record syringe is filled with 5 ml.

2% Planocaine P and fitted to a hypodermic needle.

Two 20 ml. Luer-Lok syringes are filled with sterile normal saline, of which ½ pint is poured into each of two sterile basins for rinsing purposes.

Technique.

The patient, who has been given 50 mgm. pethidine half an hour previously, is placed in the supine position on top of the special cassette tunnel on the x-ray table (Fig.12) and the appropriate leg is placed so that the desired area will be exposed on the x-ray films. A preliminary film may be taken to determine exposure factors but an experienced radiologist will omit this film, the factors being varied according to the thickness of the limb. The three trays of the cassette tunnel are loaded with unscreened films which overlap behind the knee, and a test dose of 1 ml. 60% Urografin is given intravenously.

When the operator has scrubbed up, he swabs the patient's groin, which has previously been shaved, with skin antiseptic and the area is draped with sterile towels. The pulsating femoral artery is palpated and a small skin wheal is raised with local anaesthetic at a point 1 cm. below the inguinal ligament over-lying the artery and 2 ml. are infiltrated around the artery.

The plastic tubing and needle are attached to a syringe and carefully filled with saline to exclude all air. When the skin is punctured in the centre of the wheal, the syringe is disconnected and the tubing remains filled.

The femoral artery is very mobile, especially in thin persons, and the needle tends to slide round the side of the artery. Accordingly, an attempt is made to immobilise the artery with two fingers placed on either side of the vessel and the needle is directed upwards at an angle of 60-70 degrees to reach the artery just below the inguinal ligament where it is least mobile (Fig.13). The middle and index fingers of the left hand are placed vertically with ½ cm. gap between them, and pressed backwards so that the artery is fixed and pulsation is felt equally in the two fingers. If the needle is gently but firmly advanced between the fingers, the pulsation of the artery is transmitted to the right hand when the needle point impinges upon it and steady pressure pushes the needle through the anterior wall yielding a rapid flow of blood into the plastic tubing.

The saline syringe is reattached and, by the intermittent injection of saline, the quality of the reflux is estimated and the patency of the needle maintained (Fig.14). A strong pulsatile flow which pushes back the piston of the syringe indicates that the needle

is properly positioned within the lumen of the artery.

The advantages conferred by the use of plastic tubing between the needle and the syringe are threefold:- movements of the syringe are buffered by the flexible tubing and are not directly communicated to the needle, thus minimising the possibility of dislodging the needle from its position within the artery; the clear tubing permits an evaluation of the quality of arterial reflux which the use of syringe and needle alone would not; and thirdly, the operator's hands can be held outside the direct beams of x-rays and protected by a horizontal screen interposed between the x-ray tube and the hands.

To ensure that the point of the needle will retain its position during injection, a second 20 ml. syringe containing saline is attached to the tubing when the first has been emptied and the forcible injection of several ml. saline will confirm that the needle is unlikely to become dislodged during dye injection. The extra time afforded by the use of this second syringe allows the radiologist and other members of the team to prepare for the dye injection.

When the radiologist indicates his readiness, the syringe containing 20 ml. 60% Urografin is connected to the tubing and injection is made using the maximum

hand pressure possible. A certain amount of lateral tremor is likely during this manoeuvre but forward movement of the syringe is avoided. Because the needle is directed upwards, the dye is forced against the arterial stream and results in the replacement of a column of blood by a column largely composed of dye. The faster the syringe is emptied, the more concentrated is the dye column and the better the quality of the film obtained. Slow injection will yield poor films, and when the dye reaches the lower leg there may be insufficient to outline the tibial vessels satisfactorily.

The average time required to inject 20 ml. of dye through a No.1 serum needle of 0.75 mm. internal diameter is 6 secs. This gives a needle velocity of :

$$\frac{\text{Volume of Dye}}{\text{A x time in secs.}} = \frac{20}{0.0044 \times 6} = 757.6 \text{ cm/sec.}$$

$$= 17 \text{ M.P.H.(Approx.)}$$

A = cross sectional area of needle

$= \pi r^2$

$= \pi \left(\frac{0.75}{2}\right)^2$

= 0.44 sq.mm. = 0.0044 sq.cm.

This is equivalent to a speed of about 17 miles per hour and results in some distension of the artery at the site of injection, producing pain of short duration. This is followed by pain in the leg due to the irritant action of the dye on the vessels. This is usually described as a burning sensation passing down the leg, the level to which the pain is felt depending on the site of arterial occlusion. Phlegmatic patients experience only slight discomfort, but apprehensive patients complain of severe pain and attempt to flex the leg to obtain ease. Movement would spoil the x-rays obtained and is avoided as far as possible by warning the patient before-hand that the burning sensation is to be expected, and by reassuring him that it is harmless. In addition, a light retaining strap is placed over the knee to minimise movement.

It is commonly noted after injection of dye that a flush develops in the skin of the thigh and lower leg due to vaso-dilatation and lasts for a few minutes. The level to which the flush reaches is an indication of the site of arterial occlusion and, in femoro-popliteal occlusion, it usually extends to just below the level of the knee joint.

When the dye injection is complete, and the radiologist has taken the desired exposures, 20 ml.

saline are injected into the femoral artery to aid in dispersal of the dye and the needle is withdrawn. The reflux at this stage should be the same as before injection, and a poor or absent reflux indicates that the needle has moved and that extra-arterial injection has probably occurred. Digital pressure over a swab for a minute or two will allow the artery to become sealed and a small dressing is applied.

The same technique is employed in the other leg and the procedure is then complete. The patient is able to rise and is sent back to the ward where he remains in bed for a few hours before resuming normal mobility.

Radiological Considerations.

With this method, it is possible to obtain only one film unless special radiographic methods are used. Automatic cassette changers may be used, but a more simple device has been used in the present series which has provided adequate information and good quality films.

This is a manually operated cassette changer (Fig.12) which consists of a plywood tunnel containing three plywood trays each with a lead screen in the base. Films are placed in the trays and when the top one is exposed the tray is extracted and the second film is

ready for exposure at whatever time interval is desired. Removal of the second tray leaves the third film ready for exposure.

Each tray contains two unscreened Kodak films which overlap behind the knee and the films are numbered 1, 2 and 3 with a graphite pencil. The right and left legs are identified by a lead marker which is fixed to the roof of the cassette tunnel. Any discrepancy in the size of the images recorded on the three films is minimal (approximately 1%) because of the long tube distance (50 ins.) employed.

With this tube distance the x-ray beam will cover a large area and this is therefore screened by a pyramidal lead and plywood cone of rectangular base which limits the beam to the area of the cassette tunnel (Fig.12). For further protection, the surgeon and assistant normally wear a lead apron of standard type. The exposure dose on the operator's hand has been tested and found to be minimal.

The radiological factors involved in the average patient are 300 mA's at 75 KV for 0.2 sec. but these are varied by the radiologist as required.

Timing.

The timing of the three exposures can be

varied according to the part of the leg one wishes to visualise. In practice, the first two exposures are made at standard intervals and only the third is varied.

The first film is exposed as the last 2-3 ml. of dye are being injected and the second is exposed as soon as the first tray has been removed from the cassette tunnel. These usually give satisfactory films of the thigh and, where the occlusion is not a long one, the arterial tree down to and including the popliteal bifurcation (Figs.15 & 16) is visualised.

The timing of the third film is varied according to an assessment of such factors as the estimated site and severity of arterial occlusion, the efficiency of the collateral circulation and the expected rate of blood flow. This is assessed from the pattern of the pulses, the severity of ischaemia, the temperature rise in the reflex vaso-dilation test, and the age and cardiac condition of the patient. The radiologist is then advised as to the delay in timing expected, and he usually exposes the third film between 5 and 8 seconds after injection.

At this stage, most of the dye has left the thigh (Fig.15) but the timing of this third film is important for adequate visualisation of the vessels of the lower leg. In fig.16, the posterior tibial and

peroneal arteries are visible throughout most of their length on the third film. Should one wish to visualise the vessels in the region of ankle and foot, the exposure of the third film requires to be further delayed.

DIFFICULTIES AND COMPLICATIONS.

1. Technical.

Most of the technical difficulties encountered with this method can be eliminated with care and experience, and films of a quality suitable for photographic reproduction can be consistently obtained.

There are many minor mishaps which may cause inconvenience. Even using Luer-Lok fittings, occasional detachment of the tubing from syringe or needle may occur.

If there is a prolonged delay between filling the syringe with dye and injection, the piston may stick owing to the viscosity of the dye. This can usually be prevented by rinsing the syringe with saline before use, and by using a freshly filled syringe for the second leg.

Movement of the leg during injection may occur when the patient experiences pain. A nervous patient will describe it as excruciating and will attempt to move his leg. Reassurance before-hand and the use of the restraining strap help to prevent or minimise

movement.

Sometimes, when the artery has been pierced but reflux is unsatisfactory and the needle is withdrawn for re-insertion, there is some leak from the artery and the needle blocks with clot when re-inserted. When this happens, the needle may be in the artery but no reflux occurs, and this may lead to repeated trauma to the vessel. Should this occur, it is wise to re-insert the needle above or below the original site in order to approach the artery through fresh tissue. It is thus important to achieve arterial puncture at the first attempt because, as time passes, each attempted puncture is less successful than the last. For this purpose, the needle must be sharp and have a short bevel, and the artery is most easily located if it is fixed by a finger on either side.

There is sometimes difficulty where the patient is thin, very stout, or where the artery is very sclerotic. In thin patients, there is little surrounding fat and the artery tends to roll away from the needle point; in obese patients, there is difficulty in locating the artery which is situated deeply and pulsation is difficult to feel; and in very sclerotic arteries, the wall is thick and difficult to penetrate, the needle tending to slide away from the vessel.

Puncture of the femoral vein may occur where the needle has inadvertently been pushed too far medially. This is indicated by a slow dark non-pulsatile reflux of blood. Should this occur, the needle is withdrawn and pressure is exerted over the site for a minute to allow the vein to seal before arterial puncture is again attempted.

2. Extravasation.

This is the commonest complication encountered and its frequency varies inversely as the experience of the operator. With practice, it is found to occur in fewer than 5% of examinations. It is caused by malposition of the needle within the artery, by the use of a needle with too long a bevel, or by an injudicious movement communicated to the needle during injection.

It leads either to extra-arterial injection (Fig.17) or intra-mural injection with stripping of the adventitious coat of the artery (Fig.18) and is very painful. The patient complains of severe pain and becomes shocked. He is pale, has a cold clammy skin, perspires freely and develops tachycardia. His condition quickly improves but it is usually unwise to perform further arteriography and he is returned to the ward.

In most cases, there are no permanent ill-effects and the procedure can be repeated in a month's time.

3. Local Thrombosis.

Thrombosis of the femoral artery at the site of arterial injection is an unusual sequel and there has been only one case in this series.

R.B., a male aged 42 years, was first seen in October, 1957 when he complained of claudication in the left calf at 200 yards distance which had been present for four months. The left femoral pulse was easily palpable but none of the distal pulses could be felt.

Arteriography resulted in partial extravasation but sufficient dye entered the artery to demonstrate an occlusion of the proximal part of the popliteal artery due to atherosclerosis. A small haematoma was present in the groin and he had a left paravertebral block performed with phenol on the following day. When he reported back a month later, the left femoral pulse could not be felt although external iliac pulsation was felt above the inguinal ligament and it was considered that he had developed a local thrombosis at the site of arteriography in addition to his popliteal occlusion. Fortunately, he had an excellent

response to paravertebral block with complete relief of claudication and was able to continue normal work as a butcher. This excellent result in spite of local thrombosis was probably due to the abundance of healthy collateral vessels present in a patient of his age.

4. Distal Thrombosis.

Although the modern tri-iodide compounds are superior to sodium iodide because of their low toxicity and less irritant properties, they do not constitute the ideal dye which would be completely non-irritant. In arteriography, dye reaches the distal arteries in fairly high concentration which may produce thrombosis, although this has been infrequent even in patients with gangrene.

As in aortography, there is a limit to the amount of dye which should be injected. In bilateral femoral arteriography, 40 ml. of 60% Urografin are used, and, if for any reason, a second injection is required on either side a further injection of 20 ml. can safely be given. A total dosage of 60 ml. for a bilateral examination has been well tolerated in all cases in which it was necessary, but it is probably unwise to exceed this amount.

Because of the dangers of further thrombosis,

patients with incipient or established gangrene seldom had arteriography performed prior to 1961, but since then an increasing number of these patients have undergone arteriography in the hope of finding lesions suitable for arterial surgery. In most cases, there has been no deterioration which could be ascribed to arteriography but in two patients the sequence of events is interesting.

J.T., an elderly man of 72 years was first seen in 1957 with incipient gangrene of his left foot. Because of the possibility of early deterioration, he was given a paravertebral phenol block on the following day with a good temperature response and the condition of the foot improved. Rest pain was relieved and the foot appeared healthier. Arteriography revealed a short occlusion in the region of the adductor opening and both tibial arteries were occluded, the peroneal being the sole source of blood supply to the foot (Fig.19). Deterioration followed arteriography and he developed established gangrene of the fourth left toe within a few days. However, this remained localised and in the course of the next few months the toe separated and healed. It is of interest to note that he developed a similar condition of the right foot in 1961 and, following a satisfactory phenol injection, gangrene of

the tip of the right fourth toe separated and healed.

The deterioration which occurred so soon after arteriography suggested that the injection of dye was a major factor in the production of gangrene.

W.McL., a man aged 64 years, was admitted in October, 1960 for investigation of claudication of the right leg at 50 yards distance which had been present for four months. Arteriography was performed and he was found to have a popliteal occlusion. Paravertebral block with phenol was performed on the following day and the foot became warm. On the same evening, he developed a pain in the chest with pyrexia and did not look well, but there was no fall of blood pressure to suggest coronary artery thrombosis and x-ray of chest was negative. On 6th October, 1960, two days after arteriography, the right leg became cold and cyanosed and a further arteriogram showed that his occlusion had extended and the entire popliteal artery was occluded. His general condition remained poor and on 12th October, 1960 he developed a hemiplegia and died. Permission for post-mortem was not obtained.

The sequence of events in this patient are difficult to elucidate. He appeared to have an exacerbation of an existing chronic bronchitis after

arteriography and phenol block and had two further thrombotic incidents. It is possible that the extension of the popliteal occlusion which led to gangrene was precipitated by arteriography but, as the second occlusive incident was cerebral, it is possible that the leg thrombosis was coincidental and part of a terminal illness.

Apart from these two cases, arteriography has been successfully performed without deterioration in other ischaemic limbs.

5. Iodism and Allergy.

As in aortography, there have been no patients developing signs of iodism or of allergic reaction to Urografin. 1 ml. 60% Urografin is administered routinely by the intravenous route a few minutes before performing arteriography and no reactions have resulted. Those patients, in whom arteriography has been repeated at intervals, have had no allergic reactions.

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From this experience, during which many arteriograms have been performed with relatively few incidents, it is considered that femoral arteriography is a safe procedure. The method employed is easily

performed and yields good quality films of both thigh and leg.

Visualisation of the vessels of the ankle and foot has not been undertaken very frequently by this method, because it is considered that the information which would be obtained is not as important as that derived from thigh and lower leg films. It is possible to obtain films of the foot by taking three late exposures, in one of which the plantar and digital arteries can be seen.

DESCRIPTION OF THE NORMAL ARTERIOGRAM.

Because most of the patients investigated have arterial changes of atherosclerosis, few normal arteriograms have been seen. However, some patients investigated for possible arterial changes have been found to have normal vessels.

Fig.20 shows the appearance of a normal femoral artery, the most striking feature being a well-filled main channel with a few small poorly outlined branches. This is in contrast to the numerous collateral vessels found in most cases of arterial occlusion (Fig.41).

The division of the common femoral artery occurs at a high level and its course is short before it

divides into superficial and deep arteries. The division is difficult to see as films are exposed to give the greatest detail in the middle and lower thigh and the degree of penetration at the groin is usually insufficient to yield a good picture at this level. The branches of the superficial femoral artery are small and few in number, whereas the profunda artery has numerous branches. The lateral circumflex artery can be seen in the film of the left thigh (Fig.20) as it passes laterally but the artery, which accompanies the nerve to vastus lateralis, is superimposed on the femur and is less readily visible. This branch is much better demonstrated in Fig.78 where the collateral circulation has developed owing to an occlusion in the adductor region. The lower branches of the profunda are muscular and perforating arteries, but cannot be named separately because of the difficulty in distinguishing them in a film taken in one plane.

In the lower part of the thigh, the superficial femoral artery passes through the adductor opening to become the popliteal artery. The site of this opening may be difficult to define but the descending genicular artery or its saphenous branch may be visualised and as this is usually given off before the adductor opening (Gray,1962) it can be assumed that the

adductor opening lies immediately below this branch (Fig.60).

In the lower leg films of a normal femoral arteriogram (Fig.21), the popliteal artery can be seen to give off the sural branches which supply the gastrocnemius and other muscles. At a variable distance below the level of the knee joint, the popliteal artery divides into anterior and posterior tibial arteries, the former passing between the tibia and fibula above the interosseus membrane before coursing down the front of the leg. In a few cases, the popliteal bifurcation may be high and the posterior tibial arises at (Fig.22) or just below (Fig.23) the level of the knee joint, the main artery giving rise to anterior tibial and peroneal arteries lower down. In the A-P film, it is sometimes difficult to see the anterior tibial artery because it is superimposed on the dense shadow of the cortex of the tibia. In Fig.21, the tibial recurrent artery can be seen passing upwards towards the knee joint.

The posterior tibial artery passes downwards for a variable distance before giving off its peroneal branch and courses medially to reach the ankle behind the medial malleolus. The peroneal artery descends and inclines somewhat laterally until it reaches the ankle where it breaks up into a number of anastomotic branches,

including the perforating peroneal branch which passes to the front of the leg to anastomose with the anterior tibial. The termination of this artery is best seen in the abnormal arteriogram (Fig.19). Although the shadow of the peroneal artery is also superimposed on the tibia, it usually overlies the less dense shadow of the medulla and can be traced downwards with relative ease (Fig.21).

A lateral view of the lower leg (Fig.24) may be used to demonstrate the course of the posterior tibial artery, the shadow being projected behind the tibia and fibula, and, in this illustration, it can be seen passing behind the medial malleolus to form the plantar arteries. However, the anterior tibial and peroneal arteries may be difficult to see in this view and the A-P position, which is more comfortable for the patient, is the one which is normally used.

The peroneal artery is a very important source of supply to the lower leg and may sometimes outrank the posterior tibial in size and importance. Figs. 23 & 25 illustrate peroneal arteries which by virtue of their size and direction appear to be the direct continuation of the main trunk and the posterior tibial appears to be a lateral branch.

Femoral Vein Filling.

With this method of arteriography, three films of thigh and three of lower leg are obtained (Figs.15 & 16). The third thigh film does not usually demonstrate the arterial pattern well because most of the dye has passed into the lower leg. In some cases, however, the femoral vein contains sufficient dye to produce a well-defined outline on the third film (Fig.26). In this illustration, the valveless main channel is seen together with numerous branches which are frequently double, i.e. venae comitantes.

Fig.27 illustrates the three films of the right thigh in one patient. The first film shows a short occlusion at the adductor opening with a large collateral arising from the unoccluded lower end of the superficial femoral artery. In the second, the arterial pattern is indistinct and the hazy outline of numerous small vessels is seen. The third film shows a striking picture of venous filling with numerous paired branches and filling of the femoral and profunda veins in the upper part of the thigh.

In the illustration of the arteriogram of the same patient's left thigh (Fig.28), only the first and third films are shown. Both the superficial femoral and profunda veins are seen and the pattern of

distribution of arteries and veins corresponds very closely.

In the many films in which femoral vein filling is seen, the third exposure has been taken at the normal timing of 7 seconds, yet the dye is densely concentrated in the femoral vein. This seems too short a time for sufficient dye to have passed through the capillary bed and the possibility of arterio-venous shunts must be considered. These were demonstrated in the foot of 2 out of 13 legs amputated for atherosclerotic gangrene (Edwards, 1959) but have not been described in the thigh. Whether this is the explanation for the venous filling is not known and the possibility appears to require further investigation.

CHAPTER FOUR.

ARTERIAL CHANGES IN ATHEROSCLEROSIS.

CHAPTER FOUR.

ARTERIAL CHANGES IN ATHEROSCLEROSIS.

From the moment when the vascular system differentiates from the primitive mesenchyme, the arterial wall undergoes continous changes during development, maturation, and ageing. During development the arterial wall adapts itself to the stresses engendered by pulsation and arterial pressure and Robertson (1960) has demonstrated cushion-like thickenings composed of longitudinal muscle and elastic tissue in the walls of the popliteal, lower brachial, and coronary arteries of the foetus. These occur mainly at the mouths of branches and he considers that they are a physiological reaction to a pulsatile longitudinal stress. He observed this process continuing during the first two decades of life and noted that these muscular cushions became especially prominent at the mouths of branches of the femoral and popliteal arteries. With increasing age, the internal elastic lamina of these cushions commenced to split and the musculo-elastic intimal layer became nodular followed by vascularisation and subsequent deposition of lipid.

This process is accelerated in atherosclerosis, in which the predominant lesion is the formation of

intimal plaques containing lipid. At the same time, there is progressive medial fibrosis and calcium may be deposited in the media or in the intima.

The effects on the artery are the resultant of the luminal narrowing caused by intimal thickening and plaque formation; weakening of the wall due to medial fibrosis; constriction produced by peri-arterial fibrosis; and rigidity caused by calcification.

The relationship between these factors and arterial flow can be studied on the arteriogram.

1. Intimal Thickening and Plaque Formation.

Intimal thickening is usually most severe in the posterior wall of an artery and leads to narrowing of the lumen and a consequent reduction in blood flow. As the volume of flow is proportional to the fourth power of the radius, a small decrease in diameter has a more serious limiting effect than might otherwise be expected. Intimal thickening leads to irregularity of outline of the vessel on the arteriogram and tends to become more severe with the passage of time (Fig.29).

Plaque formation can be recognised on the arteriogram by the presence of localised stenosis or by a decrease in density of the dye column. These changes are illustrated in Fig.30 where plaques are seen in the

adductor region of the superficial femoral artery and in the popliteal artery - two sites at which they frequently develop.

Although the volume of blood flow in the vessel is reduced by these changes, there is an increased rate of flow through the stenosed area which militates against the deposition of thrombus and in some cases the artery may remain patent in spite of stenosis. Fig.31 shows an aorta with symmetrical narrowing at the aortic bifurcation and obliteration of the lumbar and inferior mesenteric arteries due to extensive atherosclerosis. This lesion has not progressed during the past three years and Fig.32 illustrates severe bilateral adductor stenosis in a man of 74 years of age which has also not progressed to occlusion.

In other cases, fibrin is deposited on an atherosclerotic plaque leading to progressive narrowing and ultimate occlusion (Fig.33). This process is frequently seen in the lower abdominal aorta at operation when pale gelatinous thrombus loosely adherent to the intima has to be removed, and it is common when there is calcification and ulceration of the plaque.

The final thrombosis and occlusion may take place when the circulation rate is slowed e.g. during bed rest, or when a portion of fibrin separates and

blocks the nearly occluded vessel. Local factors determine whether there is occlusion of the main vessel itself or obliteration of side channels only (Fig.31).

If the main vessel becomes obliterated by thrombosis (Fig.29), thrombus is propagated as far as the nearest proximal branch. If the rate of flow into this branch is sufficiently high to overcome the local factors favouring thrombosis, occlusion will be halted at this level but, if the vessel is small and the flow rate is poor, it may be overwhelmed by thrombosis which will continue to spread until a branch is reached whose rate of flow is sufficient to limit the occlusion.

Similarly, thrombosis extends downwards until a satisfactory rate of flow from a major branch prevents further extension. When thrombosis is halted, the occlusion is bounded above by a branch (the 'limiting' collateral) which anastomoses through tortuous connections with the branch below the occlusion (the re-entrant collateral). There is a reversal of flow in the latter vessel (Longland, 1953) which carries blood back into the main channel. Fig.29 illustrates the limiting and re-entrant collaterals (derived from branches visualised prior to occlusion) connected by a network of collateral vessels.

2. Dilatation.

Although progressive narrowing and eventual occlusion is the usual sequence of events, weakening of the arterial wall due to medial fibrosis may predominate and lead to early dilatation. One very striking appearance occasionally found on arteriography is the 'beaded' appearance which results when dilatation and stenosis alternate at short intervals (Fig.34). This is to be differentiated from the 'beaded' or crenated appearance described by Wickbom and Bartley (1957). This may appear during arteriography by the Seldinger technique due to the development of static pressure waves (Theander, 1960) and is different in appearance to the fixed deformity of the arterial wall shown in Fig.34.

A more advanced degree of this condition is illustrated in Figs.35 and 36. This type of vessel with a lumen of variable diameter develops a turbulent flow and it is believed that these arteries are liable to early extensive thrombosis which may endanger the viability of the limb. The artery illustrated in Fig.34 thrombosed 18 months after the arteriogram was performed and a mid-thigh amputation was required. The artery illustrated in Fig.36 thrombosed six months after the first arteriogram. This patient, who was only 45 years

old, developed severe intermittent claudication and ischaemic rest pain, amputation being averted by sympathetic block.

Fig.37 illustrates a vessel in which multiple dilatation of a more severe degree has occurred and early aneurysmal dilatation is present. In other cases, dilatation is eccentric leading to the formation of a saccular aneurysm (Fig.38).

Dilatation and aneurysm formation frequently affect the lower abdominal aorta where arterial pressure is high, and Fig.39 shows early aneurysmal dilatation of the aorta. In these cases, the aortic wall is often found to be split into two layers (intimal lining and attenuated media) by extensive circumferential deposits of pultaceous cholesterol debris.

Arterial dilatation may also be seen in the post-stenotic dilatation which occurs beyond a stenosed segment (Fig.40). This is generally believed to be due to the turbulence produced when a rapidly flowing stream of blood through the stenosis reaches the wider lumen beyond and produces an increased lateral pressure which leads to dilatation of the weak arterial wall. Robicsek and his colleagues (1958), however, showed that increased lateral pressure did not occur beyond the stenosis and they ascribe the dilatation to the effects of turbulence

and cavitation on a weak arterial wall.

Should arterial occlusion eventually develop, the dilatation may persist distal to the occluded segment (Fig.41), indicating that thrombosis has extended proximally.

3. Fibrosis.

In the atherosclerotic artery, fibrosis of the media leads to weakness of the arterial wall, but fibrosis also occurs outside the vessel producing adhesion to neighbouring structures. The degree of fibrosis is proportional to the severity of the atherosclerotic changes and it is most severe in relation to the posterior wall of the artery. Dense fibrotic adhesions may develop between the aorta and inferior vena cava and behind the right common iliac artery as it crosses the left common iliac vein. Operative dissection may be difficult and, in the surgery of abdominal aneurysm, it is safer to excise the anterior and lateral walls of the sac leaving the posterior wall in situ than to try and separate it from the vena cava.

In rare cases, fibrosis may play a major part in constricting an artery and its excision at operation may relieve stenosis and permit improvement in blood flow. Only one case of severe perivascular

fibrosis has been operated on.

H.S., a 32 year old woman, complained of bilateral claudication at 100 yards and, on aortography, was found to have narrowing of both common iliac arteries. At operation, there was no occlusion but dense fibrous tissue surrounded both arteries and appeared to constrict them. When the fibrous tissue was excised, the arterial diameter returned to normal and her claudication was relieved. Three years later, she developed severe left sided claudication due to occlusion of the external iliac and common femoral arteries and also had evidence of bilateral renal artery stenosis. The common iliac arteries, however, were still patent.

Occasionally, localised dense periarterial fibrosis has been found which has led to difficulty in mobilising the aorta. In these cases, intra-mural extravasation of dye has occurred at the corresponding site during aortography and is believed to have caused the fibrosis. Extra-mural extravasation has not resulted in a similar degree of fibrosis.

4. Calcification.

Calcium is deposited in long-standing atherosclerotic plaques and favours the development of

ulceration and thrombosis. It is, however, seldom extensive except in Monckeberg's sclerosis where calcification of the media occurs without intimal lesions and the artery remains patent.

In atherosclerosis, Lindbom (1950) has pointed out that calcification tends to support the arterial wall and maintain patency whereas uncalcified portions of the same vessel show narrowing of the lumen. Harrison (1933) showed this experimentally in rabbits. He treated rabbits with large doses of calciferol to produce medial calcification then gave them standard cholesterol feeding to induce atheroma. He found that the portions of aorta which remained mobile developed cholesterol deposits while the rigid calcified areas remained free and he also found that the greatest deposition of cholesterol occurred at the margins of the calcified areas.

Fig.42 supports these findings as the diameter of the lumen is maintained in the calcified region and severe narrowing without calcification occurs lower down. Fig.43 illustrates a proximal occlusion of the superficial femoral artery with calcification in the adductor region, and the luminal diameter is normal at the calcified areas and reduced elsewhere.

Evidence of calcification on a straight

x-ray of the lower leg is often regarded as confirmatory evidence of arterial occlusion. This supposition is frequently erroneous as extensive calcification is due to Monckeberg's sclerosis in which the artery is patent, and even in severely calcified atherosclerotic vessels it is usually possible to demonstrate some degree of patency (Fig.44).

CHAPTER FIVE.

THE DEVELOPMENT AND LOCALISATION OF INTIMAL PLAQUES.

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THE DEVELOPMENT AND LOCALISATION OF INTIMAL PLAQUES.

The principal lesion in atherosclerosis is the intimal plaque and arterial occlusion develops at the sites of plaque formation. These lesions show a predilection for certain specific portions of the arterial tree in the lower limb which may be classified as : -

1. The Adductor Region.

This region comprises the adductor canal and opening and is the commonest site for arterial occlusion in the lower limb (Fig.29). - Leriche and Bertrand, 1946.

2. The Bifurcations of the Large Arteries.

Plaque formation is common at the bifurcations of the large arteries - i.e. aortic, iliac, popliteal. Examples of lesions at these three sites are illustrated in Figs.45, 46, and 47.

3. At the Origins of Lateral Branches and Collateral Vessels.

Plaque formation at the origin of a large branch is frequent and collateral vessels may become narrowed at their origin by involvement in the atherosclerotic process (Fig.48).

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In the lower limb, atherosclerotic lesions affect mainly the larger arteries and Dible (1956) showed that the peroneal and plantar arteries were relatively free from severe atherosclerotic changes in limbs amputated for gangrene. This accords with the evidence of Young and his colleagues (1960) who showed that the degree of atherosclerotic change in an artery was proportional to its radius.

The distribution of lesions in the larger arteries is not uniform and their location is probably determined in some way by local mechanical factors (Duguid and Robertson, 1957).

The development of lesions at specific sites may be due to alterations in the mechanics of blood flow at these sites, to changes in the arterial wall, or to a combination of both mechanisms.

In 1962, Murphy, Rowsell, Downie, Robinson, and Mustard studied the pattern and distribution of thrombotic deposits in mechanical models and found that they were strikingly similar to the pattern of incipient atherosclerosis at comparable sites in the arterial system. The earliest and only consistent component of the flow chamber deposits was the blood platelet, and, in another paper, Murphy and Mustard (1962) claimed that

the "mean platelet adhesive index" was significantly greater and the "plasma thromboplastin time" was significantly shorter in a group of atherosclerotic patients when compared with a control group. Geissinger, Mustard, and Rowsell (1962) found microthrombi on the normal intima of young swine adjacent to the orifices of the intercostal vessels and bifurcations, and believe that platelet thrombi may be important in the production of atherosclerotic lesions.

In 1946 and 1948, Duguid published his studies on coronary and aortic atherosclerosis and suggested that intimal thickening in atherosclerosis was due to the incorporation and organisation of mural thrombi which subsequently had lipid deposited in them. His findings were substantiated by Morgan (1956) and the chain of evidence was carried a stage further when Friedman and Byers (1961) produced experimental thrombi in the arteries of hypercholesterolaemic rabbits and found that thrombus formation invariably led to the formation of an intimal plaque. These plaques accumulated lipid in sites which "simulated exactly" the distribution of lipid in human plaques and differed in appearance from the fatty streaking which occurs with cholesterol feeding alone.

Alterations in the mechanics of blood flow are likely to influence the pattern of deposition of thrombi and Texon, Imparato, and Lord (1960) regarded the velocity of blood flow, the calibre of the lumen, and the arterial pattern as important factors. In an interesting experiment, they implanted an autograft of carotid artery into the femoral artery of a dog. This had the effect of lengthening the artery which became S-shaped and the graft developed atherosclerotic changes in the inside wall of the curve. In this connection, it is interesting that the adductor region of the femoral artery has a natural S-bend (Fig.59) and that the atherosclerotic popliteal artery develops similar types of curve (Fig.66).

In addition to these factors, changes in blood viscosity may be important. Dintenfass (1962) measured the viscosity of blood samples taken at random by a cone-in-cone rotational viscometer and found that one sample showed a 10-fold increase in viscosity compared with the others.

It is therefore possible to explain the development and localisation of atherosclerotic lesions on the basis of alterations in blood flow and thrombus deposition. This, however, infers that intimal changes

are secondary to the deposition of thrombus and there is a considerable body of evidence to suggest that the primary change is intimal.

Robertson (1960) found that intimal cushions develop at the mouths of branches during foetal and early adult life and believes that this intimal hyperplasia develops in adaptation to the stresses of arterial pulsation. With increasing age, the cushions became nodular and the internal elastic lamina fragmented followed by vascularisation and the deposition of lipid. Friedman and Byers (1961) believed that the origin of the plaque depended on preceding intimal hyperplasia and that damage to the intima by thrombosis, haemorrhage, physical, or chemical factors could produce a non-specific intimal hyperplasia in response to such damage.

It is probable that some relationship exists between diet, serum lipids and atherosclerosis and Parker (1960) found that changes occurred in the internal elastic lamina of rabbit's coronary arteries as early as 24 hours after the onset of cholesterol feeding and these changes preceded the appearance of cellular elements. Page (1954) postulated that lipids are normally transported through the arterial wall from within out

and that lipids may be deposited in excess under certain conditions, and Adams, Bayliss, and Ibrahim (1962) demonstrated the early deposit of tritium-labelled cholesterol in the intima by autoradiographic studies. This preceded the concentration of cholesterol in the media and was believed to be due to infiltration from the blood.

If cholesterol is filtered in this way, its deposition must be due to failure of normal metabolism or mechanisms of transport or to the presence of excessive amounts of cholesterol.

In histochemical investigation of normal and atherosclerotic aortas in man, Adams, Bayliss, and Ibrahim (1962) found that enzyme activity progressively falls in the ageing and atherosclerotic vessel, and suggest that these enzyme defects may interfere with the synthesis of phospholipid and protein which are important in the transport and dispersion of cholesterol and other lipids. Dixon (1958) states that lack of phospholipid may lead to inadequate dispersion of lipid and that excess cholesterol appears to interfere with the synthesis of phospholipid and to antagonise phospholipid in its action on emulsions.

The deposition of lipid in atherosclerosis occurs in the deepest layer of the intima which is

normally nourished by diffusion from the lumen of the vessel. The vasa vasorum do not penetrate the intima until plaque formation is developing (Winternitz, Thomas, and le Compte, 1937, 1938) and it has been suggested that anoxia occurs in the portion of intima next to the media. Anoxia is known to interfere with the processes of lipid metabolism (Dixon, 1958) and so may lead to accumulation of cholesterol in this layer.

More recently, it has been suggested that changes in the ground substance of the arterial wall may occur in atherosclerosis but evidence on this point is conflicting. Buddecke (1962) found changes in the quantitative relationships of the various acid mucopolysaccharides in atherosclerotic vessels but Böttcher and Klynstra (1962) found no correlation between the acid mucopolysaccharides and different degrees of atherosclerosis.

The frequent occurrence of intimal haemorrhage in atherosclerotic vessels was first noted by Paterson (1936) and Winternitz, Thomas, and le Compte showed that the atherosclerotic intima was permeated by a network of fine vascular channels derived from the vasa vasorum and from the lumen of the artery and that haemorrhage from these vessels was common.

They suggested that the fatty material in the plaque was derived from the breakdown products of these haemorrhages. Morgan (1956) confirmed the frequent occurrence of intimal haemorrhage in atherosclerotic intima. Paterson, Moffatt, and Mills (1956) demonstrated the presence of iron pigment in early fatty lesions and Friedman, Byers, and St.George (1962) stated that lipid and cholesterol were derived from the vessels vascularising the initial lesion and that their accumulation was increased by haemorrhage from these new vessels.

Intimal haemorrhage may result from the effects of stress on the arterial wall and be a factor in the development and localisation of arterial occlusion as Lindbom (1950) found that the incidence of intimal haemorrhage in the femoral and popliteal arteries was greatest in the segments where occlusion was commonest.

Intimal haemorrhage undoubtedly occurs in the atherosclerotic intima but it is not the primary cause although it may play a part in the further development of the plaque whose origin is determined by other factors.

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The aetiology of atherosclerosis is a complex subject and the search for the ultimate cause continues although it is possible that there is no single cause

and that its onset is determined by a number of factors.

From the viewpoint of a surgeon accustomed to consider peripheral vascular disease in terms of arterial occlusion, it is possible that individual liability to atherosclerosis is determined by racial and genetic susceptibility, by dietary habits of races or individuals, or by endocrine or hormonal factors and that the localisation of lesions in specific areas is determined by alterations in the mechanics of blood flow at bifurcations, curves, and branches or by the development of stress in the arterial wall.

That mechanical factors are important in determining the localisation of lesions is supported by the evidence presented in the next chapter.

CHAPTER SIX.

THE ORIGIN OF FEMORO-POPLITEAL OCCLUSIONS.

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THE ORIGIN OF FEMORO-POPLITEAL OCCLUSIONS.

When one wishes to study arterial occlusion in patients with intermittent claudication it is not possible to utilise amputation specimens as the arterial lesion in these cases is more extensive than in claudication and has led to the development of gangrene. Autopsy specimens are also unsuitable because patients coming to post-mortem are seldom known to have had claudication during life and autopsy studies are therefore concerned with the study of occlusion as an incidental finding.

In claudicating patients, the evidence of occlusion is found in the arteriogram but the study of the lesions is rendered difficult by the complexity and variety of occlusions found. In the following analysis, a method has been adopted which makes it possible to undertake comparative studies of arteriograms and this has yielded information as to the sites of origin of occlusion in the femoral and popliteal arteries.

Material.

Most of the patients who presented with intermittent claudication due to occlusion below the

common femoral artery from 1957 to 1960 underwent bilateral femoral arteriography.

305 men were examined in the out-patient department in whom the femoral pulses were normal and the absence of aorto-iliac occlusion was assumed. All had intermittent claudication and symptoms and signs of incipient or established gangrene were absent.

Of these, 17 were not x-rayed because they were elderly or unfit and an additional 24 have been discarded for the following reasons :-

6 had no visible lesion on arteriography and, in the absence of other causes, the diagnosis of claudication was doubtful; in 6 patients without evidence of occlusion, the claudication appeared to be related to cardiac insufficiency; one patient had no occlusion and treatment of his anaemia 'cured' his claudication; and, in 11 patients, the arteriograms were technically unsatisfactory due to extravasation or inadequate visualisation.

There remained the bilateral arteriograms (528) of 264 men available for study i.e. 86.6% of the original number.

Method of Study.

During these years the technique of arteriography was standard with a constant tube distance of 50 ins. The magnification resulting from this method is illustrated in Fig.49, where lead markers were placed at 5 cm. intervals on the thigh at the approximate level of the femoral and popliteal arteries and lines were drawn on the x-ray film at 5 cm. intervals. The magnification can be calculated by comparison of the recorded distances between the markers and the lines drawn on the x-ray film. As the vessel descends, it inclines backwards and lies closer to the x-ray film so that the magnification at the level of the popliteal artery is less than at the level of the superficial femoral artery.

Calculation shows that the magnification in the lower part of the thigh is approximately 4% increasing to 8% in the upper part of the thigh.

The femoral and popliteal arteries are normally visualised on the 1st and 2nd films of the arteriogram (Fig.15) and the difference in magnification between these two films approximates to 1%. The principal error, therefore, is the 4-8% magnification on to film and the minor difference between the 1st and 2nd films has been ignored. As the conditions of

radiography were standard throughout, it is considered that comparative measurement of films from different patients is possible provided that the data obtained do not demand a high degree of accuracy in measurement.

It is difficult to obtain some fixed point on the arteriogram from which occlusions can be measured. The degree of x-ray penetration of the groin is often poor and it may be impossible to define the common femoral junction and utilise it as a fixed point for measurement. In other films, the exact point of division may be obscured by superimposition of the origins of the superficial and deep femoral arteries and it is uncommon to obtain such a clear picture of the common femoral artery as is illustrated in Fig.50.

Lindbom (1950) utilised the lesser trochanter as a fixed point for measurement and related the length of the occlusion to the distance between the lesser trochanter and the popliteal bifurcation. He then reduced the measurement to a 'standard' length in order to compare occlusions.

The popliteal bifurcation, however, is variable in site and, in the present study, simple measurement of the distance of the occlusion from the knee joint has been used. This method gives a graphic picture of the site and extent of an occlusion when

expressed as e.g. "an occluded segment 6 cms. long whose lower level was 13 cms. above the knee joint level". This method does not apply any correction for variation in the total length of the artery.

To compare occlusions, it is necessary to have a scale against which occlusions can be recorded. In 29 films in which the common femoral junction could be accurately defined, the mean distance from knee joint level was 40.034 cms. with a standard deviation of 1.5 cms. (Range:- 36 to 43 cms.). The majority were grouped around the 40 cms. distance and this has been adopted as the upper level of the scale. Because the upper segment of the superficial femoral artery is usually either wholly patent or wholly occluded, the recording of occlusions by this method is not materially influenced by adopting any level from 36 cms. upwards as the 'standard' level for the common femoral junction.

The adoption of a 'standard' level for the popliteal bifurcation is more difficult as this sometimes lies at or above the knee joint. Major variations, however, were uncommon and, as none of the arteries with a high popliteal bifurcation was occluded (Figs.22 & 23), the recording of occlusions was not affected. In 97 observations there were 6 high bifurcations (all patent) and, in the remainder, the

mean distance from the knee joint to the bifurcation was 5.08 cms.(Standard deviation 0.9 cms.). The majority of observations occurred at 5-6 cms. below the level of the knee joint and 5 cms. has been adopted as the 'standard' distance of the popliteal bifurcation from the knee joint. Where the segment below the knee joint is patent, the recording of occlusions is unaffected whatever the actual length of the segment. Where occlusion involves this portion of the popliteal artery and the actual level of the bifurcation is not visible, it has been assumed to lie at 5 cms. for the purpose of recording the occlusion.

The length of the adopted scale for the femoral and popliteal arteries is therefore 45 cms. Zero represents the level of the knee joint, 40 cms. the common femoral junction and minus 5 cms. the popliteal bifurcation.

Fig.51 illustrates the application of the method.

The distances in centimetres from the knee joint (midway between the femoral and tibial condyles) to the upper and lower limits of an occlusion are measured and a vertical line is drawn against the scale on graph paper to represent the occlusion. In this figure,

the occlusions in the right leg of 42 patients with bilateral claudication have been charted and all types of occlusion are represented. The long occlusions are seen to extend up to the level of the common femoral junction and one extends the whole length of the femoral and popliteal arteries i.e. from 40 to -5 cms. The short occlusions lie mainly at the middle and lower parts of the scale.

When occlusions are drawn in this way, it is possible to construct a histogram of the rate of occlusion at different levels by counting the number of occlusions recorded at each level and plotting horizontally the number obtained against a similar scale. The histogram for the right leg of these 42 patients is seen on the right hand side of Fig.51.

Results.

In Fig.51, the principal feature of the histogram is the large peak at 18 cms. where the rate of occlusion is maximal. There is also a high incidence around this point, the whole peak occupying the portion of the scale between 14 and 25 cms. By reference to the left hand side of the figure, it is evident that this peak is due to the number of occlusions which start or finish within this region and also to the short

occlusions which lie wholly within this region. This portion of the scale is therefore the site of maximum incidence of arterial occlusion and corresponds to the adductor region (Lindbom 1950).

At the upper part of the histogram, the striking feature is the absence of variation between 30 and 40 cms., which indicates that no occlusion in this group has started or finished within this segment. Because thrombosis spreads up and down an artery to the nearest branch whose rate of flow is sufficiently rapid to halt the further spread of thrombosis, the absence of variation in this segment of the histogram suggests that collateral vessels in the upper segment of the superficial femoral artery are either few in number or too small to halt the spread of thrombosis (Fig.50).

The lowest part of the curve from 13 to -5 cms. corresponds to the popliteal artery and appears smooth. The rate of occlusion is less at this level indicating that the popliteal artery is less liable to occlusion than the superficial femoral artery

In the 528 arteriograms, 273 (51.7%) showed occlusion of the femoral and/or popliteal artery, and as 22 arteries had a double occlusion, 295 femoro-popliteal occlusions were found.

Fig.52 is the histogram of 295 femoro-popliteal occlusions. The large adductor peak is prominent and extends from 14 to about 27 cms., being again maximal at 18 cms. The lower part of the curve is not completely smooth and appears to have two steps:- from 5 to 14 cms. and from 2 to -5 cms. Slight variation in the 30 to 40 cm. segment indicates that occlusions are occasionally limited by collaterals in this region.

When analysed in this way, the long occlusions are recorded at multiple levels and over-shadow the pattern of the short occlusions which are recorded at relatively few levels. By excluding the long occlusions and constructing a histogram of short occlusions it is possible to obtain a different and more significant pattern.

Short Occlusions.

As the record of each short occlusion must contain within its length the point of origin of the occlusion, one would expect that the shorter the occlusions charted, the more precise would be the indication of their site of origin. In practice, the number of observations diminishes with increased selectivity and the number of occlusions measuring 2 cms.

or less (25 in number) is too small for analysis. Accordingly, occlusions of 5 cms. and less (88 in number) and 3 cms. or less (46 in number) have been charted.

Fig.53 is the histogram of the distribution of the 88 occlusions measuring 5 cms. or less. Three distinct peaks are visible. The main peak is maximal at 18 cms. and extends from 14 to 21 cms., and corresponds to the adductor peak illustrated in Fig.52. It contains 61 (69.5%) of the 88 occlusions confirming that the majority of short occlusions arise in the adductor region.

The second peak extends from 5 to 10 cms. and contains 11 occlusions (12.5% of 88). The third peak extends from 0 to -5 cms. and also contains 11 occlusions (12.5%).

The remaining 5 occlusions which do not fall within these 3 peaks occupy intermediate positions. Their number is small because short occlusions appear to aggregate at three specific sites in the femoral and popliteal arteries.

When the 46 occlusions of 3 cms. or less are charted (Fig.54), the histogram obtained is similar to that of the 5 cm. occlusions and shows the same three peaks. The adductor peak is maximal at 17 cms., extends from 14 to 21 cms., and contains the majority of the

occlusions - 32 (69.6%). The middle peak at 5 to 10 cms. from the knee joint contains 5 occlusions (10.9%) and the lowest peak from 1 to -5 cms. contains 6 occlusions (13%), there being 3 occlusions occupying intermediate positions.

The pattern of short occlusions illustrated in Figs.53 & 54 suggests that short occlusions arise at three specific sites, and, by inference, that femoro-popliteal occlusions originate at these three sites.

'Incipient' Occlusions.

Another method by which one might expect to obtain evidence concerning the sites of origin of femoro-popliteal occlusions is by charting the distribution of atherosclerotic plaques which narrow the vessel to such an extent that occlusion appears imminent. Fig.55 is the photograph of an 'incipient' adductor occlusion, the degree of narrowing being such as to suggest that thrombosis and occlusion will not be long delayed.

With plaques of this magnitude extending over more than 1 cm. of vessel, the observation has been recorded at a distance from the knee joint equivalent to the mid-point of the plaque.

In the series of 528 arteriograms, there were 167 atheromatous plaques producing severe narrowing and their distribution is charted in Fig.56.

The peak incidence is at 19 cms. with a spread from 14 to 24 cms. which corresponds fairly closely to the adductor peaks illustrated in Figs.53 and 54 and this segment contains over 60% of the total number of observations. There is an increased incidence of lesions between 0 and 8 cms. which does not correspond exactly to the middle peak from 5 to 10 cms. in Figs.53 and 54 but there is an increased incidence of plaques and of short occlusions in the popliteal artery above the level of the knee joint.

There is a low incidence of plaques in the popliteal artery below knee joint level in the area covered by the lowest peaks of Figs.53 and 54 but there is a high incidence of lesions at the popliteal bifurcation. As the lowest peak in the histogram of the 3 cms. occlusions (Fig.54) tends to rise towards the popliteal bifurcation (-5 cms.), it is possible that the popliteal bifurcation is the site of origin of these low occlusions.

Fig.57 illustrates narrowing at the popliteal and posterior tibial bifurcations, and the lesion at the popliteal bifurcation appears likely to produce anterior

tibial occlusion without affecting the main artery. On the other hand, the lesion in Fig.47 is producing narrowing of the lowest portion of the popliteal artery and, if occlusion were to develop, it would seem liable to spread up the popliteal artery to the nearest large collateral vessel. This type of lesion might therefore lead to a low popliteal occlusion (Fig.58) and produce the type of occlusion responsible for the lowest peaks of Figs. 53 & 54. Comparison of Figs.47 & 57 suggests that only some plaques at the popliteal bifurcation produce low popliteal occlusions, whereas others produce anterior tibial or posterior tibial occlusions.

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From the evidence furnished by the histograms it would appear that femoro-popliteal occlusions originate at three specific sites :-

1) in the adductor region which lies from 14-24cms. from the knee joint and is responsible for 60-70% of femoro-popliteal occlusions;
2) in the portion of the popliteal artery which lies above the knee joint;
3) at the popliteal bifurcation.

THE CAUSE OF OCCLUSION IN THE FEMORAL AND POPLITEAL ARTERIES.

a) The Adductor Region.

The histograms in Figs.52, 53 and 54 indicate that the segment from 14-24 cms. is the region where the incidence of occlusion is maximal and where plaque formation is commonest (Fig.56). This appears to represent the adductor region (Lindbom, 1950), although it is difficult to define this region on the individual arteriogram. The adductor canal and the opening in the adductor magnus tendon through which the femoral artery passes to become the popliteal artery can vary in site and the customary A-P view of the arteriogram gives little indication of the level of the adductor opening.

The lateral view (Fig.59) gives a better indication of how the superficial femoral artery inclines posteriorly to become the popliteal artery, and this arteriogram suggests that the adductor opening might lie about 15 cms. from the knee joint.

The descending genicular artery (Fig.60) may occasionally be easily identified on the arteriogram and as it usually leaves the main vessel just proximal to the adductor opening (Gray, 1962), it can be used as a guide to the level of the opening. In 20 arteriograms in which

it was readily identified, this artery and/or its subcutaneous saphenous branch was usually found to arise between 13 and 15 cms. although the distance of its origin from the knee joint varied from 12 to 16 cms. On the other hand, it has occasionally been seen at operation to take origin from the upper end of the popliteal artery just beyond the adductor opening and its position is therefore only an approximate guide to the adductor opening.

It is generally accepted that the adductor region is the commonest site of occlusion in the arteries to the lower limb (Leriche and Bertrand, 1946) but estimates of the frequency of occlusion at this level tend to vary. Mavor's (1958) estimate that this region was the primary site of thrombosis in 83% of 223 cases (including aorto-iliac occlusions) appears very high and Dunlop and Santos' (1957) incidence of 76% was based on a small series of only 41 arteriograms. Singer (1963) has recently reviewed a series of arteriograms at St. Mary's Hospital and found that 64% of femoral arteries were thrombosed in the adductor region, cases of aorto-iliac occlusion being excluded.

In the present series, the maximum incidence of occlusion in the femoral and popliteal arteries (Fig.52) was found at 18 cms. where 60.5% of the 273

occluded arteries were thrombosed. This number, however, only relates to one point in the arterial tree and does not give the number of occlusions located or arising in the adductor region. It is possible to obtain some evidence concerning the incidence of occlusion in the adductor region from Figs.53 and 54 where the number of adductor occlusions was respectively 69.5% and 69.6%. Further indirect evidence may be obtained from Fig.56 where 62.3% of the atherosclerotic plaques were found in the adductor region.

These figures are more closely allied to the 64% incidence noted by Singer than the figures quoted by Mavor and Dunlop and Santos, and it seems reasonable to assume that the adductor region will be the site of origin of between 60 and 70% of all femoro-popliteal occlusions.

The commonly accepted theory of the cause of occlusion at this level is the repeated trauma experienced by the pulsatile artery where it lies in intimate contact with the tendon of adductor magnus (Fig.61)- Palma, 1959. This could produce intimal haemorrhage which Lindbom (1950) has shown to be common in the adductor region and which is incriminated by Winternitz, Thomas, and Le Compte (1938) and by

Morgan (1956) as the most important factor in the increase in size of atherosclerotic plaques.

Lindbom (1950) produced arteriographic evidence to show that the artery is relatively fixed in the adductor canal. He made two arteriographic exposures of the artery on one film with the quadriceps muscle relaxed during one exposure and contracted in the other, and found that the artery presented one image in the adductor region and a double image above and below. He suggested that stress is engendered between this segment and the more mobile artery above and below leading to intimal haemorrhage and subsequent deposition of cholesterol. Personal attempts to reproduce this finding have, however, been unsuccessful.

This is a variation of the stress theory and, although there is no doubt that vessel damage and thrombosis may be produced by the major trauma of a penetrating or closed injury (Fig.62), it is more difficult to evaluate the effects of recurrent minor trauma in the production of atherosclerotic lesions.

There is some evidence to show that another factor - the tendency for plaque formation to occur at the origin of large tributories - may be involved in the development of atherosclerotic plaques and occlusion in

this region. Robertson (1960) has shown that intimal cushions develop at the origin of branches in the femoral and popliteal arteries in early adult life and that, with increasing age, they become nodular and vascularised and the eventual deposition of lipid completes their transformation into atherosclerotic plaques. Support for this theory is afforded by the evidence that branches are numerous in the adductor region and in the popliteal artery above the level of the knee joint where occlusion is commonest. The frequency with which large branches arise from these segments can be shown as follows : -

Each occlusion is limited above and below by a collateral vessel whose rate of flow has been sufficient to halt the further spread of thrombosis. Small arterial branches are often obliterated by the spreading thrombus and the collaterals bounding an occlusion tend to be derived from the larger branches of the superficial femoral and popliteal arteries.

In Fig.63, the upper and lower levels of occlusion have been plotted separately for each of the 295 femoro-popliteal occlusions found. The distribution curve on the left of this figure is the distribution of the 'limiting' collaterals above the occlusion, and the curve on the right is the distribution of the main 're-entrant' collateral below each occlusion.

By summation of the curves in Fig.63, Fig.64 is obtained. This is a combined histogram of the 'limiting' and 're-entrant' collaterals bounding the occlusions and should be related to the distribution of the larger and more important branches of the femoral and popliteal arteries although it is not the pattern of distribution of all the collaterals in each case.

The presence of a large number of major collaterals in the adductor region is evident and these numerous vessels are responsible for the frequency with which short occlusions occur in the adductor region, i.e. it is the numerous branches which determine the occurrence of short occlusions and not vice versa. The opposite is seen in the upper part of the superficial femoral artery where collaterals are few (Fig.50) and occlusions are long.

It is possible, therefore, that the high incidence of collateral vessels in the adductor region favours the development of plaques either by degeneration of the intimal collar at the mouth of each vessel or by contributing to the fixity of the artery noted by Lindbom (1950). There is also a negative correlation between the distribution of branches and plaque formation in that intimal thickening (Lindbom, 1950) and plaque development (Fig.56) are uncommon in the

regions where branches are few i.e. in the upper part of the superficial femoral artery and in the popliteal artery below the level of the knee joint.

It is not claimed that plaque formation at the origin of branches in the adductor region is the cause of the high frequency of thrombosis in this region but it is believed to be an important factor, additional to the part played by the adductor tendon itself.

b) The Popliteal Artery.

Mavor (1958) stated that the development of thrombosis at a site other than the adductor region was uncommon, and implicated the popliteal artery as the site of thrombosis in only 10% of cases. Similarly, Leriche and Bertrand (1946) described only 15 popliteal occlusions in 133 occlusions of the lower leg. On the other hand, Haimovici, Shapiro, and Jacobson (1960) found the incidence of popliteal occlusion was much higher - 32.7%. These differences in the incidence of popliteal occlusion may be due to variations in the type of case included in these series as popliteal occlusion is commoner in gangrene than it is in claudication.

Figs.53 & 54 indicate that there is a segment of the popliteal artery above the level of the knee joint

which is the origin of about 12% of all femoro-popliteal occlusions in claudicating patients, and Fig.56 suggests that this site of origin is not a specific one but is rather diffuse.

Lindbom's (1950) observation that the distal popliteal artery is comparatively free from intimal thickening and the known low incidence of occlusion in the popliteal artery below the level of the knee joint (Martin, 1958) are confirmed by the relative absence of plaque formation (Fig.56) and the low incidence of occlusion at this level (Fig.52). Mavor (1958) noted that the segment of the popliteal artery below the level of the knee joint is "free of branches and usually healthy" and did not make the deduction that the absence of branches in this segment (Fig.64) could be the reason for its relative freedom from atherosclerosis.

The cause of occlusion in the popliteal artery above the level of the knee joint has never been adequately explained. Haimovici, Shapiro, and Jacobson (1960) suggest that the popliteal artery above the knee is subjected to trauma during bending and stretching of the knee joint but do not explain why the lesions should arise mainly above the knee joint.

The popliteal artery in this region is surrounded by adipose tissue which allows it to move

freely during flexion of the knee joint and it is not confined by a fixed anatomical structure comparable with the adductor tendon.

Fig.65 illustrates the early popliteal lesion. This patient has developed an area of popliteal narrowing in a typical site with previous evidence of severe atherosclerosis.

A lateral arteriogram of the popliteal artery (Fig.66) during knee flexion shows that the atherosclerotic artery develops a large primary and smaller secondary curves due to the arterial elongation which develops in atherosclerosis. In this figure, branches leave the main artery at the ends of the principal curve and it is possible that the artery is tethered to contracting muscle at these points and is free to develop a curve only between these branches. If this is so, and the curve pattern of the tortuous atherosclerotic popliteal artery is determined by the site of origin of its large vessels, the curve pattern will vary from patient to patient as the origin of the large branches varies.

Lindbom (1950) showed that calcification in the popliteal artery develops on the summit of the convexity of the curves and suggested that this part of the artery was subject to the greatest stress in knee

movement. It might be expected that atherosclerotic plaques would also form at the summit of the convexity in response to stress, and Fig.67 suggests that this may be so. On the other hand, Texon, Imparato, and Lord (1960) implanted an S-shaped autograft in the femoral artery and found that atherosclerotic changes were most severe in the concave wall of the curve.

It is therefore possible to postulate that the variable curve pattern of the popliteal artery is the cause of variation in the site at which atherosclerotic plaques develop, and this would explain the scattered observations recorded in Fig.56 where plaques were found to develop at a variable level in the popliteal artery above the level of the knee joint. The variable level of origin of the sural vessels from this segment (Fig.64) could determine the curve pattern of this segment of the artery and so be responsible for influencing the development of occlusion at this level (Figs.53, 54).

There is a possible alternative explanation for the development of lesions in the popliteal artery above the level of the knee joint.

Intimal thickening is common at the origin of branches and Lindbom (1950) was able to show that

the distribution of branches and of intimal thickening was similar in the femoral and popliteal arteries. Increase in this intimal thickening could lead to plaque formation and ultimate occlusion and would explain the relationship between the large branches in the popliteal artery above the knee joint (Fig.64) and the development of atherosclerotic plaques (Fig.56) and occlusion (Figs.53 and 54) in this segment. On this basis, the relative absence of branches below the knee joint (Fig.64; Mavor, 1958) would explain the low incidence of plaque formation (Fig.56) and the low incidence of occlusion in this segment (Fig.52; Martin, 1958).

There is no definite evidence to show whether lesions develop in the popliteal artery due to plaque formation at the summit of the curves or to plaque formation at the origin of branches, but it is possible that both mechanisms could be responsible for the development of occlusion.

c) The Popliteal Bifurcation.

Fig.56 indicates that plaque formation is common at the popliteal bifurcation, and it appears probable that low popliteal occlusions (Fig.68) develop from lesions at this level (Fig.47).

Mavor (1958) stated that spread of thrombosis in the popliteal artery was in a distal direction. A total popliteal occlusion as illustrated in Fig.69 could arise in two ways. As Mavor suggests, the lesion could develop in the upper part of the popliteal artery and spread distally obliterating the sural arteries until the bifurcation was reached. Alternatively, the lesion could commence at the popliteal bifurcation and spread upwards, again obliterating the sural arteries. Fig.60 shows a sural artery which is grossly narrowed at its origin and suggests that the second mechanism of upward spread is occurring in this case.

In general, it is believed that upward spread of occlusion from the popliteal bifurcation is important and Figs.53 and 54 suggest that it may account for about 12% of all femoro-popliteal occlusions.

CHAPTER SEVEN.

THE COLLATERAL CIRCULATION.

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When occlusion develops in a main artery, the nourishment of the distal part of the limb depends on the available blood flow through alternative channels - the collateral circulation.

Where there are two major arteries acting in parallel as in the forearm (the radial and ulnar arteries) and these are united distally by major arterial channels (the palmar arches), occlusion of either main artery produces relatively little circulatory disturbance as the remaining channel is adequate to provide for the metabolic requirements of the hand. Although this system is theoretically duplicated in the lower leg, the distal connections through the malleolar and plantar arches are small and occlusion of one lower leg artery is likely to produce symptoms of arterial insufficiency, especially where the remaining vessels are affected by atherosclerosis.

In the lower limb, occlusion is commonest in the femoral and popliteal arteries and relatively common in the aorta and iliac arteries. This blocks the main arterial flow and the blood supply to the distal parts depends on the anastomoses available between the branches

proximal and distal to the occlusion, the symptoms produced by occlusion depending on the rate of onset of occlusion and on the arterial segment involved.

If thrombosis develops suddenly, as in embolism, secondary or consecutive thrombosis is liable to overwhelm and obliterate the adjacent normal branches on whose integrity the collateral circulation depends and ischaemia will be severe.

In atherosclerosis, on the other hand, the onset of occlusion is insidious and is preceded by a period during which the main artery is narrowed and the volume of blood flow is reduced. During this time, the distal arterial pressure is reduced and there is an increased pressure differential in the collateral vessels leading to an increased rate of flow through them and a consequent increase in their diameter (Nothnagel, 1889) accompanied by increased arterial tension within them and consequent hypertrophy of the arterial wall (Thoma, 1893). Fig.55 illustrates the development of the collateral circulation at this stage in response to narrowing of the main artery.

Thus, when occlusion finally develops, the collateral circulation is already available and sufficient blood is conveyed to the distal parts of the limb to maintain viability except when occlusion is

extensive. Following occlusion, the collateral vessels undergo a further increase in size and efficiency leading very often to an improvement in symptoms in the first few months after occlusion has developed.

From the site of the initial occlusion, thrombosis spreads up and down until halted by the increased rate of flow through a large branch and the occlusion becomes bounded by the nearest efficient arterial branches. In many cases, the collateral vessel below the occlusion is larger than that which arises proximal to the occlusion (Fig.70). Above the occlusion, blood leaves the main artery by various branches and by the 'limiting' collateral situated at the upper end of the occluded segment, and re-enters the patent main artery distal to the occlusion by the main 're-entrant' collateral (Longland, 1953.) and other branches below the occlusion. The connecting channels between the limiting and re-entrant collaterals vary in size and length and are characterised on the arteriogram by their tortuous appearance (Figs.71, 72).

When the collateral circulation is efficient, the spread of thrombosis is limited by large collaterals and the occlusion becomes 'stabilised'. Under these conditions, the patient's claudication may remain static and the prognosis is good (Bloor, 1961).

The disease process remains, however, and may involve the origins of the collateral vessels themselves producing narrowing of the vessel as it meets the main artery (Figs.48, 60). In the course of time, thrombosis may obliterate these collaterals and lead to extension of the occlusion which will remain segmental only if there are other adequate collateral vessels to limit the spread of thrombosis.

If the collateral circulation is inefficient when occlusion occurs, the primary thrombosis may be extensive and produce severe symptoms or the occlusion may stabilise temporarily and extend at a later date because the distal rate of flow is insufficient to prevent further thrombosis.

EFFICIENCY OF THE COLLATERAL CIRCULATION.

The localisation of thrombosis to a segment of main artery and the degree of disability produced by occlusion depend on the efficiency of the collateral circulation, and this is determined by such factors as the severity of the atherosclerotic changes, the age and cardiac status of the patient, and the site and extent of arterial occlusion.

a) Severity of Atherosclerosis.

When atherosclerotic changes are widespread and severe, the collateral vessels are liable to become narrowed at their origin (Fig.60) or the main vessels feeding the collaterals may also be diseased (Fig.73). Under such circumstances, the flow through the collateral circulation is reduced and symptoms may be severe.

On the other hand, where only mild atherosclerotic changes are present, hypertrophy of collateral vessels readily occurs and this is sometimes evident in the profunda femoris artery (Fig.23) - (Mavor, 1952.)

b) Age.

To a large extent this is correlated with the previous factor as the severity of atherosclerotic changes tends to increase with age, although there are many exceptions to the rule. In younger patients, the collateral circulation is usually more efficient and an extensive or major occlusion may produce only a minor disability. Fig.74 illustrates an aortic occlusion in a young diabetic patient of 37 years, yet in spite of this serious lesion he does not have any pain when walking on level ground and only experiences claudication on hills. Fig.71 illustrates a double femoro-popliteal

occlusion which virtually amounts to total femoro-popliteal occlusion. This patient, aged 44 years, suffered from claudication only, although this type of occlusion frequently gives rise to gangrene.

The elderly patient, however, cannot develop such a good collateral circulation and is liable to experience severe claudication or to develop gangrene when arterial occlusion is extensive.

c) Cardiac Status.

The cardiac state of a patient may affect the severity of symptoms experienced. Patients with severe cardiac insufficiency and impaired cardiac output due, for example, to auricular fibrillation or congestive cardiac failure may experience claudication even with a patent arterial tree and improvement in claudication distance is not possible until their cardiac efficiency increases. Where occlusion is present, the collateral blood flow in patients with cardiac insufficiency will be poor. The occlusion sets up a peripheral resistance which the failing heart cannot overcome and in extreme cases of right heart failure gangrene of the toes may develop.

d) Site of Occlusion.

The anatomical pathways of the collateral circulation developed in response to arterial occlusion are described separately with the individual lesions.

In general, the collateral circulation developed in aorto-iliac occlusion is more efficient than that in femoro-popliteal occlusion (De Bakey, 1958). To some extent, there is a gradation in efficiency of the collateral circulation according to the site of occlusion, the efficiency being highest in aorto-iliac occlusion and poorest in popliteal and tibial occlusions.

In 1932, Gibbon and Landis studied the vasodilatation occurring in the toes in response to immersion of the hands in warm water and showed that this can be used as a test of vasomotor activity. When skin temperature thermocouples are attached to the great toes and connected to a sensitive standardised galvanometer, a direct measure of the vasodilatation response to reflex heating is obtained. Lewis and Pickering (1931) showed that these vasomotor changes are mediated through the efferent pathways of the sympathetic nervous system and this test may be used as an index of the rise in temperature which can be produced in the foot by sympathectomy.

When this reflex vasodilation test is used

in patients with arterial occlusion, different types of curves may be obtained (Fig.75). When a patent arterial channel to the toe is present (i.e. one tibial artery can be occluded without altering the shape of the curve), the curve rises rapidly and is S-shaped. When continuity of the main arterial tree is interrupted by atherosclerotic occlusion, the commencement of the rise is delayed, the rate of rise is slower, and the maximum height of the curve is usually reduced.

Fig.75 illustrates three types of abnormal curve. One is the 'femoral lag curve' described by Reid (pers.comm.) where the curve lags behind the normal one but ultimately reaches maximum levels. This type of curve is uncommon but it is almost diagnostic of occlusions of the proximal part of the superficial femoral artery or of a short adductor occlusion with good collateral vessels. When the site of occlusion is more distal or the femoro-popliteal occlusion is of moderate length, the second type of abnormal curve occurs with a slow delayed rise to intermediate levels. The third curve is virtually a straight line with little evidence of a rise in temperature and is found in popliteal or extensive femoro-popliteal occlusions. The collateral circulation is poor in such cases and the prognosis as to viability of the leg may also be poor.

To some extent, therefore, the efficiency of the collateral circulation as measured by the reflex vaso-dilation test is related to the site of occlusion and the rate of rise of the curve is slower in the more distal occlusions.

e) Length of Occlusion.

The length of an occlusion also affects the efficiency of the available collateral circulation. Where the occlusion is short, the collateral channels are also short (Fig.76) and usually efficient. In a few cases, they may even form a 'bridge' across the gap, and an unusual example of this is seen in Fig.77 where the anastomosing collateral vessels have hypertrophied to form a channel which is almost as wide as the main artery.

In other occlusions, the collaterals run a longer course before anastomosing with distal vessels (Fig.78) and their efficiency is correspondingly reduced although they may compensate for this by their multiplicity.

In lengthy occlusions, the collaterals take a long and often devious course and in the extreme case of total femoro-popliteal occlusion the channels derived from the profunda femoris have to travel to below the

knee to effect union with branches of the tibial arteries (Fig.79). In such circumstances, the collateral circulation is poor and there is a risk of gangrene.

THE EFFECT OF SYMPATHECTOMY ON THE COLLATERAL CIRCULATION.

The arteries of the lower limb are controlled by tonic vaso-constrictor impulses from the lumbar sympathetic chain. The collateral circulation in patients with arterial occlusion is subject to the same control and release of sympathetic activity will result in dilatation of collateral vessels and a consequent increase in blood flow. As sympathetic control is released by reflex heating, the reflex vaso-dilation test gives a measure of the potential increase in collateral circulation it is possible to obtain after lumbar sympathectomy. The reflex vaso-dilation curve found in patients who have undergone sympathectomy (Fig.80) suggests that the collateral channels are more or less maximally dilated at all times after sympathectomy.

Experiments on rabbits (Longland, 1953) suggest that sympathectomy promotes early development of the full potential of the collateral circulation but that the number and size of collaterals developed in response to arterial ligation is not increased by sympathectomy. However, arteriography in patients who have undergone

sympathectomy shows extensive development of anastomotic channels which has not been seen in non-sympathectomised patients.

J.C., a miner aged 54 years, was seen in 1958 when he complained of bilateral claudication at 100 yards distance of four years duration. A left lumbar sympathectomy had been performed in 1955 without benefit.

Femoral arteriography revealed a long femoro-popliteal occlusion in the right leg with reconstitution of the popliteal artery and a total femoro-popliteal occlusion in the left leg (Fig.81) with anterior tibial and peroneal occlusions.

The left foot was very warm and, in spite of the extensive arterial occlusion, his sympathectomy appears to have promoted the development of an extensive collateral circulation which ensured the viability of the leg. Collateral development of this degree has only been seen in patients who had undergone lumbar sympathectomy or phenol sympathetic block some years before.

The reflex vaso-dilation test of this patient is illustrated in Fig.80. The curve of the right leg shows a moderate but incomplete rise, and that of the left leg starts at a high level and shows very little

rise, indicating that sympathectomy has produced virtually maximum vaso-dilatation.

Following phenol injection of the right lumbar sympathetic chain, his right foot also became very warm but his claudication distance was not improved. Bilateral selective nerve crush was performed and he subsequently was able to walk for more than a mile without stopping and he returned to work at the coal face.

The extensive development of collateral vessels following sympathectomy found in this and other cases does not accord with Longland's findings in rabbits where the number and size of collaterals was not increased by sympathectomy, and it may be that the effects of sympathectomy in man are different to those in rabbits.

CHAPTER EIGHT.

THE PATTERN OF ARTERIAL OCCLUSION.

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Arterial occlusion causing intermittent claudication may be single or multiple, unilateral or bilateral, and may affect any portion of the arterial tree from the abdominal aorta to the plantar arteries. Certain parts of the arterial tree are more prone to occlusive disease than others and it is possible to recognise certain common types of occlusion on arteriography and to attempt to classify them.

It is customary to consider two main groups of occlusions : - aorto-iliac occlusions affecting the aorta and iliac arteries and femoro-popliteal occlusions involving the femoral and popliteal arteries (De Bakey, 1958). These can be differentiated clinically by the character of the femoral pulse and this grouping is important in relation to symptomatology and the type of surgical treatment undertaken in each.

Tibial and peroneal occlusions are frequently regarded as mere concomitants of femoro-popliteal occlusions and an infrequent cause of claudication e.g. 8% of 223 cases (Mavor, 1958), but this study suggests that they are more common than is generally realised, and they have been analysed as a separate group.

A. THE PATTERN OF AORTO-ILIAC OCCLUSION.

Since Leriche's (1940) classical description of thrombosis of the aortic bifurcation, it has been recognised that other types of aorto-iliac disease occur and that the pattern of aorto-iliac occlusion is varied and complex.

The pattern of aorto-iliac occlusion has been studied in a consecutive series of 90 aortograms in which lesions responsible for producing intermittent claudication were found, patients with ischaemic rest pain or gangrene having been excluded. This group is necessarily a biassed sample of the patients presenting with aorto-iliac occlusion and claudication, because approximately one-third of the patients seen at the Out-Patient Department were not subjected to aortography either because they were too old or unfit to undergo this investigation or were considered unsuitable for reconstructive surgery. Most of the aortograms, therefore, were performed in patients under 60 years of age. In addition, minor aorto-iliac lesions are likely to have been excluded from consideration because the femoral pulse was not sufficiently altered in character to indicate the necessity for aortography and, in these cases, bilateral femoral arteriography, the easier and

safer examination, was undertaken.

In the classification of lesions from aortograms, it has been necessary to exclude minor atherosclerotic irregularities and areas of narrowing, and incipient lesions have only been included where the degree of arterial narrowing was severe and the lesion has been the cause of symptoms.

It is difficult to deduce the site of origin of some occlusions as consecutive thrombosis tends to obscure the pattern, and this difficulty is greatest in considering common iliac occlusions which may originate in the artery itself or from lesions at the aortic or common iliac bifurcations. However, an attempt has been made to deduce the probable site of origin of occlusion from the type of lesion present. For example, in Fig.82 it has been assumed that the left common iliac occlusion originated in the common iliac artery itself as there is an early equivalent lesion on the right side, and, in Fig.83, a similar origin could be assumed as there is no evidence of narrowing of the patent common iliac artery at the aortic bifurcation. However, the presence of external iliac occlusion suggests that occlusion in this case may have originated at the right common iliac bifurcation. Fig.84 shows a right common iliac occlusion which has presumably originated at the aortic bifurcation.

Combined common and external iliac occlusions have been assumed to originate from lesions of the common iliac bifurcation (Fig.83) although it is possible that the lesion originated either at the aortic bifurcation or in the common iliac artery and the external iliac artery became occluded by distal spread of thrombosis.

Certain deductions and assumptions have therefore been made as to the origin of individual occlusions but careful study suggests that there are three common sites of origin of aorto-iliac occlusions - the aortic bifurcation, the common iliac artery itself and the common iliac bifurcation.

1. Occlusions of the Aorta.

In 31 of the 90 cases, the arterial lesion appeared to originate from the aortic bifurcation (Table 1).

The commonest lesion was atherosclerotic narrowing at the bifurcation (18 cases) which was mild in two cases (Figs. 9 and 31) and severe in 16. This usually resulted in severe stenosis of the orifices of both common iliac arteries (Fig.45) but in three cases narrowing of the common iliac artery was unilateral. In one case, however, the incipient lesion was in the

lower abdominal aorta itself (Fig.85).

Table 1 :- Classification of Aorto-Iliac Occlusions in 90 Aortograms according to probable site of origin.

In this table, only the principal lesion causing symptoms is charted and additional lesions are not recorded.

Site of Origin of Lesion.		Type of Lesion.	
Aortic Bifurcation	- 31	Aortic Occlusion to renal art.	- 4
		to inf.mes.art.	- 5
		Incipient Bifurcation Lesion Unilateral	- 3
		Bilateral	-15
		Common Iliac Occlusion Unilateral	- 1
		Bilateral (one stenosed)	- 3
Common Iliac Artery	- 28	Incipient Occlusion Unilateral	-16
		Bilateral	- 3
		Common Iliac Occlusion Unilateral	- 4
		Bilateral (one stenosed)	- 5
Common Iliac Bifurcation	- 24	Incipient Common Iliac Bifurcation	- 2
		External Iliac Occlusion	-12
		Internal Iliac Occlusion	- 2
		Combined Iliac Occlusions Common and External	- 6
		External and Internal	-2
Common Femoral Artery	- 7	Common Femoral Occlusion	- 7

In four cases, the common iliac artery was occluded on one side with evidence of narrowing of the other artery in three of these due to a bifurcation lesion (Fig.86), and there were nine cases of aortic occlusion.

The development of stenosis at the aortic bifurcation is therefore relatively frequent and important as it appeared to be the site of origin of 22 bifurcation lesions and 9 aortic occlusions.

In a few patients, stenosis due to fibrosis appears to predominate (Fig.9), but, in the majority, intimal thickening and thrombus deposition lead to progressive narrowing and eventual thrombosis. During operative dissection in three of these cases, a portion of thrombus, loosely attached to an intimal plaque, became dislodged and plugged a common iliac artery, and this suggests another way in which occlusion could be precipitated.

With any bifurcation lesion, blockage of the orifices of both common iliac arteries may develop and secondary thrombosis will extend up the aorta and down the common iliac arteries. The outflow through the lumbar arteries is usually insufficient to halt the upward spread of thrombus and the level of proximal

extension will depend on the rate of outflow through the inferior mesenteric artery. If this is rapid, thrombosis will be halted at this level giving the type of lesion illustrated in Figs.74 and 87. This was found in 5 cases.

If the inferior mesenteric artery is narrowed at its origin or already occluded (Fig.31), thrombosis will extend up to the level of the renal arteries (Fig.88). In other cases, thrombosis may be halted at the level of the inferior mesenteric artery but may subsequently extend upwards due to progressive stenosis at the origin of this artery. Four patients presenting with intermittent claudication were found to have a complete aortic occlusion up to the level of the renal arteries and three cases (one with a bifurcation lesion and two with occlusion up to the inferior mesenteric artery) subsequently developed complete occlusion up to the level of the renal arteries. None of these patients developed gangrene although one of the latter group developed temporary ischaemic rest pain (Fig.84).

Thrombosis is usually halted at the renal arteries because of the rapid outflow of blood at this level. Occlusion involving both renal arteries is incompatible with life and is a rare event which has only been seen in one patient in whom a saddle embolus

produced aortic occlusion and subsequent embolism led to bilateral renal artery occlusion and death.

Although the inferior mesenteric artery is frequently narrowed or occluded by thrombosis, it is unusual for symptoms of colonic ischaemia to develop. When the artery is narrowed at its origin by atherosclerosis, the marginal artery of the colon hypertrophies (Fig.88) and is supplied from the superior mesenteric and rectal arteries, and when occlusion occurs, the marginal artery maintains the viability of the colon. Damage to the artery during dissection in a patient with aorto-iliac occlusion or the ligation necessitated by excision of an aneurysm do not usually affect bowel viability because of the collateral circulation which has been built up, but acute occlusion of a healthy artery as in inferior mesenteric embolism may lead to gangrene of the colon.

2. Occlusions of the Common Iliac Artery.

Occlusion of the common iliac artery may be due to lesions arising at one of three sites :-

a) the aortic bifurcation (Fig.84).

b) in the main stem of the artery itself (Fig.82).

c) the common iliac bifurcation (Figs.46 and 83).

It is not always possible to determine which of the three

sites is the origin of an individual occlusion but a reasonably accurate deduction can be made in some cases. In lesions where the occlusion is incomplete, the source of origin can be accurately localised (Fig.40).

Table 2 :- Classification of 128 major incipient and actual occlusions in the aortogram of 90 patients with intermittent claudication.

Site of Occlusion.	Number of Occlusions.
Aortic Occlusion	9
Incipient Bifurcation Occlusion	18
Stenosis of Common Iliac Artery	31
Occlusion of Common Iliac Artery	21
Occlusion of External Iliac Artery	23
Occlusion of Internal Iliac Artery	12
Stenosis of Common Iliac Bifurcation	5
Occlusion of Common Femoral Artery	9
Total	128

Lesions of the common iliac artery are the commonest form of aorto-iliac occlusion (Table 2). The 21 occlusions and 31 cases of stenosis of the common iliac artery account for 57 (43.8%) of the 128 lesions found in 90 aortograms.

The origin of lesions in the course of the common iliac artery (Figs.89 and 33) is unusual in that the lesion arises in the mid-portion of an artery which

does not give origin to large branches. In the lower limb, atherosclerotic lesions tend to arise at the bifurcations of major arteries e.g. the aorta; in relation to a fixed unyielding tendon - the adductor tendon; or in an artery subject to curve formation and giving off large branches - the popliteal artery; but the common iliac artery does not have any of these relationships. It is therefore surprising that lesions in the artery itself are so common and, although it may be adherent to the left common iliac vein as it crosses it, the degree of fixity induced does not seem to be a sufficient source of stress as to favour the development of plaques.

In a study of blood flow in a model constructed to resemble the aortic bifurcation, Murphy and his colleagues (1962) found that platelet thrombi were deposited at the bifurcation and that the two tributories were often free from deposits in their proximal one inch with further linear deposits beyond this. This would approximate to the middle of the common iliac arteries and could explain these lesions on the basis of alterations in blood flow and thrombus deposition.

An alternative explanation is possible. The atherosclerotic aorta tends to become elongated and, in cases where the aortic bifurcation is relatively free of disease and not fixed, it has been noted at operation

that there is an up and down movement of the aorta with each pulsation. If we assume that the aorta is fixed above by the renal and superior mesenteric arteries, and the common iliac bifurcation is fixed by the origin of the internal iliac artery, the elongated Y-shaped mobile segment would tend to buckle at the middle of the common iliac arteries leading to stress in the arterial wall at the middle of the vessel i.e. the site of origin of many lesions (Fig.40). This stress could lead to intimal hyperplasia and, eventually, to plaque formation.

It is possible that the relative fixity at the common iliac bifurcation protects the external iliac artery from undue movement and this could explain the relatively low incidence of severe atherosclerosis in the external iliac artery.

In 28 of the 90 aortograms, the principal lesion (Table 1,p.128) appeared to arise in the middle of the artery and it is therefore almost as important a source of occlusion as the aortic bifurcation. In 8 of these 28 lesions, a bilateral distribution was found.

Five of these (e.g. Fig.82) showed occlusion of the one side and an incipient occlusion of the other. If the second side were to progress to occlusion, consecutive thrombosis would lead to occlusion of the aorta itself and it is therefore possible that one or

two of the aortic occlusions (Table 1, p.128) arose from primary common iliac lesions rather than aortic bifurcation lesions as has been assumed. If this were so, it would make the common iliac artery as important a source of occlusion as the aortic bifurcation.

Bilateral disease, however, is more common than the figures for bilateral distribution of lesions suggests. At operation, it is common to find that, although the lesion is apparently unilateral, the severity of atherosclerotic change is only a little less severe on the other side and Fig.90 illustrates this point. The small niche in the left common iliac artery is insufficient to classify this as a bilateral lesion in Table 1 (p.128), but evidence of bilateral disease is present on the aortogram although symptoms were undoubtedly unilateral.

Symmetrical changes are therefore very common in common iliac disease although the rate of progression of the two sides is usually asymmetrical.

3. Occlusions of the External Iliac Artery.

Occlusion of the external iliac artery is not uncommon and accounted for 23 (18%) of the 128 major lesions found in the 90 aortograms (Table 2, p.132). As the principal lesion causing symptoms, it was found in

12 of the 90 aortograms and in a further 8 it occurred in combination with occlusion of other iliac arteries of the same side (Table 1, p.128).

The frequency of these occlusions is rather surprising because at operation in cases with occlusion of the aorta or common iliac arteries, the external artery is usually free from severe disease. Incipient lesions of this artery have not been seen except as narrowing of its origin at the common iliac bifurcation and it is probable that occlusion is secondary to lesions of this bifurcation. The whole length of the external iliac artery becomes obliterated by consecutive thrombosis as there are no major branches to prevent total occlusion.

Fig.91 illustrates an occlusion of the right external iliac artery and there is a large notch at the left common iliac bifurcation due to plaque formation. On grounds of possible symmetry one might infer that the right sided occlusion originated from the common iliac bifurcation. On the other hand, Fig.92 shows a combined external iliac and common femoral occlusion and it is doubtful as to where the combined occlusion originated. Occlusions of the common femoral artery are usually associated with a patent external iliac, the limiting collaterals being the inferior epigastric and deep circumflex iliac arteries (Fig.93), and it is therefore

believed from aortographic and operative evidence that most external iliac occlusions originate from lesions of the common iliac bifurcation.

Bilateral external iliac occlusion occurred in only one case.

Occlusion of this artery was first described by Boyd and Jepson (1950). This was a report of two cases in which trauma appeared to be the precipitating factor and it is interesting that the method of treatment adopted was arterectomy and sympathectomy although this report appeared only 14 years ago. One patient (not included in Table 1) has been seen in whom trauma precipitated external iliac occlusion. He was a young soldier who was struck in the left groin by a rifle butt and developed a large haematoma which resolved spontaneously. During convalescence, he noticed that he was claudicating and aortography revealed a left external iliac occlusion.

Another interesting type of external iliac occlusion is that associated with the wearing of a truss. In a recent follow up of patients with known femoro-popliteal occlusions, three elderly men were seen in whom the femoral pulse had disappeared. Each had worn a truss for a reducible inguinal hernia for some years and

the disappearance of the femoral pulse in these cases would appear to be due to occlusion of the external iliac artery caused by the pressure of the truss on an atherosclerotic artery. There was no alteration in the other femoral pulse to suggest an aortic bifurcation lesion and it would seem unlikely that a common iliac occlusion had occurred in the only three patients who wore trusses and developed an absent femoral pulse. It must be assumed that the common femoral artery had remained patent, as the combination of a common femoral and femoro-popliteal occlusion would produce a threat to viability in these elderly patients, whereas, in fact, no gross deterioration of claudication distance had occurred. Unfortunately, the age of these patients precluded verification of the assumed external iliac occlusion by aortography.

This raises the interesting question as to the course to be adopted when a patient with known peripheral arterial disease requests treatment of an inguinal hernia. The prescription of a truss may lead to further occlusion due to pressure but, on the other hand, operative repair of the hernia may be followed by a thrombotic arterial incident during convalescence. The method of treatment adopted has been to undertake operative repair of the hernia with early mobilisation

of the patient, provided he was otherwise fit for operation.

4. Occlusions of the Internal Iliac Artery.

Occlusion of the internal iliac artery as the principal lesion causing symptoms is uncommon (Table 1, p.128) and it is more commonly found in combination with other iliac occlusions or as an incidental finding. Its true incidence is often not appreciated clinically as it is frequently symptomless and overshadowed by occlusion of the main arterial flow to the limb. It may, however, be recognised by the occurrence of buttock claudication and two cases have been seen in which arterial pulsation in the limb was normal but aortography confirmed that internal iliac occlusion was the cause of buttock pain.

The aortogram of one of these cases is illustrated in Fig.94 and indicates the probable origin of internal iliac occlusions from lesions of the common iliac bifurcation. However, at operation for aorto-iliac occlusion, atherosclerotic changes in the internal iliac artery are often severe and it is possible that occlusion may sometimes originate in the proximal 2 cms. of the artery rather than from the bifurcation.

5. Combined Iliac Occlusions.

Plaque formation at the common iliac bifurcation is not infrequent (Figs.46,94) and may give rise to occlusion of different types. The common and external iliac arteries have no branches adjacent to the bifurcation, and the internal iliac artery usually courses for about 1 inch before division. Should occlusion develop at the bifurcation, it is therefore probable that only one artery will be involved, the other two retaining a flow of blood which will maintain patency.

Occlusion of two arteries is unlikely as the third would probably not obtain sufficient collateral flow to maintain patency and the commonest combination of two occluded arteries should be occlusion of the common and external iliac arteries because the internal iliac artery is the most richly endowed with branches of the three. Table 1 (p.128) shows that this is so.

Occlusion of the three arteries is unlikely, as a collateral circulation will develop during the initial period of stenosis and maintain patency of most of the internal iliac at least. This pattern has not been seen except in patients with severe and extensive changes leading to gangrene.

THE ORIGIN OF AORTO-ILIAC OCCLUSIONS.

In Table 1 (p.128), the principal lesions causing symptoms have been classified according to their probable source of origin. Three sites of origin have been postulated (the aortic bifurcation, the common iliac artery itself, and the common iliac bifurcation) because these are the sites at which incipient lesions are most commonly seen. The relative incidence of occlusion originating at these sites suggests that they are of approximately equal importance in the causation of aorto-iliac occlusion although the common iliac artery itself is possibly the most important of the three, Table 2 (p.132) indicating the frequency with which incomplete lesions of this artery occur. It is, however, difficult to be dogmatic on this point as consecutive thrombosis obscures the source of origin of an occlusion and the method applied to analysis of femoro-popliteal occlusions is not possible.

THE COLLATERAL CIRCULATION IN AORTO-ILIAC OCCLUSION.

1. Aortic Occlusion.

Occlusion of the abdominal aorta may be subdivided into two principal types :- those in which occlusion extends up to the level of the renal arteries (Fig.88) and those in which thrombosis is halted at the level of a patent inferior mesenteric artery (Fig.87). The available collateral channels in the two types differ to some extent but the level to which distal thrombosis extends is also important as re-entrant collaterals may be occluded in extensive lesions. When aortic occlusion occurs in a claudicating patient, it is usually possible to assume that both common femoral arteries are patent and that extensive femoro-popliteal occlusion is absent. This assumption is possible because the combination of an aortic and an extensive femoro-popliteal occlusion is liable to produce gangrene except in a very young patient with good collaterals.

The following is a description of the major anastomoses which may play a part in restoring blood flow to the main channel distal to the aortic occlusion. The arteries available for these anastomoses vary according to the proximal and distal levels of occlusion.

The Epigastric Anastomosis.

The superior epigastric artery which is the terminal branch of the internal mammary artery and the inferior epigastric artery arising from the external iliac artery (Fig.93) anastomose in and around the rectus muscle and sheath. The development of a collateral network through these channels is vividly demonstrated at operation when the paramedian incision bleeds freely from numerous vessels and makes the incision a tedious procedure.

The Intercostal - Iliac Anastomosis.

The lower intercostal arteries derived from the thoracic aorta pass into the anterior abdominal wall to anastomose with the epigastric vessels and the circumflex iliac branch of the external iliac artery and also anastomose with the ilio-lumbar branch of the internal iliac artery in the iliac fossa. Additional anastomosis with the vessels taking part in the cruciate anastomosis also occurs.

Lumbar Anastomosis.

A rich network of collateral vessels develops in the posterior abdominal wall with contributions from the phrenic, renal, lumbar and lower

intercostal arteries anastomosing with the parietal branches of the internal iliac artery and the vesical and rectal vessels.

These various anastomoses are developed to a high degree in occlusions which extend up to the level of the renal arteries but all of the collateral pathways are long and tortuous and it is by the summation of a large number of small channels that the lower limbs are nourished.

When occlusion stops at the level of the inferior mesenteric artery, two additional and efficient pathways add their contribution to the collateral network.

The Inferior Mesenteric Anastomosis.

When one is taught as an undergraduate that the inferior mesenteric artery is the artery of supply to the hind gut, it is difficult to comprehend the anastomotic ramifications and importance of this artery. Lindström published an article in 1950 pointing out the importance of this vessel and produced some excellent photographs to illustrate his text.

It is possible at operation for aortic occlusion to appreciate that the inferior mesenteric artery gives rise to small vessels distributed to the

posterior abdominal wall in the presacral region but its major anastomotic contribution is through its anastomoses with the middle and inferior rectal arteries and with the parietal branches of the internal iliac artery supplied to the tissues around the rectum (Figs.8, 82).

The Ilio-Lumbar Anastomosis.

When some lumbar arteries remain patent, an efficient collateral circulation with relatively short wide channels is built up through anastomoses with the lumbar branches of the ilio-lumbar artery and the other parietal branches of the internal iliac artery (Fig.8).

This anastomosis is effective when aortic occlusion stops at the level of the inferior mesenteric artery. Proximal extension of thrombus to the level of the renal arteries occludes the upper lumbar arteries and reduces the effectiveness of this anastomosis although smaller parietal vessels may originate from the aorta above the renal artery and take part in a less efficient ilio-lumbar anastomosis.

2. Common Iliac Occlusion.

In occlusion of the common iliac artery, the anastomoses described above develop to a variable extent.

In bilateral lesions with occlusion on one side and stenosis on the other the collateral circulation is to a large extent the same as in low aortic occlusion. In addition, the patent lower lumbar arteries and possibly the median sacral artery which leave the aorta above the occlusion anastomose below with the ilio-lumbar and parietal branches of the internal iliac arteries. Their contribution, however, may not be large, the median sacral artery especially being affected by atherosclerotic changes in the posterior aortic wall. It is frequently absent or so reduced in size as to be relatively ineffective.

Cross-Midline Anastomosis.

The most important anastomotic circulation which develops in response to iliac occlusion is the "cross-midline" anastomosis between the parietal and visceral branches of the two internal iliac arteries. This is a striking feature of the aortogram in patients with unilateral common iliac occlusion where the artery distal to the block is reconstituted by a flow of blood derived from the intact artery on the other side. It is developed to its fullest extent in the 'younger' patient and is graphically illustrated in Fig.86 where there is almost a ladder appearance as the branches from the

intact side link up with the corresponding branches on the occluded side. In a patient with a unilateral common iliac occlusion and intact internal iliac arteries, this collateral circulation is very efficient and claudication may not be severe. The existence of this extensive network is the probable reason why unilateral common iliac occlusions respond exceptionally well to treatment by sympathetic block whereas aorto-iliac occlusions respond poorly as a whole (Reid, Watt, and Gray, 1961).

The collateral circulation across the mid-line gradually becomes reduced when progressive narrowing of the patent common iliac artery develops. The combination of occlusion of one common iliac artery and narrowing of the other is a potentially dangerous one as the cross-midline anastomosis becomes reduced in efficiency and aortic occlusion will occur when the second common iliac artery thromboses.

The efficiency of the cross-midline anastomosis is also reduced by associated internal iliac occlusion on one or both sides.

3. Occlusion of the Internal Iliac Artery.

When unilateral occlusion of the internal iliac artery occurs, the pelvic viscera are supplied by

the artery of the opposite side. When both internal iliac arteries are occluded, the parietal and visceral branches remain patent and derive an anastomotic supply from the lumbar arteries (ilio-lumbar anastomosis) and from the inferior mesenteric anastomosis.

The bladder and rectum receive sufficient blood for normal function in these cases, although, in acute thrombosis (e.g. saddle embolus), urinary retention or even gangrene of the pelvic viscera may result. In atherosclerosis, however, where occlusion is preceded by a period of stenosis, the collateral circulation is developed sufficiently well as to maintain visceral function.

4. Occlusion of the External Iliac Artery.

When occlusion of the external iliac artery is the sole lesion, a very efficient collateral circulation develops on the lateral wall of the pelvis in which the obturator artery plays the major part (Fig.92).

The obturator anastomosis develops between the obturator branch of the internal iliac artery and the medial circumflex branch of the profunda femoris artery, the two vessels ramifying on the outer aspect of the obturator membrane. The pubic branch of the

inferior epigastric artery (the abnormal obturator) also anastomoses with the obturator artery and helps to reconstitute the main vessel distal to the occlusion.

There is also an anastomosis between the ilio-lumbar artery and the deep circumflex iliac artery and the gluteal arteries anastomose with the lateral circumflex branch of the profunda femoris in the cruciate anastomosis.

B. OCCLUSION OF THE COMMON FEMORAL ARTERY.

Atherosclerotic changes at the bifurcation of the common femoral artery are frequently severe because atherosclerotic lesions show a predilection for the bifurcations of large arteries, but occlusion is uncommon (Table 2, p.132). Occlusion of the superficial femoral artery usually stops at this level because of the high rate of flow into the profunda artery and, similarly, occlusions of the external iliac artery seldom lead to occlusion of the common femoral artery because of the large re-entrant flow through the profunda anastomoses.

Lesions of the common femoral artery are best studied in the aortogram and in the 90 aortograms analysed, there were 9 occlusions of the common femoral artery.

One unusual case has recently been seen in which the patient had patent iliac arteries but bilateral common femoral occlusion was present associated with bilateral femoro-popliteal occlusions threatening the viability of both limbs.

In general, however, common femoral occlusion is uncommon and in extensive aorto-iliac occlusion it is usual to find that the common femoral artery is patent and available for bypass grafting.

C. OCCLUSIONS OF THE FEMORAL AND POPLITEAL ARTERIES.

The femoral and popliteal arteries constitute the main arterial channel from the groin to the popliteal bifurcation and are functionally (though not anatomically) one artery. Although lesions develop at certain specific sites in each vessel, occlusion tends to spread from one to the other and the length of an occlusion is determined by the collateral vessels and not by anatomical divisions.

Fig.52 is the histogram of the 295 femoro-popliteal occlusions found in the 264 patients undergoing bilateral femoral arteriography previously discussed. The upper part of the curve from 30-40 cms. shows relatively little variation and lesions seldom arise in this segment (Fig.56). The lesion illustrated in Fig.50 is uncommon and most of the occlusions involving the upper part of the superficial femoral artery are due to spread of occlusions originating elsewhere.

The relatively flat upper portion of the curve is due to the number of long occlusions which originate in the adductor region and spread upwards to reach the common femoral junction. The occlusions which extended up to the common femoral junction numbered 91 and, of these, 86 measured 20 cms. or more.

It is therefore unusual to have a short occlusion at this level. As the total number of femoro-popliteal occlusions measuring 20 cms. or more was 94, it is also unusual for a long occlusion to involve another part of the femoro-popliteal artery.

Fig.95 is the distribution of the lower level of the 91 occlusions extending to the top of the superficial femoral artery. There is a large peak in the adductor region from 14 cms. upwards although there is a rather surprising number whose lower level lies from 10 to 13 cms. Fifty-five lie between 14 and 25 cms., seventeen lie between 10 and 13 cms. and nineteen lie between 9 and -5 cms. The distribution of these long occlusions suggests that the majority have originated in the adductor region and spread upwards, their length being due to the relative absence of large collateral vessels in the upper half of the superficial femoral artery. The long occlusions which extend down to the popliteal artery may either have originated there or have spread downwards from the adductor region due to secondary thrombosis.

Upward spread of thrombosis is probably the commoner mode of extension and this was graphically illustrated in a case at operation.

J.W., a man of 46 years, had an incipient adductor occlusion (Fig.96) with claudication at 100 yards distance. He was admitted to hospital one week before operation for anticoagulant therapy but this was contra-indicated by a history of duodenal ulcer. At operation, recent thrombus was found to extend from the site of the incipient lesion to the common femoral artery (Fig.97). The thrombus was not adherent and was believed to have developed during his pre-operative stay in hospital.

Types of Femoral and Popliteal Occlusions.

It is difficult to describe an occlusion of the femoral and popliteal arteries seen on the arteriogram in such a way that the reader or listener can visualise the type of occlusion present. Haimovici, Shapiro, and Jacobson (1960) have attempted to classify occlusions of the femoral and popliteal arteries and they describe nine types of occlusion based on a study of 102 consecutive arteriograms in 91 patients. However, they found that 33.3% of cases had combined occlusion patterns with features of two different types of occlusion. In view of the immense variety of these lesions with regard to site and length, any classification must lead to over-simplification and inevitably some occlusions

will have the characteristics of two different groups.

One method would be to describe the occlusion in terms of the distance of its two ends from the level of the knee joint. For example, an occlusion could be described as being at 14-20 cms. from the knee joint, which would indicate that it lay in the adductor region and was 6 cms. in length. The disadvantage of such a method would be the need for measurement as a ruler is not always readily available.

An alternative method would be to classify femoro-popliteal occlusions in groups provided that it was possible to elucidate some specific pattern of occlusion. For example, an occlusion at 14 to 20 cms. could be described as a 'short adductor occlusion' if the adjective 'short' could be defined as a certain range of length of occlusion.

When the length of each occlusion is measured and tabulated as in Table 3, (page 155) it is possible to see that occlusions are most commonly short (1 to 10 cms.) or long (20 cms. or over) although the average length of the femoro-popliteal occlusions studied (295 in number)was 12 cms. Only 39 (13%) occlusions measured between 11 and 19 cms. and only 19 (6%) were 31 cms. or over. The largest group of occlusion was the 'short' occlusions (up to and including 10 cms.) which

accounted for 57% of the total.

In view of the relatively few occlusions between 11 and 19 cms. it would seem possible to describe the occlusions of 10 cms. or less as 'short'. If occlusions measuring over 10 cms. in length were described as 'long' it is probable (Table 3) that they would measure over 20 cms. in length.

TABLE 3.

THE LENGTH OF FEMORO-POPLITEAL OCCLUSIONS.

Length of Occlusion :- cms.	No. of Occlusions.	
1 - 5	88)	57%
6 -10	80)	
11 -15	21)	13.2%
16 -20	18)	
21 -25	41)	23.4%
26 -30	28)	
31 -35	11)	
36 -40	4)	6.4%
41 -45	4)	

When a large number of occlusions are drawn graphically as in Fig.51, some types of occlusion occur frequently and can be recognised. Fig.98 is constructed with the lower level of occlusion on the abscissa and the length of occlusion on the ordinate. The upper level of each occlusion is represented by a dot placed

according to the lower limit and length of each of the 295 femoro-popliteal occlusions.

If a vertical line is drawn between 13 and 14 cms. as an arbitrary division between the femoral and popliteal arteries, all the occlusions on the right of the line lie wholly within the superficial femoral artery i.e. there are 147 superficial femoral occlusions. If a second line is drawn from a point on the ordinate between 18 and 19 cms. (i.e. 13-14 cms. from the knee joint) to meet the base of the vertical line, an oblique line is obtained. The occlusions below and to the left of this line lie wholly within the popliteal artery i.e. 56 occlusions. The occlusions represented in the remaining rhomboidal area are the occlusions involving portions of both the femoral and popliteal arteries i.e. 92 occlusions.

In this figure, a tendency towards the formation of groups can be recognised. The observations lying in an oblique line at the top of the figure represent the 91 occlusions extending to the common femoral junction; the observations on the ordinate are the 32 occlusions extending up from the popliteal bifurcation of which the majority are from 5 to 11 cms. in length; the largest group lies at the bottom right of the figure; and a numerically smaller group lies

between these and the popliteal bifurcation group.

Long Occlusions.

The long occlusions measuring 20 cms. or more lie on or above the horizontal line at 20 cms. in Fig.98. There are 94 in all - six of them measuring 20 cms.

The 91 occlusions extending up to the common femoral junction (Fig.95) lie in an oblique line at the top of Fig.98. 86 of these measure 20 cms. or more which means that only 8 of the 94 'long' occlusions lie elsewhere in the femoro-popliteal artery.

Long occlusions, therefore, usually reach the common femoral junction and it is possible to describe them as 'long superficial femoral occlusions' or 'long femoro-popliteal occlusions' according to whether the lower level of the occlusion lies in the superficial femoral or popliteal arteries.

A long superficial femoral occlusion (Fig.99) lies wholly within the superficial femoral artery (i.e. lies to the right of the vertical line in Fig.98 and measures 20 cms. or more. From this definition, there are 50 such occlusions, and each extends to the common femoral junction.

The long femoro-popliteal occlusions are

those which involve portions of both arteries and measure 20 cms. or more (Fig.117). These are 44 in number and lie in the rhomboid at the upper left of Fig.98.

Two of these affect the total length of both arteries and extend from the common femoral junction to popliteal bifurcation. This extensive type of occlusion is relatively rare in intermittent claudication (2 out of 295 occlusions) but is common in the elderly atherosclerotic patient with gangrene (Fig.79). Because of this it deserves a separate description e.g. total 'femoro-popliteal occlusion'. The use of this term would therefore define an occlusion of the total length of the superficial femoral and popliteal arteries.

This leaves 42 long femoro-popliteal occlusions of which 34 extend up to the common femoral junction. The term 'long femoro-popliteal occlusion' would therefore define an occlusion of 20 cms. or more which would have an 80% (34/42) probability of extending up to the common femoral junction.

The long occlusions can thus be classified into

Long superficial femoral occlusions	- 50
Long femoro-popliteal occlusions	- 42
Total femoro-popliteal occlusions	- 2

Haimovici, Shapiro, and Jacobson (1960) did not differentiate between long and short occlusions in their classification. Occlusions of the superficial femoral artery were proximal (3.6%), distal (17.5%), or 'entire' (15.2%) and occlusions of the popliteal artery were proximal (16%), distal (6.5%) or 'entire' (10.2%). 'Entire femoral and popliteal artery' occlusions (total femoro-popliteal occlusions) totalled 10.2% but their material included only 33% of patients with intermittent claudication, the remainder having rest pain, ulceration, or gangrene, hence the high incidence of these extensive occlusions.

It is difficult to compare their classification with the foregoing as the only 'long' occlusions they appear to describe are the long superficial femoral (entire superficial femoral artery - Haimovici) and the total femoro-popliteal occlusion (entire femoral and popliteal artery - Haimovici), no provision being made for the many long occlusions which extend well down into the popliteal artery (Fig.95).

Short Occlusions.

When the long occlusions of 20 cms. and more are subtracted from the total number of femoro-popliteal occlusions (295) there remain 201 other

occlusions. Of these, 168 measure 10 cms. or less, representing 57% of the total (Table 3, p.155) or 84% of the remaining 201. It seems reasonable, therefore, to designate a 'short' occlusion as one measuring from 1-10 cms. in length especially as the occlusions from 11-19 cms. number only 33 and show no definite tendency to occur in groups.

Examination of Fig.98 suggests that the short occlusions lie in 3 separate groups :- those which extend upwards from a lower level of -5 cms. (21 in number); a second group whose lower level is from 4 to 7 cms. (26 in number); and the largest group whose lower level extends from 10 to 21 cms. (106 in number). To separate off these 3 groups would involve discarding only 15 of the 168 short occlusions, which appears to be acceptable in classification. Eight of these lie around the level of the knee joint and measure 2 to 7 cms. in length but are difficult to introduce into the classification because they are infrequent and variable.

a) Low Popliteal Occlusion.

The first group of 21 occlusions (23 in number if 11 cm. occlusions were included) extends upwards from -5 cms for a variable distance (Fig.98). These are the occlusions which extend upwards from the

popliteal bifurcation and are so frequently limited by the sural arteries (Figs.58, 68).

In this group, there is an aggregation of occlusions (17 in number) measuring 5 to 10 cms. in length corresponding to an upper level of 0 to 5 cms. above knee joint level. These occlusions form an anatomical and clinical group with specific characteristics and could be designated 'low popliteal occlusions' (Distal popliteal occlusion - Haimovici, 1960). They are limited above by a collateral vessel which is usually derived from the sural arteries and extend downwards to the popliteal bifurcation or beyond.

b) Total Popliteal Occlusion.

There are other similar but more extensive occlusions which extend upwards from the popliteal bifurcation and occlude almost the whole length of the popliteal artery (Fig.69). These are few in number in Fig.98 where only six are seen to extend from the popliteal bifurcation to between 9 and 16 cms. from knee joint level. As these occlusions involve almost the whole length of the popliteal artery, they can be called 'total' or 'complete popliteal occlusions' ('Entire popliteal artery' - Haimovici, 1960). They are not often found in patients who have intermittent claudication

because they usually produce severe ischaemia leading to gangrene. The age of a patient with this type of occlusion is of paramount importance as elderly patients have poor collaterals and often develop gangrene (Fig.69) whereas younger patients develop good collaterals and may suffer from claudication only (Fig.100).

c) Short Popliteal Occlusions.

The group of 26 occlusions measuring 1 to 10 cms. in length with a lower level at 4 to 7 cms., involve either the upper part of the popliteal artery alone (Fig.101) or transgress into the lower part of the superficial femoral artery (Fig.98), crossing the oblique line which denotes the arbitrary division between 13 and 14 cms. These occlusions almost separate into two sub-groups - those which are truly short (1-7 cms.) and lie wholly within the popliteal artery (15 in number) and those which are longer (9cms. or more) and involve the lower part of the superficial femoral artery.

Typical examples of this group of short popliteal occlusions are seen in Figs.76 and 101 where they can be seen to lie in the upper part of the popliteal artery above the knee joint, and are limited by well developed collateral vessels.

d) Short Adductor Occlusions.

The third group of occlusions in Fig.98 numbers 106, and their lower level ranges from 10-21 cms. This is not a homogeneous group as those on the left of the scale involve the upper part of the popliteal artery and those with a lower level of 14 to 21 cms. involve the superficial femoral artery only. Secondly, the occlusions at 10 cms. (10-12, 10-12, and 10-14) are overlapped by the longer occlusions of the previous group whose lower level is 7 cms. (7-12, 7-13, 7-16 and 7-17).

However, the majority of this group lie in the adductor region and for this reason it is convenient to describe them as 'short adductor occlusions' (Fig.29). This term infers an occlusion measuring 10 cms. or less which lies principally in the femoral artery between 14 and 21 cms. from knee joint level. The lowest members of this group could with reason be described as 'short femoro-popliteal occlusions' because they also lie in the upper part of the popliteal artery and the upper members of the group could be called 'short superficial femoral occlusions' but there is a disadvantage in trying to produce too complex a classification for lesions which vary so much in site and length.

On the basis of the foregoing classification, it is therefore possible to classify most of the 295 occlusions in the following groups :-

TYPE OF OCCLUSION.		NUMBER OF OCCLUSIONS.
Long superficial femoral occlusion	-	50
Long femoro-popliteal	" -	42
Total femoro-popliteal	" -	2
Low popliteal	" -	21
Total popliteal	" -	6
Short Popliteal	" -	26
Short Adductor	" -	106
TOTAL		253

This classification accounts for 253/295 (86%) of the occlusions of the femoral and popliteal arteries producing claudication. The types of occlusion described are indicated diagrammatically in Fig.102.

THE COLLATERAL CIRCULATION IN OCCLUSIONS OF THE FEMORAL AND POPLITEAL ARTERIES.

The collateral circulation in occlusions of the femoral and popliteal arteries depends on the anastomotic vessels supplied from the profunda femoris artery and on the collateral vessels which arise from the main artery proximal and distal to the occluded segment.

1. The Profunda Femoris Artery.

As the common femoral artery is seldom occluded and the profunda femoris is rarely affected by atherosclerosis, this pathway is of paramount importance to the circulation of the occluded limb. As the profunda femoris descends in the thigh, it gives off numerous muscular branches and also perforating branches which pass to the back of the thigh. The perforating branches anastomose freely with each other and form connections with the cruciate anastomosis above and with the genicular and sural branches of the popliteal artery below.

In the series of 528 arteriograms in 264 claudicating patients there were no occlusions of the profunda femoris artery although occlusion has

occasionally been seen in patients with gangrene. Lindbom (1950) found 4 occlusions of the profunda femoris in 108 limbs showing occlusion and this incidence, which is higher than in the present series, is probably due to the variety of material he included in his survey. Similarly, Haimovici, Shapiro and Jacobson (1960) reported occlusion of the profunda femoris and noted that it was commoner in diabetic than in non-diabetic patients. Singer (1963) in a study of arterial calibre found the profunda artery to be essentially normal in 72% of cases.

Fig.41 shows a profunda femoris artery which branches as it descends, the main stem becoming progressively smaller, and the branches appear to be directed towards the main channel. From this figure, it is possible to understand that the further an occlusion extends downwards from the common femoral junction, the fewer and smaller are the collateral vessels available from the profunda to reconstitute the main vessel.

It is possible to illustrate the importance of the lower level of occlusion in diagram form (Fig.103) where the more distal lesion is seen to make contact with fewer and smaller branches of the profunda femoris.

As a corollary to this, the proximal level of occlusion is of less importance than the distal except

where large collateral vessels are derived from the upper unoccluded segment as in short adductor occlusions (Fig.29). One might therefore expect some correlation between the distal level of occlusion and the patient's claudication distance, but this has not been found.

The richness of the collateral supply through the profunda femoris depends to some extent on the age of the patient. In a 'young' patient (i.e. under 50 years), the profunda femoris may hypertrophy to a remarkable degree (Fig.23) and produce an abundant network of efficient collaterals. This vessel is in marked contrast to the relatively small profunda visualised in the normal arteriogram (Fig.20). Mavor (1952) noted a similar hypertrophy of the profunda femoris in a young man of 33 years who had a long femoro-popliteal occlusion which he believed to have been caused by trauma. In elderly patients, the profunda femoris is less able to respond to occlusion of the superficial femoral artery and, when it also is involved by atherosclerosis as in Fig.73, viability of the limb may be endangered.

The numerous branches of the profunda take part in a number of important anastomoses. The lateral circumflex branch takes part in the important cruciate anastomosis at the great trochanter but also forms anastomoses through its numerous muscular branches and

the artery to vastus lateralis (Fig.78). This branch accompanies the nerve to the same muscle and, because of its length, anastomoses with the muscular branches of the lower part of the superficial femoral artery and with branches of the popliteal artery. The medial circumflex artery anastomoses principally with the obturator artery and its greatest importance is in external iliac or common femoral occlusion (Fig.93). The perforating branches of the profunda are not readily recognised in the ordinary A-P view of the arteriogram but they are of great importance in the supply of blood to the lower leg in femoro-popliteal occlusion.

Although the profunda artery is important in all occlusions in the thigh it is of paramount importance in long superficial femoral, long femoro-popliteal, and total femoro-popliteal occlusions as it is the only major collateral derived from the main artery above the occlusion.

Short Adductor Occlusions.

These are the commonest of all femoro-popliteal occlusions and the cause of their relative shortness is the abundant supply of collateral vessels at this level (Fig.64).

Although the anastomotic supply derived from

the profunda femoris is important, the most important anastomosis in this type of occlusion is that which occurs between the limiting and re-entrant collaterals (Fig.29).

Short Popliteal Occlusions.

These occlusions also tend to be short because of the number of collateral vessels available, and the efficiency of the collateral circulation to a large extent depends on the anastomoses developed between the limiting and re-entrant collaterals (Figs.76, 77).

Low Popliteal Occlusions.

These are limited above by the sural vessels and the tibial arteries are reconstituted at or below the level of the popliteal bifurcation (Figs.58, 68). These lesions are found to respond well to sympathetic block and Fig.104 shows the remarkable number of collateral channels which can develop in this type of occlusion.

Total Popliteal Occlusions.

This type of occlusion eliminates the sural collaterals and also those derived from the medial and

lateral genicular arteries (Fig.69). The main proximal collateral is the descending genicular artery and its saphenous branch. In some cases (Fig.105), the saphenous artery may enlarge sufficiently as to produce a palpable pulse on the medial side of the knee.

The efficiency of the collateral circulation in total popliteal occlusion is poor and reconstitution of the main artery is often absent on the arteriogram (Fig.69). Gangrene is likely to develop and arterial surgery is ineffective because there is no adequate patent distal artery to which an arterial graft can be anastomosed.

D. OCCLUSIONS OF THE TIBIAL AND PERONEAL ARTERIES.

In the 528 arteriograms studied, the arteries of the lower leg showed evidence of occlusion in 241 (45.7%). These occlusions may themselves produce symptoms but, in most cases, their importance lies in their association with femoro-popliteal occlusion and in the effects of the combined lesions.

In the 528 arteriograms,

74(14%) showed no evidence of occlusion or narrowing.

213(40.3%) showed a femoro-popliteal occlusion alone (incipient lesions being included.)

167(31.6%) showed a femoro-popliteal lesion with associated lower leg occlusion.

74(14%) showed a lower leg occlusion only.

The ratio of sole femoro-popliteal to sole lower leg occlusion is 213/74 suggesting that femoro-popliteal occlusion is approximately 3 times more frequently the initial lesion than tibial occlusion.

This 3/1 ratio may not be a true one because femoro-popliteal occlusions produce slowing of the distal circulation and so may predispose to thrombosis of diseased tibial arteries. The reverse is unlikely to be

true i.e. lower leg occlusion is unlikely to produce secondary femoro-popliteal occlusion. Hence, in the 167 with combined occlusions, the tibial occlusion may sometimes be a secondary phenomenon and the initial lesion may have been in the femoral and popliteal arteries. For this reason, the true ratio of the initial site of thrombosis may be greater than 3/1 in favour of femoro-popliteal occlusions.

On the other hand, if incipient femoro-popliteal lesions are ignored, the grouping becomes :-

116 showed no evidence of occlusion.

171 had femoro-popliteal occlusion only.

107 had femoro-popliteal and tibial occlusion.

and 134 had tibial occlusion only.

On this basis, the ratio of sole femoro-popliteal to sole tibial occlusion is 171/134 suggesting that lower leg occlusions are nearly as common as femoro-popliteal occlusions.

The 528 arteriograms were analysed to determine the relative frequency and the pattern of occlusion of the anterior tibial, posterior tibial and peroneal arteries.

In lower leg occlusions there are seven possible combinations, i.e., occlusion of one artery (three types), occlusion of two arteries - (three

combinations) and occlusion of all three vessels. These seven combinations are tabulated in Table 4 to show the relative frequency of the various types of occlusion.

Table 4 :-

Occlusion of the Tibial and Peroneal Arteries.

Type of Occlusion.	Number of Occlusions.	% of Lower Leg Occlusions.	Incidence in Series (% of 528).
Post Tibial -	97	40.2	18.4
Ant.Tibial -	54	22.4	10.2
Peroneal -	7	2.9	1.3
Ant./Post Tibial -	46	19.1	8.7
Post.Tib./Peroneal -	22	9.1	4.1
Ant.Tib./Peroneal -	3	1.2	0.6
A.T./P.T./Peroneal -	12	5.0	2.3
Total	241	99.9%	45.6%

In considering the frequency of the various lesions recorded in this table it must be remembered that these patients were suffering from intermittent claudication only, and that patients with incipient or established gangrene are not included. If the latter were included, the frequency and severity of occlusions in the lower leg would be much higher.

From this table, it is seen that the common types of occlusion in the lower leg are isolated

posterior tibial occlusions, isolated anterior tibial occlusions, and a combined occlusion of the anterior and posterior tibial arteries. These three groups comprise 82.7% of the occlusions found. When only one vessel is occluded it is found that there is a ratio of posterior tibial/anterior tibial/peroneal of 97/54/7, indicating that, when only one artery is occluded, occlusions of the posterior tibial are the most common and that peroneal occlusion alone is unusual. This ratio approximates to 14 : 8 : 1.

When one considers occlusions of the three arteries, either alone or in combination, there were 177 posterior tibial, 115 anterior tibial, and 44 peroneal occlusions. This gives a ratio of 4/2.5/1 and shows that peroneal occlusions are not necessarily rare, although they are uncommon as isolated lesions. This ratio differs from that found by Lindbom (1950) which was 8/4/1 in a total of 147 occlusions, but confirms the relative frequency of occlusion of the three arteries.

The reason for the higher incidence of peroneal occlusions in combination is that many of them are secondary to occlusion of the main stem of the posterior tibial artery or to lesions of the posterior tibial bifurcation.

Because the anterior tibial and peroneal arteries do not share a common stem, a combined occlusion of both arteries is uncommon - 3 in this series.

From Table 4 (p.173), it can be seen that 54.4% of all arteriograms showed no lower leg occlusion ; 29.9% had occlusion of one lower leg vessel ; 13.4% had occlusion of two lower leg vessels ; and only 2.3% had occlusion of all three lower leg vessels.

Comparable figures are few but Linton and Darling (1962) reported occlusion of two lower leg vessels in 22% of 76 patients operated on for claudication and Taylor (1962) showed that the incidence of lower leg occlusion was high in patients operated on for gangrene. He found that 24% of 51 patients had no lower leg occlusion ; 35% had occlusion of two lower leg vessels ; and 41% had occlusion of all three lower leg vessels in some part of their course.

1. OCCLUSIONS OF THE POSTERIOR TIBIAL ARTERY.

The posterior tibial artery commences at the bifurcation of the popliteal artery into anterior and posterior tibial arteries at a level which is variable but is usually about 5-6 cms. below the knee joint. It passes down the back of the leg deep to the gastrocnemius and soleus muscles and gives off muscular branches to these and to the other muscles of the posterior compartment of the leg. It becomes relatively superficial at the ankle where it lies behind the medial malleolus and passes into the sole of the foot to divide into the plantar arteries. Shortly after it takes origin from the popliteal artery it gives off a large branch - the peroneal artery. This artery may be as large as the posterior tibial artery and, in some cases, it may even appear to be the logical continuation of the posterior tibial artery (Fig.25).

In describing arteriograms, confusion may arise concerning the two parts of the posterior tibial artery i.e. the main 'stem' from the popliteal bifurcation to the origin of the peroneal branch, and the posterior tibial artery proper from this level to its division into the plantar arteries. A.K.Henry (1945) has rightly condemned current anatomical nomenclature

and points out that the anterior tibial artery is merely a lateral branch arising from a main stem which is continued down to the junction of posterior tibial and peroneal arteries. He prefers French Nomenclature which describes the popliteal artery as extending to the divergence of anterior and posterior tibial arteries; the main 'stem' of the posterior tibial artery as the 'tibio-peroneal trunk'; and the site of origin of the peroneal artery as the 'tibio-peroneal fork'.

This has proved to be a little clumsy to use and it has been more convenient to describe the first part of the posterior tibial artery before it gives off the peroneal artery as the 'stem' of the posterior tibial artery, to describe the site of origin of the peroneal artery as the 'posterior tibial bifurcation', and to consider the artery from the origin of the peroneal branch to the sole of the foot as the 'posterior tibial artery proper'.

There were 177 occlusions of the posterior tibial artery. These are illustrated in Fig.106 where they have been grouped into 11 different patterns, the relative frequency of these being indicated by the numbers below each diagram.

By far the commonest type is an occlusion of

the posterior tibial artery throughout its length from the posterior tibial bifurcation downwards (Fig.76). This accounts for 81 (45.8%) of the 177 occlusions.

Occlusion of the stem alone, in whole or in part, occurred in 20 cases and in 19 others the stem and artery were occluded from the popliteal bifurcation to the ankle (Fig.107).

Although most posterior tibial occlusions are complete there is sometimes a sufficient blood flow through collateral vessels derived from the peroneal artery to reconstitute the posterior tibial artery just above the level of the ankle joint. This type of occlusion was present in 11 cases and is illustrated in Fig.108.

Finally, in 46 cases only a portion of the vessel was obliterated. In the majority (41) the proximal part of the artery was patent but it diminished in size as it descended and petered out at different levels (Fig.109). In some of these lesions, calcification was found in the lower part of the vessel but gross calcification as illustrated in Fig.108 was rare.

From arteriographic evidence, it is not possible to deduce the site of origin of these occlusions as was possible in femoro-popliteal occlusions, but it seems possible that some of them arise from

atherosclerotic lesions at the popliteal or posterior tibial bifurcations similar to those illustrated in Fig.57. Lindbom (1950) showed that in his material the highest incidence of occlusion in the posterior tibial artery was distal, and it is therefore probable that many occlusions commence distally as in Fig.109. On the whole, the development of collateral vessels following occlusion of the posterior tibial artery is poor and for this reason, occlusion is likely to become complete. The paucity of collateral vessels may be due to the frequency with which gross atherosclerotic changes affect the whole length of the vessel.

Although an isolated posterior tibial occlusion was the commonest type of lower leg occlusion and occurred in 97 legs (Table 4, p.173), it is frequently associated with either an incipient or established femoro-popliteal occlusion. In such cases, the combined effect of the two lesions is liable to produce a greater disability than either occlusion alone.

Another lesion with which posterior tibial occlusion is often associated is an occlusion of the anterior tibial artery - 46 cases (Fig.19). This type of combined occlusion was found to be bilateral in 11 cases

so that these 11 patients with bilateral symmetrical anterior and posterior tibial occlusions account for 22 of the 46 arteriograms in which this lesion was found. In these lesions, the peroneal artery is the sole source of blood supply to the foot and Dible (1956) described this type of lesion as 'peroneal leg' (Fig.118). In his material, which was derived from limbs amputated for gangrene, he found a 'peroneal leg' in 38% of cases. The incidence in the present series of claudicating patients is only 8.7% (46/528), a further indication that lower leg occlusion is less extensive in claudicating patients than in those suffering from gangrene.

There were only 3 cases in which the posterior tibial artery was the sole patent artery in the lower leg (Fig.110), the anterior tibial and peroneal vessels being occluded. Where two of the lower leg vessels are occluded and the third remains the sole supply to the lower leg, the relative frequency of the vessels remaining patent is posterior tibial 3, anterior tibial 22 and peroneal 46. The high incidence of occlusion in the posterior tibial artery and the low incidence of sole patency confirms that atherosclerosis affects the posterior tibial artery more severely than the other two lower leg vessels (Dible, 1956).

Although the posterior tibial artery has such a close relationship to the muscles of the posterior compartment of the lower leg, occlusion may be symptomless in many cases. Where the patient has a femoro-popliteal and posterior tibial occlusion in one leg, and a solitary posterior tibial occlusion in the other, the latter is usually symptomless, the patient complaining of unilateral claudication only. Similarly, if he has a femoro-popliteal occlusion in one leg and a posterior tibial occlusion in the other, the latter is again usually symptomless, because the effect of the femoro-popliteal occlusion in producing claudication is the greater.

The probable reason for this is that the branches to the gastrocnemius and soleus muscles arise principally from the popliteal artery and occlusion proximal to the origin of these sural arteries gives more severe claudication than when only the posterior tibial is occluded.

One aspect which it has not been possible to determine in this survey of arteriograms is the site of pain in the calf muscles in relation to the type of lesion present. The gastrocnemius and soleus muscles are supplied from the sural vessels arising from the popliteal artery above the level of the knee joint and

the lower parts of both muscles are supplied from the posterior tibial and peroneal arteries. One might expect that calf claudication in femoro-popliteal occlusion would be felt principally in the upper part of the calf where the muscle bulk is greatest, and that patients with posterior tibial occlusion only would experience pain in the lower part of the calf i.e. in the soleus muscle. Subsequent observation has shown this to be substantially correct and calf claudication can to a large extent be differentiated into two types - upper and lower - although the latter is relatively uncommon.

2. OCCLUSIONS OF THE ANTERIOR TIBIAL ARTERY.

The anterior tibial artery commences at the popliteal bifurcation and passes forwards above the interosseus membrane to reach the anterior compartment of the leg. It lies deeply in the anterior compartment but becomes more superficial as the muscles become tendinous and at the front of the ankle it lies lateral to the tendon of Extensor Hallucis Longus.

Occlusion of the anterior tibial artery is less common than occlusion of the posterior tibial artery, either as the sole lower leg lesion (AT:PT::54:97) or with regard to the total number of occlusions (AT:PT:115:177). (Table 4, p.173). A relatively large number, however, are bilateral either as the sole lower leg lesion or in combination with other lower leg occlusions. In 10 patients, bilateral anterior tibial occlusion occurred as the sole lesion and, in 21 patients, occlusion was bilateral in combination with occlusions of the posterior tibial or peroneal arteries. Anterior tibial occlusion was therefore bilateral in 31 patients accounting for 62 or rather more than half of the total 115 occlusions.

It is sometimes difficult to determine the exact pattern of occlusion of the vessel because in the

A-P view of the arteriogram, the shadow of the vessel may be superimposed on the cortex of the tibia and difficult to distinguish (Fig.109). In most occlusions the vessel terminates after a short course (Fig.110) at or near the upper level of the interosseus membrane which may be related to its occlusion in a manner similar to the relation of the adductor tendon to femoral occlusion. Incipient lesions are rarely seen at this site but Fig.111 illustrates an anterior tibial artery with an area of narrowing about 2 cms. from its origin.

Measurement on film of the distance between the origin of the artery and the site of occlusion was undertaken and the results are shown in Table 5.(p.185) Precise comparison of distances is not possible because the artery takes origin from the front of the popliteal artery (Henry, 1945) and passes forwards. Hence the image on an A-P film is a foreshortened reproduction whose length on film is of necessity less than that in reality.

Table 5 :-

Occlusions of the Anterior Tibial Artery - length of patent vessel measured on film from the Origin.

Distance of Occlusion from Origin.		No. of Occlusions.
0 (i.e. Total Anterior Tibial Occlusion).	-	39
1 cm.	-	10
2 cms.	-	36
3 cms.	-	14
5 cms.	-	8
10 cms.	-	4
Occlusion at the ankle joint.	-	2
Occlusion of mid-portion.	-	1
Occlusion of the upper segment with reconstitution lower down.	-	1
Total	-	115

Complete occlusion of the vessel was present in 39 cases (33.9%) i.e. 1/3 of the occlusions recorded. In some of these, occlusion was secondary to a popliteal occlusion and there was no evidence of reconstitution. This, however, accounts for only a few of the 39 occlusions as the anterior tibial artery is usually reconstituted by flow through its recurrent branch (Fig.58) when the lower segment of the popliteal artery is occluded. Most of these complete occlusions occurred

where the popliteal artery and posterior tibial stem were patent, and they probably originate from the type of lesion illustrated in Fig.57 where the origin of the vessel is narrowed by an atherosclerotic plaque.

The commonest anterior tibial occlusion is that in which the vessel has a short patent segment and is occluded after a short course (Fig.110). In 60 occlusions, the vessel appeared to be patent for a distance of 1-3 cms. on the arteriogram.

Other types of occlusion are relatively uncommon (Table 5, p.185).

Anterior tibial occlusions are usually symptomless and it is rare for a patient to experience anterior tibial claudication. This is usually due to the presence of femoro-popliteal or posterior tibial occlusions in the same or the opposite leg producing calf claudication. Because the calf muscles have a greater work load in walking than the anterior tibial muscles, anterior tibial claudication is masked. Occasionally, a patient with a unilateral or bilateral anterior tibial occlusion as the sole lesion will experience anterior tibial claudication, and there was one patient with bilateral anterior tibial claudication in the series.

In 22 cases (9.1% of arteriograms showing

lower leg occlusion), the anterior tibial artery was the sole channel of blood flow to the foot. In such cases, the posterior tibial and peroneal arteries are occluded (Fig.107) and the foot is supplied through the malleolar and metatarsal anastomoses of the anterior tibial artery.

3. OCCLUSIONS OF THE PERONEAL ARTERY.

The peroneal artery commences in the posterior compartment of the leg where it takes origin from the posterior tibial artery. In many cases it is the larger of the two vessels and is a more logical continuation of the main vessel than the posterior tibial artery (Fig.25). As it descends, it deviates laterally to the back of the lateral malleolus where it anastomoses with the posterior tibial artery through malleolar branches and gives off a perforating peroneal branch between the malleoli which becomes superficial in front of the lateral malleolus and anastomoses with the malleolar branches of the anterior tibial artery. In some cases of anterior tibial occlusion, the perforating peroneal artery may hypertrophy and pulsation can be felt in front of the lateral malleolus.

The peroneal artery was occluded in 44 arteriograms but occlusion of the artery alone is a rare occurrence (Table 4, p.173). This was present in only 7 arteriograms (1.3% of all arteriograms or 2.9% of lower leg occlusions), and in only 3 of these was it the sole lesion in the leg the other 4 being associated with femoro-popliteal occlusion.

In the majority (34), there was an associated occlusion of the posterior tibial artery and in 3 cases there was associated occlusion of the anterior tibial artery. Peroneal occlusion, therefore, is usually associated with posterior tibial occlusion and as the latter is by far the commonest solitary lesion, it is reasonable to assume that the peroneal occlusion is secondary to the posterior tibial occlusion. Even where peroneal occlusion occurs alone, it probably originates from a plaque at the posterior tibial bifurcation (Fig.57).

In 33 of the 44 cases, the peroneal artery was completely occluded. In 7, the peroneal artery petered out and was occluded in its lower half and in 4 the proximal half of the artery was occluded with reconstitution of the lower half.

In many ways, the peroneal artery is to the lower leg what the profunda femoris artery is to the thigh, yet the analogy is not exact. Lesions of the profunda artery are rare whereas peroneal occlusions do occur. The relative freedom from occlusion of the peroneal artery is probably due to its deep course in which it is protected by muscles, and it is therefore less liable to stress and strain in the same way as the profunda femoris is protected by the thigh muscles.

Occlusion of the artery would be even more infrequent if it were not involved by primary posterior tibial occlusion producing secondary peroneal occlusion.

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Collateral Circulation in Lower Leg Occlusions.

The anastomotic links between the three lower leg vessels are not good except at the ankle and foot. Each vessel may receive important collaterals in low popliteal occlusions, the posterior tibial artery being reconstituted by the saphenous or sural arteries, and the anterior tibial artery through anastomosis via its recurrent artery. On the whole, collateral vessels arising from the lower leg arteries are small and infrequent and occlusion is therefore liable to be complete. The ankle anastomoses are relatively poor, although it has been found difficult to study the details of this region when the method of arteriography used is constructed so as to demonstrate femoro-popliteal occlusions. In some cases, however, the anastomosis between the arteries at the ankle is sufficient to reconstitute the distal portion of an occluded vessel (Fig.108).

CHAPTER NINE.

THE SYMMETRY OF ARTERIAL OCCLUSION.

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Evidence as to the symmetry, or otherwise, of atherosclerotic lesions in the lower limb is conflicting. Dible (1956) found four cases in which the lower leg occlusions were remarkably symmetrical although such findings may be due to the coincidental occurrence of common lesions. Rodda (1953) examined both legs in 10 autopsy specimens and concluded that the degree of intimal disease in both legs was similar but that the distribution was not symmetrical. Lindbom (1950) examined the arteriograms of 17 patients with occlusion in one leg, and found that intimal thickening was most marked at corresponding levels in the other leg in 15 of these and in his bilateral autopsy studies considered that the extent and degree of intimal thickening and of calcification showed a definite symmetry. In established occlusions, however, he found that thigh occlusions were more common on the left side and that lower leg occlusions were more common on the right side. Both observations were statistically significant ($P < 0.02$) and he was unable to offer any explanation.

When viewed from a clinical standpoint,

symmetry appears unlikely as intermittent claudication is most commonly unilateral and, when it is bilateral, one leg is usually more severely affected than the other. Similarly, gangrene is usually unilateral. Only occasionally does the second leg become gangrenous and even then it rarely does so at the same time.

In the present study, symmetrical incipient or established occlusions have been found with sufficient frequency as to suggest that symmetry may be more common than expected. Some of the observations can be explained on the basis of the coincidental occurrence of common lesions as, for example, in Fig.32 where symmetrical incipient lesions are present in the commonest site of all, the adductor canal. Fig.104, on the other hand, illustrates bilateral low popliteal occlusions whose symmetry includes the pattern of distribution of the collateral vessels.

A number of cases have been seen in which arteriograms of both legs of a patient show virtually identical lesions in respect of the site of occlusion and the pattern of the collateral circulation. Symmetry is more likely to be noticed when bilateral femoral arteriography is routine than when only the claudicating leg is x-rayed and the small number of

bilateral studies in the literature may explain the lack of references to symmetrical occlusions.

Fig.112 illustrates bilateral total popliteal occlusions of similar lengths each having a large collateral of similar appearance leaving the main vessel proximal to the occlusion. Fig.70 also shows a roughly symmetrical pattern especially with regard to the developing collateral circulation. Fig.113 shows symmetrical occlusion of all three lower leg vessels below the popliteal bifurcation with reconstitution of the peroneal vessel and Fig.114 also shows a strikingly symmetrical pattern. From these examples and others not illustrated, there seems no doubt that symmetrical lesions do occur although it is difficult to estimate their frequency.

Symmetry is seldom found in the aortogram in cases of aorto-iliac occlusion. Lesions may be present in both common iliac arteries but one side is usually more severely affected than the other (Fig.5). At operation, however, it is often found that the distribution of atherosclerotic lesions is similar on both sides.

Although it is possible to cite examples of symmetrical lesions, one cannot assume from such evidence that most lesions are symmetrical.

However, the evidence in favour of symmetry becomes more provocative when one considers that lesions may be symmetrical in origin but not necessarily simultaneous in time i.e. lesions in the two legs may develop at similar sites but, for local reasons, the one leg is more advanced than the other.

When a patient presents with unilateral intermittent claudication there is frequently an established occlusion in one leg and atherosclerotic changes without occlusion in the other. In such a case, the lesions may have been originally symmetrical but when the patient is seen asymmetry has arisen. At a later date, the unoccluded leg may progress to occlusion and, provided the other leg has not deteriorated, the occlusions may then appear symmetrical.

Fig.115 shows symmetrical femoro-popliteal occlusions which occurred at different times, the left in 1959 and the right in 1960. The occlusions are now symmetrical but between 1959 and 1960 there would be no evidence of symmetry as only one femoral artery was occluded.

Alternatively, the first leg may progress to further occlusion while the second leg remains at an earlier stage.

A.B., a man of 56 years, was seen in 1958 when he complained of bilateral claudication of 6 months duration. Both femoral pulses were normal but the distal pulses were absent on both sides. Shortly after his first visit he developed rest pain in the left foot and sympathectomy failed to give relief. Gangrene developed and below knee amputation of this leg was required. Although the clinical results in the two legs are dis-similar, the original femoral arteriograms (Fig.116) show symmetrical changes. It is probable that he initially had bilateral superficial femoral occlusions and, at a later date, developed an occlusion of the left popliteal artery leading to gangrene and amputation.

Again, there may be symmetrical femoro-popliteal occlusions but the occurrence of a severe lower leg lesion on one side may produce clinical asymmetry.

P.C., a man of 69 years, developed left sided claudication in 1956 following coronary artery thrombosis. When first seen in 1959 he had rest pain in the left foot and subsequently developed gangrene and required amputation. At no time had he complained of his right leg but arteriography showed that he had

symmetrical femoro-popliteal occlusions (Fig.117). The greater severity of symptoms in the left leg was due to the presence of left anterior and posterior tibial occlusions (Fig.118). In this case, therefore, symmetry could not be suspected from his clinical symptoms but the pattern of occlusion in the thighs was similar.

From the above examples, therefore, it is possible that the pattern of atherosclerotic lesions and arterial occlusion is frequently symmetrical but the rate of progression in each leg may be different, i.e. lesions may be symmetrical but not necessarily synchronous.

CHAPTER TEN.

MULTIPLE ARTERIAL OCCLUSION.

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When occlusion develops at one level in the arterial tree, the blood flow distal to the occlusion is reduced and, if the collateral circulation is poor, extension of the original occlusion may occur. On the other hand, there may be severe narrowing of the vessel distal to the occlusion and the consequent slowing of the circulation may precipitate thrombosis at the distal site, thus producing two separate occlusions with a patent portion of the main vessel between. Multiple occlusion may also develop when there is an established occlusion and thrombosis takes place at a second more proximal site. The severity of the symptoms experienced by the patient will, of course, be greater than with a solitary lesion and gangrenous change is therefore more common in multiple occlusions.

The two arterial lesions do not necessarily need to be actual occlusions as the combination of a narrowed segment at one part and an occlusion elsewhere will lead to more severe symptoms than either alone.

J.P., a diabetic of 47 years was first seen in 1958 when he complained of bilateral claudication

more severe in the left leg. The only actual occlusions were right anterior tibial/peroneal and left anterior tibial occlusions (Fig.120). However, there was narrowing of the left femoral artery due to an atherosclerotic plaque (Fig.119) and it appears that the additive effect of the two lesions in the left leg was responsible for the more severe claudication in this leg.

Common examples of multiple occlusion in the same leg are the combination of femoro-popliteal and tibial occlusion and the combination of aorto-iliac and femoro-popliteal occlusion.

COMBINED FEMORO-POPLITEAL AND TIBIAL OCCLUSIONS.

In the series of 528 arteriograms studied, 167 had a femoro-popliteal lesion with associated occlusion of one or more of the vessels of the lower leg. 60 of these were incipient lesions and there were only 107 (20%) arteriograms in which there was an actual femoro-popliteal occlusion with associated lower leg occlusion. In 8 arteriograms there was a double femoro-popliteal occlusion with associated lower leg occlusion.

In the majority of cases, only one lower

leg artery was occluded and this was usually the posterior tibial artery. In none of these claudicating patients was a femoro-popliteal occlusion associated with occlusion of all three lower leg arteries, and it is presumed that such a combination would lead inevitably to gangrenous change.

The combination of a femoro-popliteal with a tibial occlusion is not necessarily serious because where one or more of the lower leg arteries remains unoccluded there is a patent main channel from the knee to the foot and the arterial tree is completely interrupted at only one level i.e. at the femoro-popliteal occlusion.

COMBINED AORTO-ILIAC AND FEMORO-POPLITEAL OCCLUSION.

The combination of an aorto-iliac with a femoro-popliteal occlusion is potentially more serious than a femoro-popliteal/tibial combination, because the main arterial flow is interrupted at two places and the blood flow to the foot is diverted twice into collateral vessels. This combination may therefore produce gangrene, especially in the elderly patient whose collateral circulation is relatively inefficient.

In the series of 90 aortograms studied, the femoral and popliteal arteries were shown to be patent

in 53 (59%). Femoro-popliteal patency in other five patients with aortic occlusion could not be demonstrated by aortography but can reasonably be assumed because aortic and femoro-popliteal occlusion combined would almost certainly lead to gangrene. If this assumption is correct, the distal patency rate would be 64.4%.

The high patency rate of the distal arteries in aorto-iliac occlusion was first noted by De Bakey and his colleagues (1958). Shepherd and Warren (1960) reported a femoro-popliteal patency rate of 59.6% in 52 cases operated on and Singer (1963) found that the femoral arteries were occluded in 25% of patients with aorto-iliac lesions whereas 64% of femoral arteries were occluded in those who had no aorto-iliac occlusion. The low incidence of femoro-popliteal occlusion in patients with aorto-iliac occlusion is probably due to the protective effect of the lowered distal arterial pressure (Singer, 1963).

Analysis of the 90 aortograms shows that femoro-popliteal patency varies according to the type of aorto-iliac occlusion present. The patency rate is high in aortic and common iliac occlusions and low in external iliac, combined iliac and common femoral occlusions.

Table 6 :-

The incidence of proven patency of the Femoral and Popliteal Arteries in 90 cases of Aorto-Iliac Occlusion causing Intermittent Claudication.

Site of Occlusion.	Number of Cases.	Femoro-Popliteal Patency.
Aorta	9	4 (44.4%)
Aortic Bifurcation	18	15 (83.3%)
Common Iliac Stenosis	19	15 (79.0%)
Common Iliac Occlusion	13	9 (69.2%)
External Iliac Occlusion	12	4 (33.3%)
Combined Iliac Occlusions	8	4 (50.0%)
Others	4	1 (25.0%)
Common Femoral Occlusion	7	1 (14.3%)
Total	90	53 (59%)

CHAPTER ELEVEN.

CLINICAL ASPECTS OF INTERMITTENT CLAUDICATION.

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CLINICAL ASPECTS OF INTERMITTENT CLAUDICATION.

Intermittent claudication in the lower limb may affect any group of muscles to which the arterial inflow is reduced. It is most commonly felt in the calf muscles because of the frequent occurrence of femoro-popliteal occlusions and because the calf muscles perform the greatest amount of work during walking.

Pain may be experienced in other muscle groups concurrently with calf pain where these muscles have a deficient arterial supply e.g. the thigh muscles in aorto-iliac occlusion, the small muscles of the foot in tibial or plantar occlusions.

In other cases, the main arterial flow is not reduced and claudication develops in muscle groups affected by occlusion of lateral channels e.g. buttock claudication in internal iliac occlusion, anterior tibial pain in anterior tibial occlusion.

The rate of onset of pain depends on the size of the deficit in arterial supply to the muscles and on the amount of work they are required to undertake. The distance a patient can walk before experiencing pain is therefore variable and depends on

the speed at which he walks and whether the ground is level, uphill, or downhill. Hillestad (1963) found experimentally that the most important factors influencing the claudication distance were the rate of walking and the weight carried by a patient.

Boyd (1949) attempted to classify intermittent claudication into three types :-

Grade 1 - a cramp which does not force the patient to stop and which can be walked off.

Grade 2 - a claudication distance of 50 feet or more.

Grade 3 - a claudication distance of less than 50 feet.

Most patients fall into class 2 and the classification has not been generally adopted.

Intermittent claudication is almost invariably due to atherosclerotic occlusion of a major artery and the arterial deficit depends on the site and extent of occlusion and on the efficiency of the collateral circulation developed round the occluded segment. The development of occlusion and the degree of arterial insufficiency are affected by various factors such as age, sex, diabetes mellitus, cardiac insufficiency, anaemia, hypertension and, possibly smoking.

A. ASSOCIATED FACTORS IN INTERMITTENT CLAUDICATION.

1. Age.

Atherosclerotic arterial occlusion in the lower limb is uncommon below the age of 40 (Fig.121) and becomes increasingly common in older patients. Rodda (1953) found a 40% incidence of arterial occlusion in the limbs of non-ischaemic patients over 60 years of age examined at post-mortem. Occlusion was multiple in 14% of patients and Lindbom (1950) also commented on the multifocal nature of occlusion in ischaemic patients over 60 years.

When the age of onset of symptoms is considered in decades, the largest group of patients with intermittent claudication is aged 50-60 (Fig.121), a finding which accords with that of Taylor and Calo (1962). In Fig.121, the percentage distribution by age groups of 67 patients with aorto-iliac occlusion and 305 patients with femoro-popliteal or tibial occlusion examined between 1957 and 1960 has been charted. The mean age of the two groups is 53.6 years and 57.5 years respectively. Patients with aorto-iliac occlusion tend to be younger and this group accounted for 30% of the patients under 50 years although forming only 18% of the total number of patients. This tendency to an

earlier age of onset for aorto-iliac occlusion favours a high operability rate in these lesions which are technically easier to deal with than femoro-popliteal occlusions.

In the 264 patients who underwent bilateral femoral arteriography, an analysis of the incidence of occlusion in relation to age has been undertaken.

In this group, the mean age at onset of symptoms was 58.6 years. There was no significant difference in the age distribution in three groups of patients :- 80 patients with bilateral claudication - mean 58.6 years ; 94 with right sided claudication - 58.8 years ; and 90 with left sided claudication - 58.6 years. The distribution of ages within these three groups was also similar.

The youngest patient was 27 years old and the oldest 81 years old. Almost half (46.2%) were between 55 and 64 years (inclusive) and 16% were below 50 years of age.

To facilitate analysis of the effect of age on the incidence of occlusion, three age groups of reasonable size were considered :- a) less than 54 years; b) 55 to 64 years; and c) over 65 years. The incidence of occlusion in the symptomless leg of

patients with unilateral claudication, the incidence of tibial occlusion in the claudicating leg of patients with unilateral claudication, and the incidence of tibial occlusion in patients with bilateral claudication were analysed with respect to these three age groups.

Table 7 :-

The incidence of arterial occlusion in the symptomless leg of 184 patients with unilateral claudication, analysed with respect to age.

Age Group Years.	Number in Group.	Number with Occlusion.+	Incidence %.
< 54	51	25	49
55-64	91	54	59.3
65 +	42	33	78.6
TOTAL	184	112	60.9

+ This column includes 20 incipient femoro-popliteal Occlusions.

In the symptomless leg of 184 patients with unilateral claudication, the incidence of occlusion was 60.9%. This rose steeply with increasing age and in elderly patients it approached 80% (78.6%).

Table 8 :-

The incidence of tibial occlusion in the claudicating leg of 184 patients with unilateral claudication analysed with respect to age.

Age Group Years.	Number in Group.	Number of Tibial Occlusions.	Incidence %.
< 54	51	13	25.5
55-64	91	35	38.5
65 +	42	29	69.0
TOTAL	184	77	41.8

The incidence of tibial occlusion in the claudicating leg of these patients also shows a rise with age and is 69% in the oldest group.

Table 9 :-

The incidence of tibial occlusion in 80 patients with bilateral claudication analysed with respect to age.

Age Group Years.	Number in Group.	Number of Legs.	Number of Tibial Occlusions.	Incidence %.
< 54	24	48	22	48.8
55-64	38	76	37	48.7
65 +	18	36	29	80.6
TOTAL	80	160	88	55

The incidence of tibial occlusion in patients with bilateral claudication also increases with age although there is no difference between the first two age groups. The incidence in patients over 65 years is high - 80.6% - and, indeed, 88.9% of patients in this age group had a tibial occlusion in one or both legs i.e. only 11.1% of these elderly patients had no tibial occlusion in either leg.

In the three factors analysed, the incidence of arterial occlusion rose steeply with age as found by Lindbom (1950) and Rodda (1953) in autopsy studies. This high incidence of lesions indicates the widespread

nature of the arterial changes and would suggest that few elderly patients are really suitable for major surgery.

2. Sex Incidence.

Intermittent claudication is approximately 6 times commoner in men than in women (Hines and Barker, 1940). This sex incidence is largely unexplained but there is evidence to suggest that oestrogen may prevent the full development of atheroma and thus exert a protective effect in women. Experimentally, cockerels in whom atheroma has been induced by a high cholesterol diet show some regression when oestrogen is also given (Pick and Colleagues, 1952) and women who have undergone bilateral adrenalectomy and oophorectomy for breast carcinoma have a significantly greater incidence of atherosclerosis than normal women (Rivin and Dimitroff, 1953). The use of oestrogen in treatment, however, has proved disappointing.

3. Diabetes Mellitus.

This condition is associated with hypercholesterolaemia and patients are known to have a higher incidence of atheromatous lesions and to develop these at an earlier age. Hines and Barker (1940)

recorded an incidence of 20.3% for all types of ischaemia but three-quarters of their patients with diabetes mellitus were suffering from gangrene. The importance of the association between diabetes mellitus and gangrene is undoubted but the incidence of diabetes in patients with claudication is relatively low.

Of the 264 men with femoro-popliteal occlusion and claudication, only 12 (4.5%) had diabetes mellitus and Bloor (1961) quotes an incidence of 4% in the 1476 cases of intermittent claudication he analysed. Although the incidence of diabetes mellitus in patients with intermittent claudication is low, urine testing for glycosuria should be a routine in patients with arterial disease.

4. Cardiac Insufficiency.

Although arterial occlusion is the principal cause of the arterial deficit, the blood flow to claudicating muscles may also be affected by cardiac insufficiency which produces a lowering of arterial pressure and reduces the rate of input to the leg. Cardiac insufficiency is important in several ways.

The onset of claudication may result from the development of a moderate degree of congestive cardiac failure. In such cases, the peripheral pulses

may be readily palpable and atherosclerotic changes without occlusion are found on arteriography. In these cases, intermittent claudication is due to a poor cardiac output and it is sometimes possible to improve the claudication by treatment of the cardiac condition.

The importance of cardiac insufficiency is also seen in the acute cardiac incident (e.g. coronary artery thrombosis). If there is a pre-existing arterial occlusion in a limb and the arterial pressure falls drastically, an acute deficiency in the arterial flow to the foot may occur and the patient will develop a cold, pale, painful limb similar to that found in embolus. With improvement in the cardiac condition, recovery of the limb is possible.

Cardiac insufficiency is important for a third reason. Those who develop acute cardiac insufficiency due to coronary artery thrombosis or congestive failure are confined to bed. The blood pressure falls and, while in bed, the circulatory rate is slowed. Conditions are then suitable for the development of thrombosis, which may, of course, be venous but, where the arterial system is already affected by atherosclerosis, arterial occlusion is possible. This may be sufficiently extensive as to precipitate gangrene but, if not, the patient experiences

no symptoms until his convalescence when he finds that he has developed intermittent claudication. Many patients, therefore, give a history of a cardiac incident or other illness enforcing a period of bed rest immediately prior to the onset of symptoms. Because of this liability to thrombosis in bed, it is wise to encourage mobility of the claudicator when he is ill.

Angina of effort is a symptom of reduced coronary artery blood flow to the heart muscle and is analogous to intermittent claudication. It is an indication of generalised disease and although both angina and claudication may be experienced by the patient at approximately the same distance, one tends to compel the patient to halt before the other is experienced. Where both are present, it is undesirable and possibly dangerous to improve a patient's claudication distance when angina will not allow the patient to walk any further.

In patients with cardiac insufficiency, therefore, a careful assessment of the clinical state is necessary before treatment is undertaken. Symptoms of cardiac decompensation, severe dyspnoea on exertion, or severe angina of effort preclude treatment of the

claudication and treatment should be directed towards improving the cardiac condition. As the cardiac state improves, improvement in claudication may result.

A moderate degree of cardiac insufficiency, previous coronary thrombosis or electrocardiographic changes do not preclude the use of simple methods of treatment such as phenol sympathetic block, and need not necessarily be a contra-indication to direct surgery on the femoral and popliteal arteries where the operative risk is low. They may, however, contra-indicate operation for aorto-iliac occlusion where the operative risk is higher.

5. Anaemia.

The presence of anaemia reduces the oxygen-carrying capacity of the available blood flow and may reduce the distance a claudicating patient can walk. It is, however, rarely the cause of symptoms (Allen, Barker and Hines, 1955). Only one case of intermittent claudication due to anaemia has been encountered.

J.M., a man of 59 years, was seen in 1958 when he complained of right sided claudication at 100 yards distance of 6 months duration. Both femoral pulses were normal but a poor left dorsalis pedis pulse

was the only distal pulse palpable. He was believed to have bilateral femoral occlusions but arteriography showed atherosclerosis without occlusion in either leg. His haemoglobin was 8.2 G/100ml. which was found to be due to an iron deficiency anaemia. Oral iron produced a rapid improvement and when his haemoglobin returned to normal he was able to walk as far as he wished. When last seen in 1961, he was in excellent health and had had no further claudication.

Intermittent claudication due to anaemia alone is uncommon, but Pickering and Wayne (1934) described the occurrence of claudication in seven out of 25 grossly anaemic patients whose haemoglobin level was less than 50% normal and in whom arterial occlusion was absent. Claudication was abolished in six of the seven patients by treatment of the anaemia.

Although anaemia is rarely the cause of intermittent claudication, it is likely to accentuate a patient's disability and routine estimation of haemoglobin should be undertaken in patients with intermittent claudication.

6. Hypertension.

Hypertension is a relatively common finding

in patients with atherosclerotic arterial occlusion (Hines and Barker, 1940 ; Singer and Rob, 1960). In the 264 men with femoro-popliteal occlusion, 58 (22%) were found to have a raised blood pressure of 160/100 m.m. Hg. or more. Bronte-Stewart and Heptinstall (1954) showed that hypertension accelerated the rate of onset and severity of atheroma in the aorta of cholesterol-fed rabbits, and it is likely that hypertension increases the severity of atherosclerosis in man. Richards (1957) has shown that hypertension is important in prognosis because hypertensive patients with arterial occlusion in the lower limb had a poorer expectation of life than those without hypertension.

7. Smoking.

Although smoking has been incriminated as a possible aetiological factor in the development of lung cancer and is usually accepted as an important aetiological factor in Buerger's disease, little has been said with regard to its association with atherosclerotic occlusion in the limbs.

Patients who attend peripheral vascular clinics are almost invariably smokers and, in the 264 men with femoro-popliteal occlusion, only one was a non-smoker. Juergens, Barker and Hines (1960) found

that only 2.5% of 401 patients were non-smokers. Although the significance of such figures is as yet unproved, Juergens, Barker and Hines found that 11.4% of those who continued to smoke developed gangrene and required amputation, whereas no patient who stopped smoking required subsequent amputation.

Whether or not smoking is of aetiological significance, there is no doubt that nicotine is a potent vaso-constrictor and is best avoided. Patients with severe ischaemia should be strongly advised to stop and at the same time should be warned to avoid putting on weight which is the usual sequel to the cessation of smoking.

Many patients, however, are unable to stop and must be regarded as compulsive smokers. It is not uncommon to see patients with a gangrenous limb puffing contentedly at a cigarette which is contracting the remaining collateral vessels.

B. THE PATTERN OF THE PERIPHERAL PULSES.

In the examination of patients with arterial occlusion, palpation of the peripheral pulses is essential for the confirmation of the diagnosis and assessment of the level of occlusion. Accuracy in elucidation of their pattern is of great importance and certain variations and fallacies must be noted.

Pulses may be really palpable at certain times and apparently absent at other times. They are most readily felt during vaso-dilatation but on a cold day may sometimes become impalpable.

When it is difficult to decide whether a pulse is present or not the pulse should be checked against the radial pulse to ascertain that the two are synchronous and that the observer is not feeling pulsation arising in the tip of his own finger.

Ludbrook, Clarke, and McKenzie (1962) stress that observer error occurs even with skilled clinicians and concluded that if three independent observers were unable to detect the posterior tibial pulse there was a high degree of probability that an abnormality was present. Absence of the posterior tibial pulse is regarded as a significant finding indicating probable arterial occlusion and they found this pulse to be

"normally" absent in only 0.2% of patients under 20 years of age.

Absence of the dorsalis pedis pulse, on the other hand, may not necessarily be significant. Ludbrook and his colleagues were unable to palpate this pulse in 8.7% of normal patients under 20 years of age although the proportion of 'normally' absent pulses rose in older patients. In some cases, there is hypertrophy of the perforating branch of the peroneal artery with palpable pulsation in front of the lateral malleolus but Ludbrook, Clarke and McKenzie noted this in only 20% of the patients in whom the dorsalis pedis pulse was absent.

The four pulses normally examined are the femoral in the groin, the popliteal in the popliteal fossa, the posterior tibial behind the medial malleolus, and the dorsalis pedis on the dorsum of the foot.

The Femoral Pulse.

The femoral pulse is the most easily felt and the most important because examination of both femoral pulses permits a clinical differentiation of the two main groups of occlusion - aorto-iliac and femoro-popliteal.

A normal femoral pulse indicates the absence

of serious aortic or iliac disease although it does not exclude mild proximal narrowing or internal iliac occlusion. Occasionally, the femoral pulse is normal in volume but on compression a thrill is felt which may be due to proximal narrowing of the iliac arteries. In such cases a bruit may be audible with the stethoscope.

A weak femoral pulse indicates the presence of proximal narrowing in the aorta, common, or external iliac arteries or a short occlusion with a good collateral circulation.

An absent femoral pulse indicates proximal arterial occlusion. If one femoral pulse is normal and the other is absent, the occlusion is unilateral. Unilateral absence of the femoral pulse can be assumed to be caused by common iliac occlusion until proved otherwise, because common iliac lesions are commoner than those in the external iliac artery (Table 2, p.132).

When both femoral pulses are absent, it can be inferred that aortic occlusion is present as bilateral iliac lesions rarely give absence of both pulses. The level to which aortic occlusion has spread cannot be determined clinically and aortography will be required to demonstrate the level of occlusion.

The absence of one femoral pulse and weakness of the other usually indicates an aortic

bifurcation lesion with unequal involvement of the common iliac arteries, but may also be present in bilateral common iliac lesions arising in the common iliac artery itself.

When both femoral pulses are weak it may reasonably be assumed that there is an aortic bifurcation lesion producing bilateral narrowing of the orifices of the common iliac arteries. Some difficulty in clinical diagnosis may arise when both femoral pulses are reduced in volume to a equal degree, as the reduction may be slight and it may then be difficult to distinguish pulsation from normal.

The Distal Pulses.

In charting the pulses, it is customary to use a simple table such as the following which may be constructed horizontally or vertically.

PULSES.

	Femoral	Popliteal	Posterior Tibial	Dorsalis Pedis
Right Leg :-	+	+	+	+
Left Leg :-	+	±	-	-

\+ indicates a pulse of normal volume.
± indicates diminished pulsation.
\- indicates an absent pulse.

This chart is usually contracted to :-

	F.	P.	P.T.	D.P.
R. -	+	+	+	+
L. -	+	±	-	-

and by constant usage in the same sequence it can be contracted to $\begin{matrix} + & + & + & + \\ + & \pm & - & - \end{matrix}$ the right leg always being placed above the left and the posterior tibial preceding the dorsalis pedis.

When the femoral pulse is normal, the different patterns found, their frequency and significance are as follows :-

+ + + + : The finding of four normal pulses indicates the presence of patent arterial channels from aorta to the ankle. The possibility of narrowing due to plaque formation in the adductor region or elsewhere is not excluded by this finding and such lesions may progress in time to complete occlusion. In patients with unilateral claudication the presence of normal pulses in the symptomless leg may lead to the erroneous conclusion that this leg is free of disease whereas the artery may be narrowed although not yet occluded.

If the patient has four palpable pulses in the leg yet experiences claudication in the calf, the possibility of cardiac insufficiency or anaemia must be

suspected and excluded by appropriate investigation.

It is also possible for the patient with normal pulses to have claudication in the muscles of the foot due to plantar artery occlusion. This is rare but it has been seen on two occasions, and can give rise to difficulty in diagnosis.

C.T., aged 67 years was seen in 1962 when he complained of pains in both feet on walking 200-300 yards. The pain appeared to develop earlier when walking uphill and was relieved by rest. All pulses were present in both legs and the feet were warm and well coloured with no pallor on elevation. As the pain appeared to be a typical claudication, possibly due to plantar artery occlusion, bilateral sympathetic block was performed with excellent temperature responses but with relatively little alleviation of claudication. Arteriography was contra-indicated by age and fitness and doubt arose as to the diagnosis. However, the development of a right femoro-popliteal occlusion 3 months later appeared to confirm the initial clinical diagnosis.

The doubts experienced in diagnosis and the difficulty of adequate demonstration of plantar occlusion by arteriography indicate that one must be

prepared to accept a typical history even where normal pulses are present.

+ - - - : This is the commonest pulse pattern found in patients with intermittent claudication. It was present in 248 legs (47% of 528 legs of 264 patients with occlusion below the common femoral artery), and was bilateral in 52 patients. 38 of the 80 patients with bilateral claudication showed this pattern in both legs. (Table 10).

Table 10 :-
The frequency distribution of the various pulse patterns found in femoro-popliteal and tibial occlusion.

	Bilateral Claudication 80 patients.	Unilateral Claudication 184 patients.		
Pulse Pattern.		Claudicating Leg.	Symptomless Leg.	Totals.
+ + + + :	9	6	77	92
+ - - - :	91 (38 Bil.)	137 (14 Bil.)	20	248
+ + - - :	33	22	32	87
+ + - + :	5	5	9	19
+ + + - :	14	6	39	59
+ - + + :	3	2	0	5
+ - + - :	3	6	5	14
+ - - + :	2	0	2	4
Totals :	160	184	184	528

This pulse pattern usually indicates occlusion of the femoral and/or popliteal arteries. It does not necessarily indicate an extensive occlusion or one with poor collateral vessels as it occurs with most short occlusions. It may also be present where the artery is narrowed but not occluded, especially in elderly patients.

+ + - - : This pattern was the second commonest abnormal pattern found (Table 10, p.227). It usually indicates the presence of a low popliteal occlusion at or below the level of the knee joint or the presence of combined anterior and posterior tibial occlusions in the lower leg. It was occasionally found in association with a short or 'incipient' femoro-popliteal occlusion.

This pattern was occasionally found in patients with no lesion in the arteriogram or in elderly patients, and may be due to diffuse tibial atherosclerosis in these patients or to observer error.

+ + - + : This pattern occurred in 19 legs and was usually due to the presence of an isolated posterior tibial occlusion.

+ + + - : This was present in 59 legs and in many

cases indicated an isolated anterior tibial occlusion. However, it was also found when the patient had an apparently intact anterior tibial artery on the arteriogram. The presence of this pattern in so many cases may be due to the vessel expending its resources in the malleolar anastomoses leaving a small impalpable dorsalis pedis artery.

+ - + + : These three combinations are uncommon
+ - + - : (Table 10, p.227). They may be associated
+ - - + : with short superficial femoral occlusions and, in the latter two, additional anterior or posterior tibial occlusion. On the other hand, the apparent absence of the popliteal pulse may be in many cases an observer error and the true record would be + + + +, + + + -, and + + - +.

In the classification in Table 10, no account has been taken of diminished volume of pulsation and each pulse has been recorded as either present or absent. Diminution of the distal pulses is frequently recorded and usually indicates the presence of a short occlusion with excellent collateral vessels lying between the diminished pulse and the more proximal normal pulse, or the presence of an incipient occlusion with

narrowing of the artery. The return of weak pulsation in a vessel distal to an occlusion may also be noted following a successful sympathectomy or phenol sympathetic block and is frequently associated with marked symptomatic improvement.

Simple palpation of the pulses can therefore provide important information which will confirm the diagnosis of arterial occlusion, differentiate aorto-iliac from femoro-popliteal occlusion, and possibly indicate the site of arterial occlusion.

There is however, a limit to the deductions which can be drawn from the pulse pattern with reference to the site of occlusion. The upper level of occlusion can be assumed to be distal to the lowest normal pulse but where both femoral pulses are absent as in aortic occlusion no conclusion as to the upper limit of spread can be reached other than that it must lie below the renal arteries. The pulse pattern also gives no information as to the lower level of an occlusion or to the presence of associated occlusions.

Arteriography is therefore necessary to delineate the extent of the principal lesion and to reveal the presence of other associated occlusions.

C. ANALYSIS OF FEMORO-POPLITEAL AND TIBIAL OCCLUSIONS IN RELATION TO SYMPTOMS.

In the series of 264 patients with occlusion below the common femoral artery, 80 (30.3%) presented with symptoms of bilateral claudication, 94 (35.6%) with claudication of the right leg and 90 (34.1%) with claudication of the left leg. The three groups, therefore are comprised of approximately equal numbers.

The femoro-popliteal occlusions were subdivided into 'incipient' occlusions - those in which severe narrowing of the artery was present ; 'short' femoro-popliteal occlusions measuring 10 cms. or less ; 'long' femoro-popliteal occlusions measuring over 10 cms. in length ; and 'double' femoro-popliteal occlusions where two separate occlusions of the femoral and popliteal arteries were present. In recording lesions of the tibial vessels, only actual occlusions have been noted and narrowing of the tibial or peroneal vessels has been ignored in this analysis.

BILATERAL CLAUDICATION.

In the 80 patients (160 legs) with bilateral claudication, two legs were found to have no occlusion. This suggests that these two patients should really be

classed as cases of unilateral claudication although the patients were adamant that symptoms were bilateral.

Incipient, short and long femoro-popliteal occlusions were almost equally represented (Table 11).

Table 11 :-

The distribution of types of occlusion in relation to symptoms in 264 patients undergoing bilateral femoral arteriography. The figures in brackets refer to the number of lower leg occlusions associated with the various types of femoro-popliteal occlusion.

Type of Occlusion.	Unilateral Claudication 184 Patients.		Bilateral Claudication.
	Claudicating Leg.	Symptomless Leg.	80 Patients- 160 Legs.
No Occlusion	0	72	2
Incipient Femoro-Popliteal	22(11)	42(17)	43(32)
Short Femoro-Popliteal	81(25)	8(4)	48(19)
Long Femoro-Popliteal	55(24)	15(8)	44(19)
Double Femoro-Popliteal	16(7)	0	6(1)
Tibial	10(10)	47(47)	17(17)
Total	184(77)	184(76)	160(88)

The incidence of concurrent tibial occlusion is over 50% (88/160) and is very high in those with incipient occlusion (32/43) - nearly 75%. This is probably

because incipient femoro-popliteal occlusion without tibial occlusion tends to be masked by occlusion in the other leg and the patient would then complain of unilateral, not bilateral, claudication.

It is of interest that the six patients with a double femoro-popliteal occlusion were all under 65 years of age, and those patients with long occlusions and associated occlusion of two lower leg arteries were also under 65 years of age. This suggests that double femoro-popliteal occlusion and the combination of a long femoro-popliteal occlusion with occlusion of two lower leg arteries are serious lesions in elderly patients which are not associated with claudication because they are liable to produce gangrene.

UNILATERAL CLAUDICATION.

In unilateral claudication, arterial occlusion is frequently bilateral. Only 72 of 184 patients were without evidence of lesions in the symptomless leg (Table 11, p.232), and 112 patients, therefore, had a femoro-popliteal or tibial occlusion in the symptomless leg. Irregularity of arterial outline due to atherosclerosis was evident in those without occlusion.

It must be presumed that claudication was unilateral in these 112 patients because of the masking effect of a more severe lesion in the claudicating leg which forced the patient to halt before symptoms could develop in the less severely affected leg.

The common types of occlusion in the claudicating leg were short femoro-popliteal occlusions - (81/184) - 44%, and long femoro-popliteal occlusions (55/184) - 30%. These two types accounted for approximately 75% of the lesions producing unilateral claudication.

In the symptomless leg, the common lesions were lower leg occlusion - 47 cases (26%), and incipient femoro-popliteal occlusion - 42 cases (23%).

The incidence of lower leg occlusions in the two legs is approximately the same - 77/184 in the claudicating leg and 76/184 in the symptomless leg. It is surprising to find that the incidence of tibial occlusion in the symptomless leg is as high as that of the claudicating leg.

What is also surprising is that the incidence of lower leg occlusion associated with femoro-popliteal occlusion is approximately 50% for all types of occlusion except short occlusions where the incidence is only 25/81 - 31%. Such a high incidence suggests that

tibial occlusion in patients with intermittent claudication is more common than is usually believed to be the case.

D. SIDE OF OCCLUSION.

One of the interesting observations made by Lindbom (1950) was that thigh occlusion was commoner in the left leg than the right and that lower leg occlusion was commoner in the right lower leg than the left. He found a statistical difference ($P < 0.02$) in favour of the left thigh and the right lower leg, but was unable to offer any explanation.

In the present material (264 cases) no such difference was found, the femoro-popliteal occlusions being divided as to 135 Right and 138 Left and the lower leg occlusions 118 Right and 128 Left. Where a double femoro-popliteal occlusion was present, this has been counted as a single lesion for this analysis and incipient lesions have been excluded. These figures indicate a slight preponderance in favour of the left thigh and left lower leg but in neither is the difference significant. This suggests that Lindbom's was an unusual chance finding.

E. RELATIONSHIP BETWEEN SITE OF OCCLUSION AND CLAUDICATION DISTANCE.

The clinical impression has been formed that some relationship exists between the site of occlusion and the initial claudication distance i.e. patients with minor occlusions had a relatively slight handicap and patients with long or multiple occlusions could only walk a short distance.

The 264 patients with occlusion below the common femoral artery were analysed. Those with bilateral claudication were excluded because of difficulty in analysis, and the patients with unilateral claudication due to femoro-popliteal occlusions only (i.e. those with associated lower leg occlusion were excluded) were subdivided according to the length and site of occlusion. There were 103 of these patients and a further exclusion of 11 elderly patients was made.

In these 92 patients, no correlation could be found between the claudication distance and the level of occlusion. These 92 patients were compared with the 62 patients of similar age in whom femoro-popliteal and tibial occlusion was present and there was no evidence of any significant difference between the claudication distances of the two groups.

In so far as the reflex vaso-dilation test is a measure of the collateral circulation available in each patient and an index of the temperature improvement obtainable by sympathectomy, attempts were made to correlate the initial claudication distance with the reflex vaso-dilation test and also to correlate the improvement in claudication distance obtained by phenol sympathetic block with the reflex vaso-dilation test. No correlation was found in either.

The absence of correlation between these various entities is probably because the claudication distance of each patient is subject to the effects of many factors and no consistent pattern occurs. Hillestad (1963) was unable to show any correlation between the blood supply to the calf muscles (measured by the plethysmograph) and the walking distance. He also compared the walking distance on a treadmill under standard conditions and found a great variation in the walking capacity of patients tested on successive days.

No inference as to the severity of the lesion is therefore possible from consideration of the claudication distance alone. Although it is true that patients with aortic occlusion, severe multiple occlusion, or ischaemia verging on gangrene have a

restricted walking distance of 50 yards or so, it cannot be assumed that patients with a claudication distance of 50 yards have a serious occlusion, because, in practice, many have relatively short occlusions with, apparently, a good collateral supply.

On the other hand, it is possible to assume that patients with a mild disability (i.e. claudication distance of ½ mile or more) are likely to have a short occlusion with good collaterals or have narrowing of the vessel only.

CHAPTER TWELVE.

TREATMENT OF INTERMITTENT CLAUDICATION.

CHAPTER TWELVE.

TREATMENT OF INTERMITTENT CLAUDICATION.

Although intermittent claudication is readily diagnosed because of its typical relationship to exercise, it is only a symptom and clinical evidence of arterial occlusion must be sought by examination of the pulses.

The presence of normal arterial pulsation makes the diagnosis doubtful and in such cases, intervertebral disc and spinal lesions, osteo-arthritis of knee or hip, prostatic carcinoma, foot strain and muscle tears must be considered as possible causes of symptoms. The possibility of cardiac insufficiency or anaemia must also be considered.

When peripheral pulsation is absent at one or more of the usual sites, arterial occlusion can be confidently diagnosed and inference may be made as to the site of occlusion. When arterial occlusion is present, this may be assumed to be due to atherosclerosis except in the young adult where Buerger's disease may ocassionally be the cause.

As there is no good evidence that atherosclerosis itself can be prevented or made to regress by dietary measures or drugs, treatment must be

directed to dealing with the effects of the arterial occlusion.

In general, treatment may be considered as

A. Medical - the prescription of general advice or drugs to alleviate symptoms or

B. Surgical - direct measures to deal with the arterial occlusion itself or indirect measures to improve the patient's walking ability.

A. MEDICAL TREATMENT.

1. General Measures.

The patient should be reassured that the condition is not necessarily serious but that he must learn to accommodate to the disability imposed. He should be advised to walk more slowly as he will then be able to walk further before halting, and should attempt to lead as normal a life as possible because neither rest nor excessive exercise will prove beneficial.

He should be advised to stop smoking as this has an undesirable vaso-constrictor effect. At the same time, he should be warned that cessation of smoking is liable to cause him to put on weight and that an increase in weight is highly undesirable. If he is

already overweight, as many are, weight reduction should be most strongly urged.

He should be advised about care of the feet as there is always a possibility of deterioration to gangrene. They should be kept clean and dry by washing and powdering and he should wear warm clothing, warm socks and loosely fitting shoes (preferably fleecy-lined in winter). Trauma of any kind must be avoided and he should not walk or stand in a crowd or cut his toe-nails too closely. Avoidance of excessive cold or heat is also desirable.

Diabetes Mellitus, if present, must be vigorously treated as inadequate treatment may lead to deterioration.

2. Drugs.

The drugs which produce dilatation of the peripheral blood vessels produce their effects either by an action on the autonomic nervous system or by a direct action on the smooth muscle of the walls of the blood vessels.

The vaso-dilator drugs acting on the autonomic nervous system may act centrally on the ganglia or peripherally at the nerve endings in the arteries. The ganglion blocking drugs which act centrally (e.g.

hexamethonium and pentolinium) and interrupt the vaso-constrictor impulses in the ganglia produce a marked fall in systemic blood pressure and have too many side-effects to permit their use as therapeutic agents. The adrenergic blocking agents, (e.g. Tolazoline) act peripherally and interfere with the release of adrenaline and noradrenaline at the sympathetic nerve endings in the arteries. These have their greatest effect on vessels with a high degree of adrenergic vaso-constrictor tone i.e. the cutaneous vessels, and produce relatively little vaso-dilatation of the muscle vessels.

The second group of drugs produces vaso-dilatation by a direct action on the smooth muscle of the arteries. Two well known members of the group are difficult to administer - both histamine and papaverine must be given intra-arterially and this produces only a transient effect. Papaverine has a limited use in arterial spasm found at operation in some cases of trauma.

Other members of this group of drugs are numerous and are said to have a dilating effect on the arteries in skeletal muscle on the basis of experimental work in animals. These claims have not been established in man, and Shepherd (1950) was unable to show any increase in calf muscle flow either at rest or during exercise after the administration of Padutin.

In patients with arterial occlusion, there is no evidence that sustained improvement in symptoms can be obtained from the oral administration of vaso-dilator drugs. Many of these drugs are capable of producing a general vaso-dilatation but none of those at present available or likely to be introduced in the future can produce a localised vaso-dilatation in the ischaemic limb. Indeed, in many patients, a fall in systemic blood pressure follows the administration of drugs and this leads to reduction in blood flow in the place where it is most needed. Gillespie (1959) in plethysmographic studies with tolazoline (Priscol), phenoxybenzamine (Dibenyline), chlorpromazine (Largactil), and promazine considered that they were useless in the treatment of ischaemia and that they were liable to cause a fall in blood flow when given orally or intravenously.

For these reasons, vaso-dilator drugs have no useful therapeutic effect in obliterative arterial disease and may be regarded as purely a placebo to the patient - and in many cases to the practitioner also.

B. SURGICAL TREATMENT.

Because the underlying lesion in intermittent claudication is arterial occlusion involving a segment of the arterial tree, the surgeon is faced with a challenge to his ingenuity and technical ability, as removal or bypass of the occlusion will produce relief of symptoms.

Martorell (1958) remarked that "In the so-called segmental arteriosclerosis, only the occlusion is segmental, not the arteriosclerosis". This is a reminder that the effects of atherosclerosis on the patient as a whole must be evaluated before attention is directed to the technical possibility of dealing with the arterial occlusion itself.

From this point of view, the follow up of 1476 untreated patients with intermittent claudication carried out by Boyd (1960) and his colleagues is of immense value. It had previously been recognised that many patients with claudication became stabilised and never showed further evidence of deterioration, but this survey has established data indicating the degree of probability of certain events.

The survival rate for all patients from the onset of symptoms was found to be 73.5% at 5 years;

38.8% at 10 years; and 22% at 15 years. Of those who die, the majority die from a vascular cause which is usually cardiac or cerebral (Richards 1957).

In a recent follow up of patients who had undergone a selective nerve crush operation (Reid, Watt and Gray - 1963) the same observation was made. Of 24 patients who died, 10 died from coronary artery thrombosis, 2 from cardiac failure, and 1 from cerebro-vascular disease i.e. 54% died from a vascular cause.

Table 12 :-

Survival Time following Selective Nerve Crush Operation related to Age at Operation.

Age Group.	< 55	55-59	60+
Survival Time in years.	4.7	5.4	3.9
Number of Patients.	7	5	11

It was found that the younger patients did not necessarily have the longest survival time. In this small group of 24, the date of death was known in 23. When these were divided into age groups of less than 55 years, 55-59 years and 60+ years, it was found that the youngest group had a shorter survival time than the middle group.

Boyd and Bloor's figures show that within 5 years of the onset of symptoms 26.5% will have died; 20.3% will have a non-fatal coronary artery thrombosis

or cerebro-vascular accident; 20.9% will develop further thrombosis in either lower limb; and 7.2% will have undergone a major amputation. As these various events will frequently occur in the same patient, there is a relatively large number who remain free of trouble for at least five years.

It is therefore necessary to assess the need for surgical treatment in the individual patient in the knowledge he may continue to lead a relatively active life for years but that an unsuccessful surgical operation may cause him to lose his life or his limb. In considering the indications for surgery it is also necessary to consider the results which may be expected from surgical treatment. It is in the assessment of these factors that the indications for operation tend to vary, sometimes quite widely, with different surgeons.

Reconstructive Arterial Surgery.

The classification of lower leg occlusions into aorto-iliac and femoro-popliteal groups becomes important when surgical measures are considered.

Aorto-iliac occlusion affects large vessels which are relatively easy to handle. The rate of blood flow is rapid when patency is re-established and this militates against further thrombosis and gives excellent

long term results. Many of the patients are relatively young with no electrocardiographic abnormalities and are fit to withstand this major procedure. Lastly, the majority of patients with aorto-iliac occlusion have patent distal vessels (page 203) and it is therefore frequently possible to restore patency of the arterial tree.

Femoro-popliteal occlusions, on the other hand, are more common in the 55-64 age group (Fig.121); are frequently extensive and associated with tibial occlusion (Table 11, p.232); and the vessels are smaller in size than the aorta and iliac arteries and are consequently more difficult to handle. Atherosclerotic changes are often severe and when patency is re-established the rate of blood flow may be insufficient to prevent early or late thrombosis. Although operations on the femoral and popliteal arteries have a lower mortality rate, the immediate and long term results are not so satisfactory.

Surgical Treatment of Aorto-Iliac Occlusion.

The methods employed in aorto-iliac surgery are either thrombo-endarterectomy or bypass grafting.

1. Thrombo-Endarterectomy.

Thrombo-endarterectomy takes advantage of the plane of cleavage which exists between the atheromatous intima and the attenuated medial coat. A split can easily be opened up in this plane and the obstructing material can be freed over long distances.

Fig.89 is the aortogram of a patient with an incipient lesion of the right common iliac artery (Fig.33) and patent distal vessels (Fig.4). In this case, the artery was incised longitudinally to above and below the occlusion, and the atheromatous material was stripped from below upwards (Fig.122). The crucial stage is the dissection of the lower end in which the atheromatous intima is cut obliquely and at a variable level to avoid leaving any 'step' which could lead to post-operative intimal dissection. This can usually be achieved without the need for suturing intima to media at the lower level of dissection.

Many surgeons prefer thrombo-endarterectomy to arterial grafting and will carry out this procedure in almost all cases. Sometimes, however, it is not possible to complete a thrombo-endarterectomy as the artery may tear at the site of a calcified plaque where a plane of cleavage is absent and one must always be prepared to undertake grafting if this should occur.

I myself prefer to undertake thrombo-endarterectomy where the affected segment is short and the rest of the vessel is relatively normal, and to undertake arterial grafting where atherosclerotic changes are extensive or severe.

Thrombo-endarterectomy has been performed in 5 cases of intermittent claudication due to aorto-iliac occlusion (Fig.123). In the second patient operated on (the lesion illustrated in Fig.8), thrombosis occurred in the post-operative period. Apart from this case, operation was successful in the other four with return of ankle pulses, and all vessels have remained patent to date.

The atherosclerotic lesions in these patients were neither extensive nor severe. All had patent distal arteries and their ages were 60,44,51,51 and 53 years.

2. Arterial Grafting.

In the surgery of occlusive disease, arterectomy, originally developed by Leriche (1937), has been abandoned. Arterectomy and end-to-end anastomosis of a graft has also been discarded except in abdominal aneurysm, and end-to-end anastomoses are only performed when the arterial lumen is large and slight stenosis does not matter i.e. in aortic and

occasionally in iliac anastomoses. Linton and Menendez (1955), using homografts in the treatment of femoral occlusions, found that only 8 out of 20 end-to-end anastomoses were patent at 6 months whereas 13 out of 16 bypass homografts were patent. The poor results of end-to-end anastomosis in the femoral artery are largely due to the inevitable stenosis which results and it is interesting to note that Murphy's original illustrations (1897) show stenosis at the suture line. A second cause of deterioration is the development of further atherosclerotic changes at or adjacent to the suture line (Szilagyi, Whitcomb, and Smith, 1956).

Bypass grafting is usually preferred because a wide anastomosis can be made and collateral vessels are not sacrificed by the operative dissection. Szilagyi and his colleagues (1960) have shown in an excellent experimental study that the optimum ratio of graft to host artery diameter is 1.5/1.

The materials available for bypass grafting are autogenous saphenous vein, arterial homografts, and synthetic prostheses.

Vein grafts are unsuitable for aorto-iliac surgery because the high arterial pressures tend to produce dilatation, and homografts have now been largely discarded as they are difficult to procure and tend to

develop aneurysmal dilatation after several years (Irvine, Kenyon, and Stiles, 1963).

Grafts of various synthetic fibres have proved satisfactory and Teflon and Dacron are at present standard prosthetic materials. Knox (1962), however, has reported the development of aneurysmal dilatation in a femoro-popliteal bypass synthetic graft after 5½ years. This graft was made of dacron weaved by a taffeta process and it is probable that the Edwards-Tapp crimped graft (1955) is more resistant to such changes, although the ideal synthetic graft has not yet been produced.

Results in Aorto-Iliac Surgery.

In the surgery of aorto-iliac occlusion, success can be achieved in a high proportion of cases. Failures undoubtedly occur and can best be classified as:-

a) Death attributable to operation.

b) Immediate Failure - thrombosis occurring before discharge from hospital.

c) Early Failure - thrombosis occurring within 1 year of operation.

d) Late Failure - thrombosis after 1 year.

Other incidents such as coronary thrombosis, cerebro-vascular accidents, or arterial thrombosis elsewhere in the leg are due to the disease itself and

are not normally regarded as technical failures but they may nullify any advantage gained by the operation. In the same way, a completely successful result with relief of claudication cannot always be achieved where the patient has an associated femoro-popliteal occlusion.

The most satisfactory method of presenting results is probably that used by Cockett and Maurice (1963) and illustrated in Figs. 123-5.

The operative mortality rate quoted for aorto-iliac operations varies from the 2-3% estimated by Boyd (1960) to the 15.4% reported by Shepherd and Warren (1960). Szilagyi, Smith and Whitcomb (1960) reported a mortality rate of 7.6%, and Cockett and Maurice (1963) 10%.

Personal experience suggests that it is more instructive to classify the mortality rate according to the type of aorto-iliac operation performed as it has been found to vary with the magnitude of the procedure undertaken.

The overall mortality rate for aorto-iliac operations performed for intermittent claudication in the Unit up to the end of 1963 was 10% (4/40). However, 5 thrombo-endarterectomies and 21 unilateral bypass grafts have been performed without mortality whereas

4 of the 14 bilateral grafts performed have resulted in death. For this reason, it is believed that death is more likely to occur in the more major procedures undertaken and that quoted mortality figures for aorto-iliac surgery may depend to some extent on the type of each operation performed.

The causes of death are instructive. The 2nd and 3rd patients who had bilateral grafts performed died in theatre owing to excessive haemorrhage through the graft. In these patients, all three anastomoses were completed before the clamps were released and, since the staged procedure described by Gillespie and Douglas (1961) was adopted, no further mortality from graft haemorrhage has occurred.

There were no further deaths for two years, but, early in 1963, two successive patients undergoing bifurcation grafts died. One patient, aged 65 years, died from coronary thrombosis on the 5th day, and another patient, aged 59 years, died from uraemia due to tubular necrosis.

Subsequent investigation has shown that abnormalities of P CO2 and plasma bicarbonate develop post-operatively although the changes are never so severe as following cardiac operations. Preliminary

estimation of urinary and blood urea is now routine and Mannitol is infused during and after operation to maintain kidney function.

The post-operative course of these patients is often remarkably smooth and the only common complication is paralytic ileus whose incidence is proportional to the degree of retro-peritoneal dissection required.

Figs. 124 and 125 show the fate of the bypass grafts performed. Szilagyi, Smith and Whitcomb, (1960) have shown that the patency rate of aorto-iliac bypass grafts does not deteriorate rapidly beyond the first year and that late failures are uncommon. In their cases, the patency rate fell slowly to 60% at 5 years. For this reason, patency rates at one year can usefully be employed as a measure of success in aorto-iliac surgery provided that these are expressed as a percentage of those patients submitted to operation. Patency rates at one year based on the number of grafts patent on dismissal from hospital are fallacious as deaths and immediate failures are thereby excluded.

Shucksmith and Addison (1962) quote a patency rate at one year of 80% (25 cases) and Cockett and Maurice (1963) quote 77% (45 cases) patent at one year.

In the unit series of 26 bypass grafts performed up to December, 1962, 21 (80.8%) were patent at one year. Quoted figures for graft patency at any stage, however, can be misleading. Four of the 40 patients operated on have developed femoro-popliteal occlusion subsequently (one three months after operation), and three others were known to have a distal occlusion at the time of operation. In these patients, therefore, the success rate, as measured by the relief of claudication, is inevitably lower than the graft patency rate.

In general, it is probable that a mortality rate of up to 10% can be expected in aorto-iliac surgery (excluding aneurysms), that the more major procedures carry the greatest risk, and that 70% of the patients operated on should have patent grafts at the end of one year, although they will not necessarily be free from symptoms.

Cause of Graft Failure.

One of the early grafts (1959) failed because of staphylococcal infection (Fig.124), and the graft had to be removed and a mid-thigh amputation performed. Infection has occurred in two other cases. One was a bifurcation graft performed in 1957 for gangrene and the

patient died on the 28th day. The other was a successful long femoro-popliteal bypass vein graft performed for claudication which had to be excised subsequently owing to disruption of the anastomosis by sepsis, fortunately without loss of limb.

Infection caused by staphylococcus aureus is a known hazard of this type of surgery where foreign material is embedded in the tissues and Schramel and Creech (1959) reported 6 cases of synthetic grafts which had to be removed because of staphylococcal infection.

On four occasions, bypass grafts have been inserted from the common iliac artery to the common or superficial femoral artery in cases of claudication or gangrene and three of these have thrombosed subsequently. It appears that the high rate of flow obtained with aortic anastomosis is essential to the continued function of the graft.

Other causes of failure are probably technical. For example, in the patient who had a bifurcation graft and died on the 5th day from coronary thrombosis, the left femoral pulse became weak post-operatively. At post-mortem, it was found that a flap of intima had become detached and was partially

occluding the anastomosis of the lower end of the graft to the external iliac artery. Such an occurrence may be a cause of failure in other cases.

Another cause of failure demonstrated by Szilagyi and his colleagues (1956) is the development of atherosclerotic plaques at the anastomotic line with narrowing and subsequent thrombosis.

Surgical Treatment of Femoro-Popliteal Occlusions.

Femoro-popliteal occlusions vary enormously in length and in the degree of associated atherosclerotic changes in the distal arteries. The results of operation vary and depend to a large extent on correct selection of patients and on the type of operation employed.

It is now established that homograft, teflon and dacron bypass grafts have a high failure rate. Shucksmith and Addison (1962) found that only 5 out of 76 long bypass grafts (teflon and homograft) were patent after three years, and Ashton, Slaney, and Rains (1962) found only 2 out of 24 teflon grafts patent at 20 months. Synthetic bypass grafts have a high failure rate and homografts tend to become aneurysmal (Irvine, Kenyon, and Stiles - 1963) so that it is possible that both will be discarded in the surgery of femoro-popliteal occlusion. Dale and Mavor (1959) published the results of an

experimental comparison of the patency rates in dogs of peripheral grafting with autogenous veins, homologous arteries and synthetic tubes. They found that the patency rate was lowest with synthetic tubes and preferred autogenous vein to homologous artery because the latter was liable to late degeneration.

The remaining methods available are thrombo-endarterectomy, autogenous vein grafts, or a combination of both.

Thrombo-endarterectomy may be performed in several ways. A long arteriotomy (Fig.126) may be performed which permits removal of the atheromatous material under direct vision but subsequent suture tends to narrow the vessel. This can be overcome by suturing a strip of saphenous vein to each side of the arteriotomy wound virtually producing a long vein patch (Edwards, 1962). Alternatively, the atheromatous material can be removed through multiple arteriotomies using arterial strippers (Cannon and Barker, 1955), and each incision can be patched with autogenous vein or teflon. Mavor (1963) has described the use of arteriotomy at the site of narrowing produced by plaque formation and the suture of a vein patch without disturbance of the plaque.

Autogenous vein was first used as graft

material as early as 1912 (Lexer, 1912: Pringle, 1913), but went out of favour for a time and only recently has it been re-introduced. Kunlin (1951) was responsible for this re-introduction and Mavor (1961) strongly advocated the use of long bypass autogenous vein grafts in patients with long femoro-popliteal occlusions. Linton and Darling (1962) have reported high patency rates in patients with claudication (91% of 76 vein grafts patent on discharge with 4 late failures) and Taylor (1962) has emphasised the superiority of long vein grafts over homografts and prostheses in patients with gangrene. One difficulty is that a suitable vein may not be available in the patient who requires a long bypass graft, although Mavor (1961) found the saphenous vein suitable in 17 out of 19 cases.

Cockett and Maurice (1963) have recently reported enthusiastically on the use of thrombo-endarterectomy combined with a vein graft. One of the major difficulties in thrombo-endarterectomy of the popliteal artery is to avoid creating a 'step' or intimal flap at the lower end of the dissection. They consider that a short bypass vein graft at the lower end of a long thrombo-endarterectomy obviates this difficulty and may increase the possibilities of success.

Personal experience of femoro-popliteal surgery for intermittent claudication has not been extensive. In 9 operations (7 thrombo-endarterectomy; 2 long bypass vein grafts), there has been no operative mortality, but only three were patent after one year. In the 7 patients undergoing thrombo-endarterectomy, clinical improvement has occurred in some even where patency has not been maintained and this is believed to be due to the incidental removal of intimal thickening at the origin of collateral vessels arising from the patent proximal segment of the artery.

The Use of Anti-Coagulants in Arterial Surgery.

The method of use of anti-coagulants in arterial surgery is variable and it is probable that no one method is superior to others.

Anti-coagulants are frequently used during operation and, in the unit, heparin is routinely injected distally when the artery is clamped. Femoro-popliteal operations have been performed using systemic heparinisation which is allowed to wear off. For a period, Warfarin was given pre-operatively and operation was performed at a level of 10-20% thrombotest but even at this level of activity re-thrombosis was seen to occur at operation in an apparently successful procedure.

Although the advantage of using anti-coagulants during operation may be doubtful, their use is safe and wound healing has been normal.

Singer and Rob (1960) found no evidence to show that long term post-operative anti-coagulant therapy was of value as there was no significant improvement in limb prognosis using phenindione. Few patients have been put on long term anti-coagulants but one very successful femoro-popliteal thrombo-endarterectomy thrombosed 8 months after operation in spite of maintained therapeutic anti-coagulant levels (10-20% thrombotest), and it is doubtful if long term anti-coagulant drugs are of any value.

Indications for Operation.

The indications for operation in patients with intermittent claudication are largely a matter of individual choice which varies according to the surgeon. Undoubtedly, the most successful cases are those in which a short arterial occlusion in relatively healthy vessels is dealt with. The patency rate is high and the long term results appear good. On the other hand, these patients with short occlusions are often able to walk a considerable distance before being stopped by claudication and are not seriously handicapped at work

or leisure. Although the post-operative morbidity is low it is difficult to justify subjecting a patient with a mild handicap to an operation which carries an appreciable hazard, especially when there is no way of knowing whether the disease will progress or not.

The disability imposed by aorto-iliac occlusion and the good results to be expected from operation suggest that suitable lesions should be operated on so far as possible.

In femoro-popliteal occlusions producing claudication the most suitable cases frequently do not have sufficiently severe symptoms to justify operation, and if the surgeon is in doubt the patient should be asked for his opinion when the possibilities and advantages of success have been discussed.

Patients are usually only considered for operation if they are below 60 years of age, have no gross cardiac or pulmonary insufficiency and can be regarded as a first class operative risk. Preferably, they should be seriously handicapped at or in travelling to work i.e. usually a claudication distance of 200 yards or less. These rather strict criteria are, of course, relaxed in patients with threatened or established gangrene.

Methods of Indirect Surgical Treatment.

1. Sympathectomy.

The effects of sympathectomy on the walking ability of patients with intermittent claudication has long been and still remains controversial. Directly opposing views are argued forcibly as for example, Smithwick's claim (1957) to have obtained relief in 51 out of 70 patients and Learmonth's view (1950) that the operation was of no value in claudication.

Shepherd (1950) found no increase in calf muscle blood flow after sympathectomy in patients with arterial occlusion and, although sympathectomy is frequently successful in relieving ischaemic rest pain (Gillespie - 1960), there is no satisfactory evidence that patients with intermittent claudication are substantially improved by operation. For this reason, and because of the successful results possible with direct arterial surgery, sympathectomy is now rarely employed in the treatment of claudication. There is, however, no doubt that sympathectomy increases the blood flow through the collateral vessels and increases the skin temperature of the foot.

2. Phenol Sympathetic Block.

With this procedure, which was first

reported by Haxton in 1949 using a 10% solution of phenol, it is possible to obtain the effects of sympathectomy without the disadvantages entailed by the performance of a major operation. The procedure is simple and safe and can therefore be used quite widely. The results obtained in patients with intermittent claudication are variable, however, and cannot be predicted in any way.

Many of the patients so treated claim to obtain very marked improvement in claudication and a number subsequently claim complete relief. Others state that they obtain no improvement.

No correlation has been found between the temperature rise on reflex vaso-dilation and the improvement in claudication distance after phenol block. Some patients who have a very warm dry limb after phenol block cannot walk much further, whereas others who have very little temperature increase state that their walking ability is greatly improved or normal. Reid's (1960) observation that the pain of Paget's disease is abolished by phenol block suggests that this procedure may interfere with afferent pain fibres in the sympathetic system and this might account for the improvement obtained by some patients.

An analysis of the results obtained by

phenol injection (1:15 solution of phenol in water) in patients with intermittent claudication was reported by Reid, Watt and Gray in 1961 and indicates that substantial improvement may result. Some types of occlusion appear to respond better than others e.g. only 10% of patients with aorto-iliac occlusion attain a reasonable standard of improvement, the sub-group contributing most of the 10% being the unilateral common iliac occlusion in which an extensive collateral circulation can be developed (Fig.86).

In femoro-popliteal occlusions, it seems that some types of occlusion respond well to phenol block and others do not. The best response is in short adductor and low popliteal occlusions where approximately two-thirds of each group respond satisfactorily. A poorer response (30% improved) to phenol block is found in patients with extensive femoro-popliteal and tibial occlusion and in patients with bilateral claudication. One group which might be expected to respond well is the group with incipient femoro-popliteal occlusion but the response is often poor and this may be because an adequate collateral system has not yet developed.

One clinical indication that phenol injection does improve claudication is found in some patients with bilateral claudication. It is usual to find that the

patient complains of one leg more than the other (e.g. R > L). Phenol injection of the right lumbar sympathetic chain may reverse the symptoms and the patient then complains that the left leg is the worse of the two. A second phenol injection on the left side restores the original balance so that the pain is again more severe in the right leg. When this cycle occurs, the end result is a marked total improvement in walking ability.

It is, of course, unwise to assume that phenol injection is the cause of any improvement obtained as claudication distance varies so much with the speed of walking and the gradient. However, it is undoubtedly true that many patients, previously treated unsuccessfully with drugs, are exceedingly grateful for the improvement obtained by this method.

3. Operations on the Calf Muscles.

There are three operations which can be applied to the calf muscles themselves which may improve claudication in some patients. These are popliteal neurectomy, selective nerve crush, and achilles tenotomy.

Popliteal neurectomy (division of the motor nerves to gastrocnemius and soleus) was first conceived by Learmonth who noted that patients did not claudicate following excision of a popliteal aneurysm together with

the nerve supply to the calf muscles. The operation has only infrequently been used, but Marston and Cockett, (1962) reported moderately good results in a small series of cases but found a high percentage of post-operative complications, especially venous thrombosis.

Selective Nerve Crush (where the nerves to gastrocnemius and soleus are crushed) has recently been reported by Reid, Watt and Gray (1963). They reviewed 65 patients after 1-10 years and claim satisfactory results in 75% of unilateral operations with a low incidence of post-operative complications.

Achilles Tenotomy (division of the tendo achilles) was reported in a comparable number of cases (72) by Boyd and Bloor in 1960. Good results were obtained in about 50% of cases.

On the whole, the indications for these three operations are restricted as direct arterial surgery is usually more rewarding but there appears to be a place for their use in patients with unilateral claudication. Both Reid and his colleagues and Boyd and Bloor consider that their use in bilateral claudication is limited.

SUMMARY AND CONCLUSIONS.

In this thesis, the pattern of atherosclerotic arterial occlusion in intermittent claudication has been the principal theme.

It has been possible to show that femoro-popliteal occlusions originate at three principal sites:- in the adductor region (60-70%); in the popliteal artery above the level of the knee joint (12%); and at the popliteal bifurcation (12%). The cause of occlusion at these sites has been discussed in relation to the frequency of arterial branching and to curve formation in the popliteal artery.

A tentative classification of femoro-popliteal occlusions has been formulated.

There is a high incidence of occlusion in the symptomless leg of patients with unilateral claudication and in the lower leg arteries of all patients. The incidence rises steeply with increasing age.

Posterior tibial, anterior tibial, and combined ant/post. tibial occlusions are the common occlusion patterns in the lower leg. Peroneal occlusion is uncommon and is usually secondary to posterior tibial occlusion.

The sites of origin of aorto-iliac occlusions

are the aortic bifurcation, the common iliac artery, and the common iliac bifurcation, occlusion of the common iliac artery being the commonest lesion found. Distal occlusion is absent in 60% of aorto-iliac occlusions and the incidence of associated femoro-popliteal occlusion is highest in external iliac and common femoral lesions.

The symmetrical pattern of many of the arterial lesions is noted.

There is no apparent correlation between the site of occlusion and the claudication distance nor between the rise in temperature during vasodilatation and the degree of improvement obtained by phenol sympathetic block.

The significance of the various pulse patterns is discussed and the methods and results of surgical treatment are briefly reviewed.

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INTERMITTENT CLAUDICATION

IN

ATHEROSCLEROTIC OCCLUSION OF THE ARTERIES

TO THE LOWER LIMB.

JAMES KENNEDY WATT

B.Sc. M.B.Ch.B.(Hons.Glas.) F.R.C.S.(Glas.) F.R.C.S.

THESIS SUBMITTED FOR THE DEGREE OF MASTER OF SURGERY

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VOLUME TWO.

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LIST OF ILLUSTRATIONS.

30. Adductor and popliteal plaques.

31. Aortic bifurcation stenosis.

32. Bilateral incipient adductor lesions.

33. Fibrin deposition in atheroma.

34. Irregularity of arterial outline.

35. 'Beading' of superficial femoral artery.

36. Total popliteal occlusion.

37. Multiple popliteal dilatation.

38. Popliteal aneurysm.

39. Early aneurysmal dilatation of the aorta.

40. Post-stenotic dilatation of the right common iliac artery.

41. Persistence of post-stenotic dilatation.

42. Calcification of arterial wall.

43. Arterial calcification.

44. Calcification of the posterior tibial artery.

45. Plaque formation at the aortic bifurcation.

46. Plaque formation at the common iliac bifurcation.

47. Plaque formation at the popliteal bifurcation.

48. Narrowing at orifices of major branches.

49. Magnification on to film.

50. Plaque formation in the proximal part of the superficial femoral artery.

51. Method of histogram construction.

52. Histogram of 295 femoro-popliteal occlusions.

53. Histogram of occlusions measuring 5 cms. or less.

54. Histogram of occlusions measuring 3 cms. or less.

55. Incipient arterial occlusion.

56. The distribution of 167 atherosclerotic plaques in the femoral and popliteal arteries.

57. Plaque formation at the popliteal and posterior tibial bifurcations.

58. Low popliteal occlusion.

59. Lateral view of the femoral artery.

60. Low popliteal occlusion.

61. The relationship of the adductor tendon to the femoral artery.

62. Traumatic occlusion of the brachial artery.

63. Histogram of the upper and lower level of occlusions.

64. Distribution of limiting and re-entrant collaterals.

65. The subsequent development of a popliteal lesion.

66. Curve formation in the popliteal artery.

67. Plaque formation in the popliteal artery.

68. Low popliteal occlusion.

69. Total popliteal occlusion.

70. The development of collateral vessels.

71. Tortuosity of collateral vessels.

72. Tortuosity of collateral vessels.

73. Atherosclerosis of the profunda femoris artery.

74. Occlusion of the abdominal aorta.

75. Composite diagram of vaso-dilation curves.

76. Collateral vessels in a short popliteal occlusion : Posterior tibial occlusion.

77. The natural development of large collateral channels.

78. The collateral circulation in adductor occlusion.

79. The development of total femoro-popliteal occlusion.

80. The reflex vaso-dilation curve after sympathectomy.

81. Collateral development after sympathectomy.

82. Occlusion of the left common iliac artery.

83. Occlusion of the right common and external iliac arteries : Left common femoral occlusion.

84. Occlusion of the left common iliac artery.

85. Incipient aortic occlusion.

86. Occlusion of the left common iliac artery.

87. Aortic occlusion to the level of the inferior mesenteric artery.

88. Aortic occlusion to the level of the renal arteries.

89. Stenosis of the right common iliac artery.

90. Incipient occlusion of the right common iliac artery.

91. Occlusion of the right external iliac artery : Left common iliac bifurcation stenosis.

92. Occlusion of the left external iliac artery.

93. Occlusion of the left common femoral artery.

94. Occlusion of the internal iliac artery.

95. Distribution of the lower level of occlusion in 91 occlusions extending to the common femoral junction.

96. Incipient occlusion at the adductor opening.

97. Recent thrombosis in the superficial femoral artery.

98. Diagrammatic representation of 295 femoro-popliteal occlusions.

99. Long superficial femoral occlusion.

100. Total popliteal occlusion.

101. Short popliteal occlusion.

102. Classification of femoro-popliteal occlusions.

103. The relationship between the profunda femoris artery and long occlusions.

104. Low popliteal occlusion.

105. Total popliteal occlusion.

106. Occlusions of the posterior tibial artery.

107. Occlusion of the posterior tibial and peroneal arteries.

108. Occlusion of the posterior tibial artery.

109. Occlusion of the posterior tibial artery.

110. Occlusion of the anterior tibial and peroneal arteries.

111. Incipient occlusion of the anterior tibial artery : Occlusion of the peroneal artery.

112. Symmetrical femoro-popliteal occlusions.

113. Symmetrical multiple occlusion in the lower leg.

114. Symmetrical multiple occlusion in the lower leg.

115. Symmetrical short adductor occlusions.

116. Symmetrical superficial femoral occlusions.

117. Bilateral femoro-popliteal occlusion.

118. Occlusion of the anterior and posterior tibial arteries : Peroneal leg.

119. Incipient left superficial femoral occlusion.

120. Occlusion of the right anterior tibial and peroneal arteries : Occlusion of the left anterior tibial artery.

121. Age distribution of patients with intermittent claudication.

122. Thrombo-endarterectomy of the right common iliac artery.

123. Results of thrombo-endarterectomy in five patients with intermittent claudication.

124. The results of unilateral bypass teflon grafts in 21 patients with intermittent claudication.

125. Results in 14 cases of bilateral bypass teflon grafts performed for intermittent claudication.

126. Thrombo-endarterectomy of the superficial femoral artery.

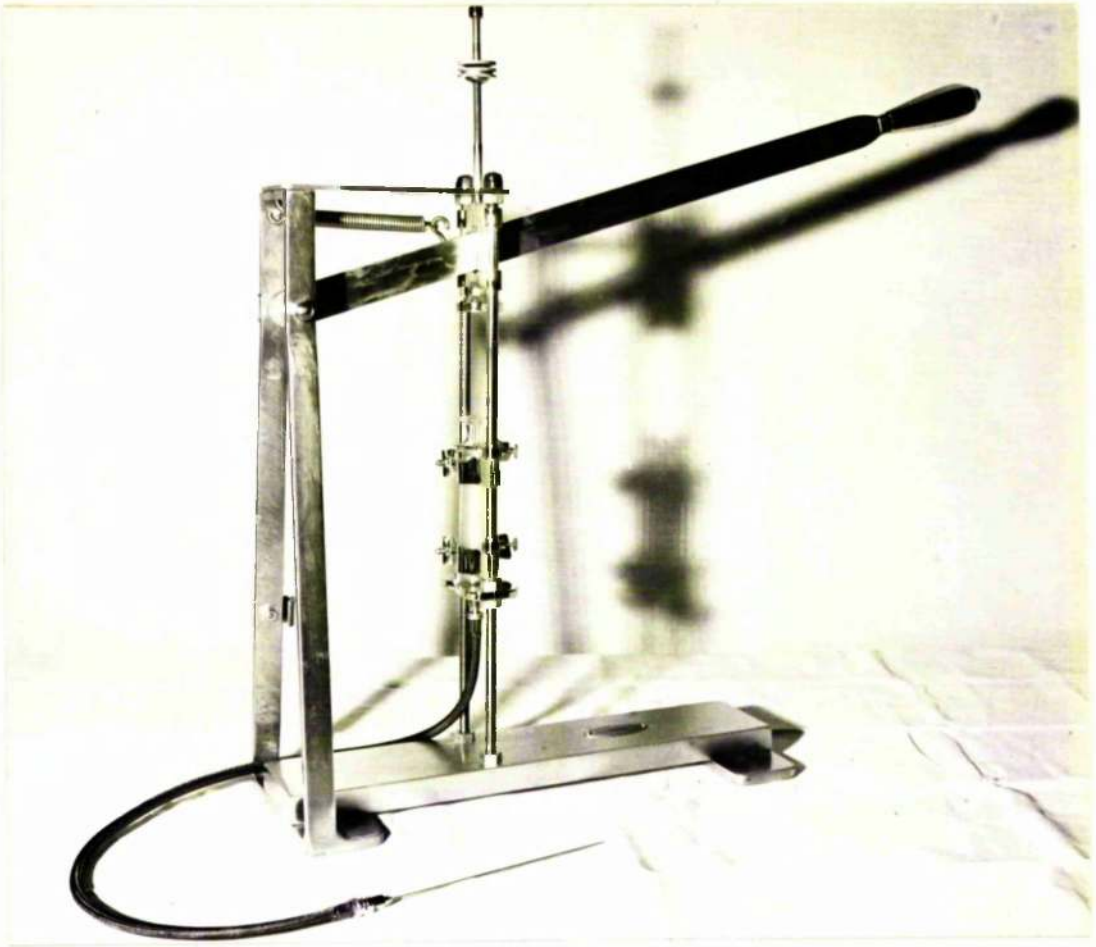


Fig. 1 : Stirling's Injector.

The apparatus is illustrated with needle, pressure tubing, and syringe in position. Pressure on the lever forces down the piston of the syringe and the metal ring at the top of the photograph stops the lever before the piston and the exit ring of the syringe make contact, thus avoiding breakages.

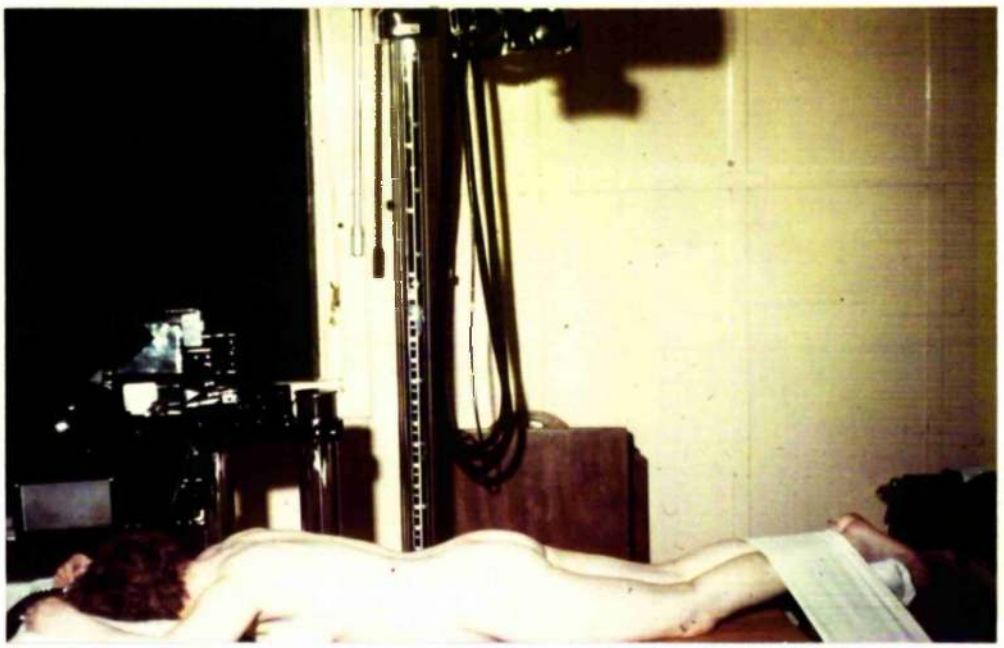


Fig. 2 : Technique of Aortography - 1.

The anaesthetised patient is placed in the prone position with the aortic region overlying the cassette tunnel and the unscreened films for visualisation of the femoral and popliteal arteries are seen in position. The legs are steadied by a light retaining strap.

The tube is centred at the level of the greater trochanters so as to cover the lower trunk and the thighs.

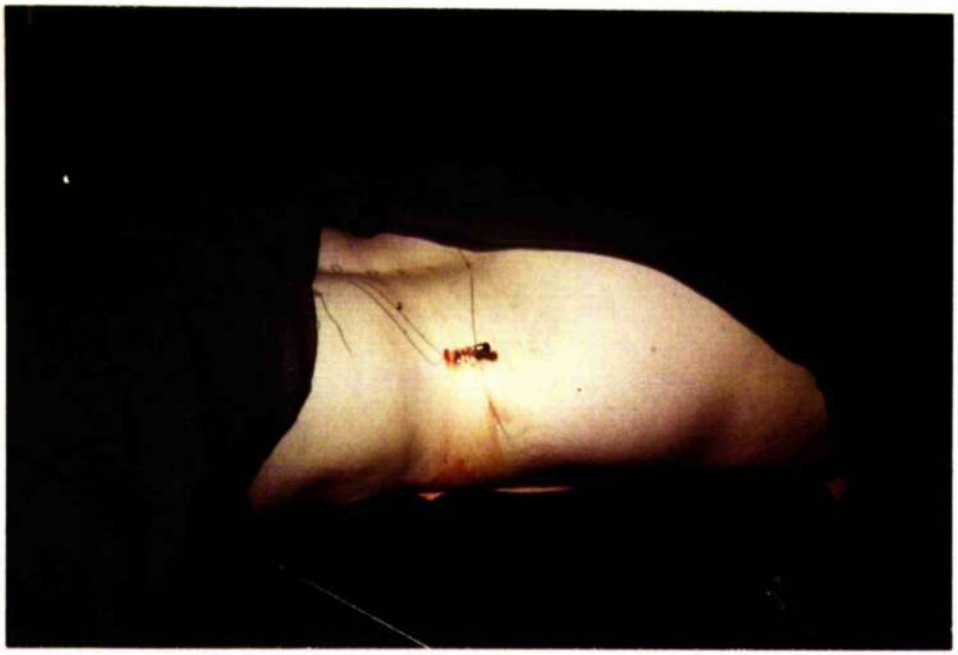


Fig. 3 : Technique of Aortography - 2.

The skin has been prepared with surgical spirit and the region towelled off. The skin markings of the tips of the lumbar spinous processes, the iliac crest, and the left 12th rib are illustrated.

The needle is directed antero-medially from below the 12th rib about 12 cms. from the midline, and a successful puncture has been achieved as indicated by the blood on the skin and the dark stain on the sheet below. The stillette has been replaced until the injector, syringe and tubing are brought over and the tubing is then attached to the needle.

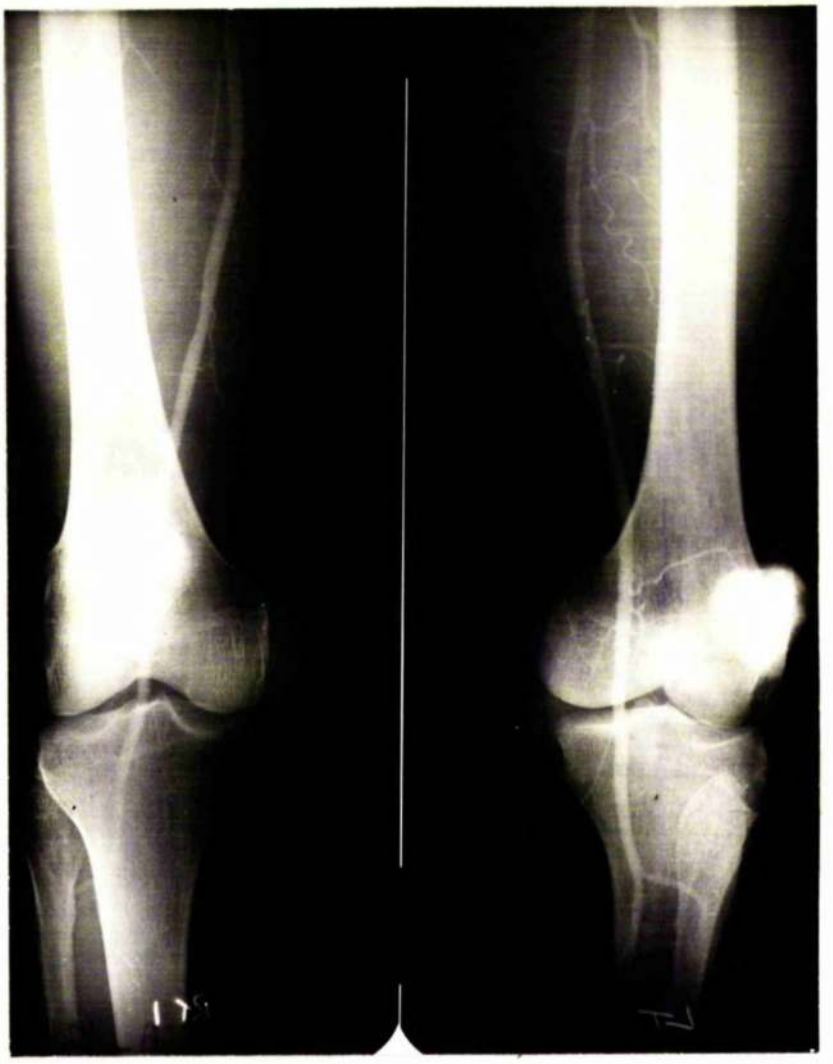


Fig. 4 : Visualisation of Femoral and Popliteal Arteries at Aortography.

The femoral and popliteal arteries are well visualised and show only mild atherosclerotic changes. The flow in the right leg is slower than in the left and the popliteal bifurcation is not visualised. This was due to a stenosis of the common iliac artery which delayed the passage of dye.

Only one film was taken on this occasion and the femoral films were taken at 7 seconds.

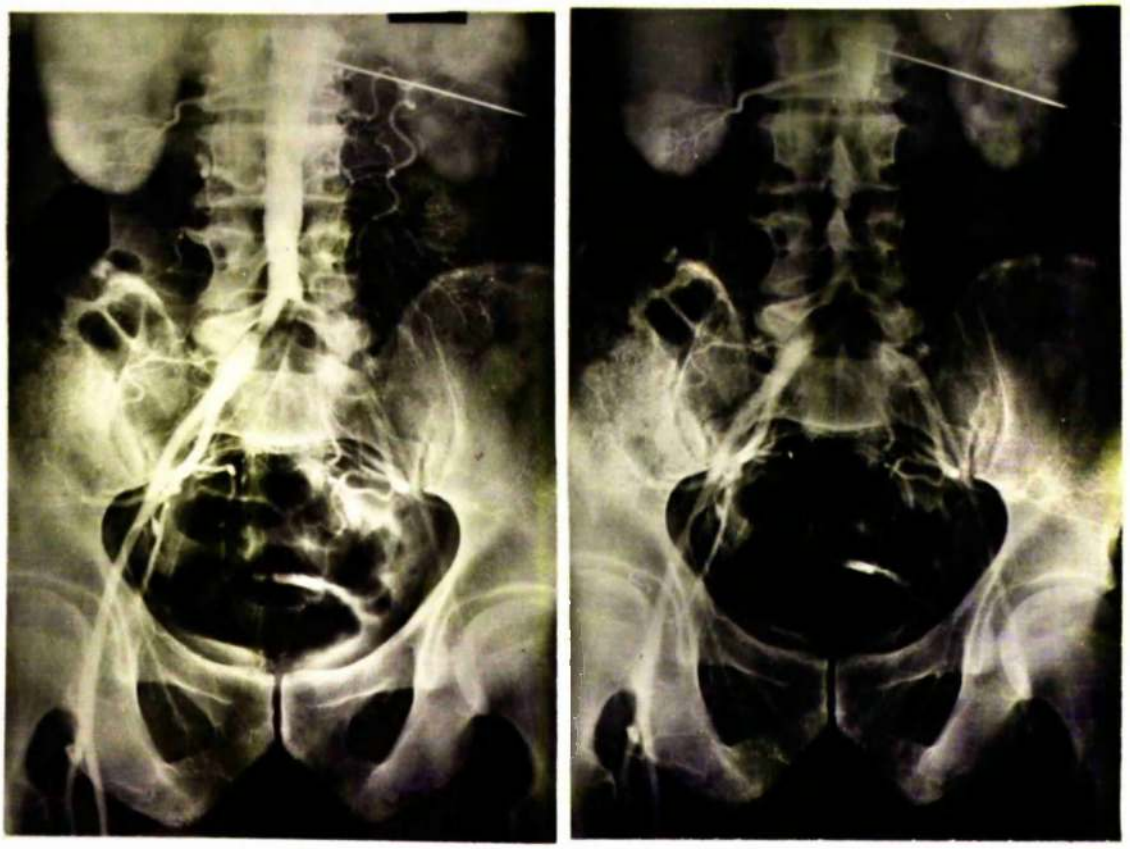


Fig. 5 : Timing in Aortography.

In the 1st aortogram film, there is an incipient occlusion of the right common iliac artery and the left common iliac appears to be occluded. A faint shadow of the left external iliac artery can be seen and the internal iliac artery is well visualised.

In the second aortic film, most of the dye has left the aorta and the reconstitution of the iliac arteries distal to the block indicates that the occlusion involves only the left common iliac artery.

The right iliac arteries are shown best in the first film as the common iliac stenosis does not hold up the dye to the same extent as the occlusion on the left side.

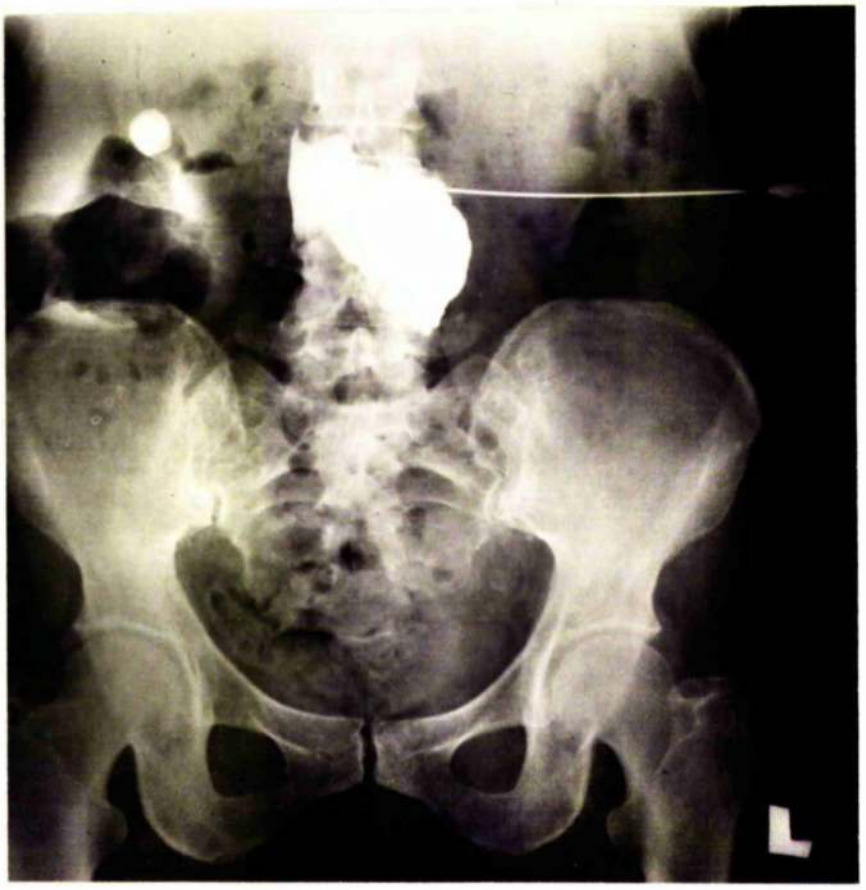


Fig. 6 : Extra-mural Extravasation During Aortography.

In this translumbar aortogram, dye has been injected outside the aortic wall giving rise to a dense shadow. No dye has entered the vessel itself. In spite of this extensive periaortic extravasation no serious damage to the aorta occurred and aortography on a later occasion was successful.

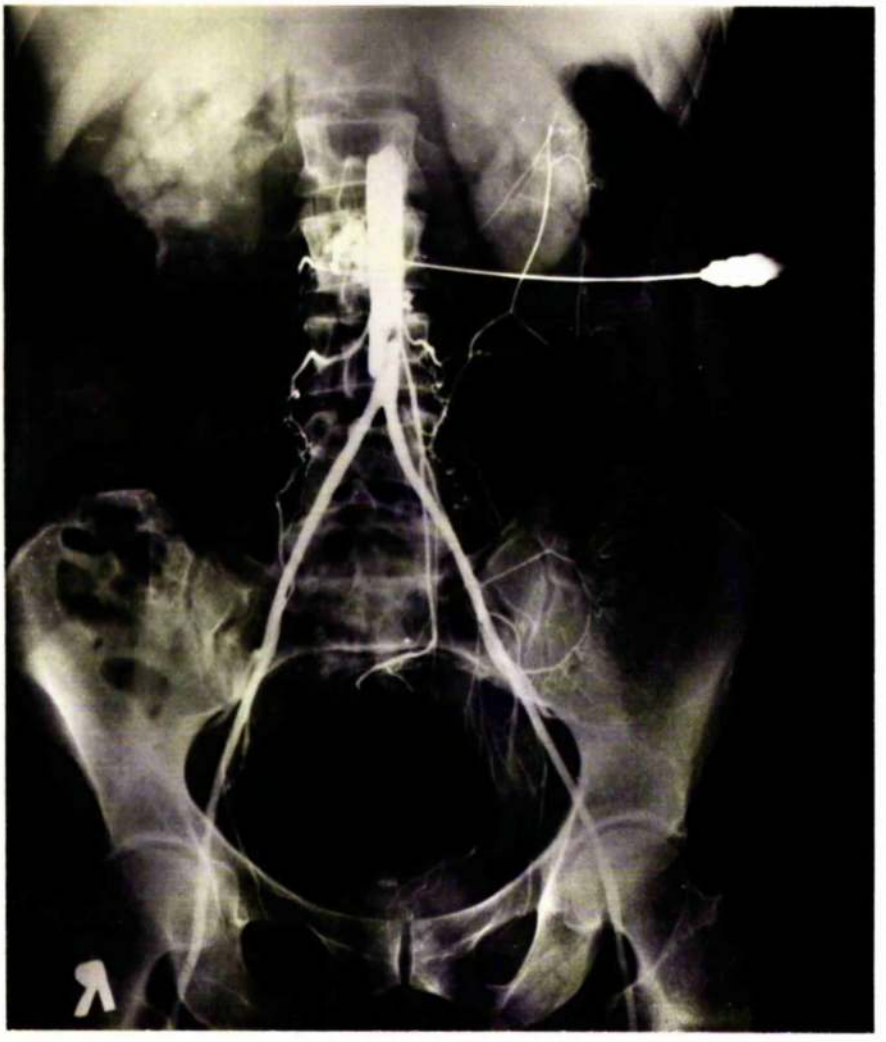


Fig. 7 : Intra-mural Extravasation of Dye.

The aortic shadow is intense and there is a small quantity of dye lying on the right of the aorta at the level of the needle. The dense shadow of the aorta is due to intra-mural extravasation which has occurred after sufficient dye has passed into the aorta to outline the vessel. There is interruption of the dye column below the level of the inferior mesenteric artery.

The pattern of supply of the inferior mesenteric artery to a colon containing faeces is well defined.

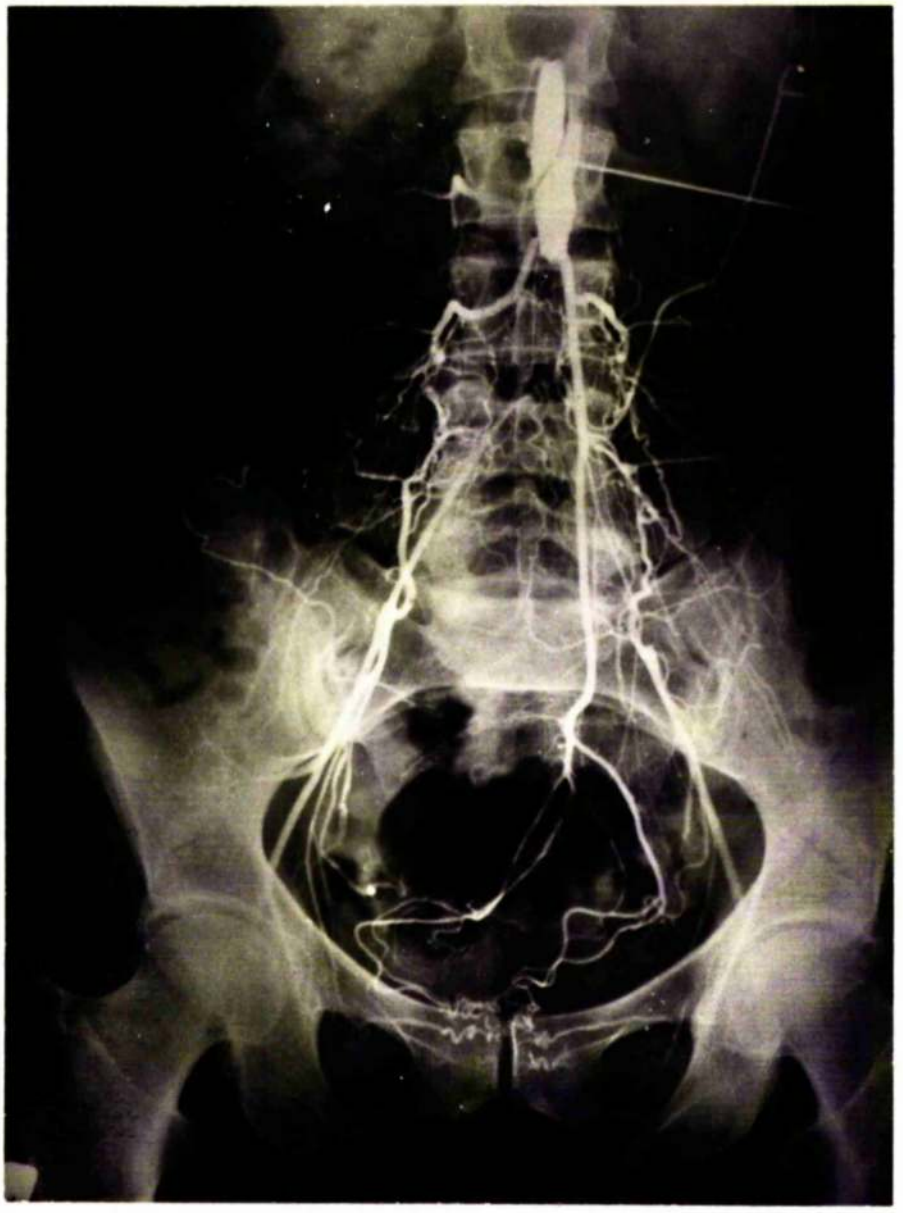


Fig. 8 : Aortic Occlusion.

This aortogram was performed several weeks after that in Fig.7. Aortic occlusion has occurred up to the level of the inferior mesenteric artery and reconstitution of both common iliac arteries occurs principally through a well developed ilio-lumbar anastomosis on each side. The superior rectal artery is seen to terminate in tortuous collateral vessels.

The appearances of a oblique line across the aorta is due to intra-mural extravasation which did not precipitate any further thrombosis. Thrombo-endarterectomy was performed subsequent to this aortogram.

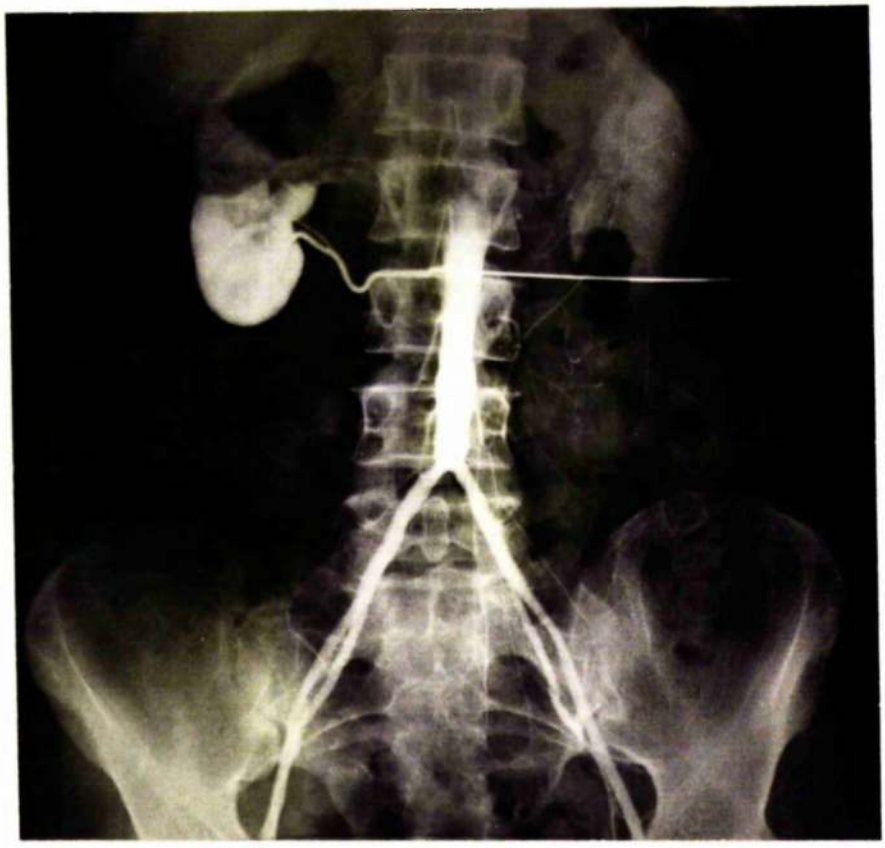


Fig. 9 : Aortic Bifurcation Narrowing.

This aortogram of a woman aged 52 years shows a moderate irregularity of the aorta due to atherosclerosis.

The needle has cannulated an aberrent right renal artery and has produced dense opacification of the lower half of the right kidney. The urine contained a few red cells and casts subsequently but no urinary symptoms developed and renal function was normal.

There is some narrowing at the aortic bifurcation more marked on the right side which produced mild thigh and calf claudication when she was first seen in 1957. In 1959, she developed bilateral short femoral occlusions with an increase in severity of her claudication but her symptoms have remained unchanged for four years.

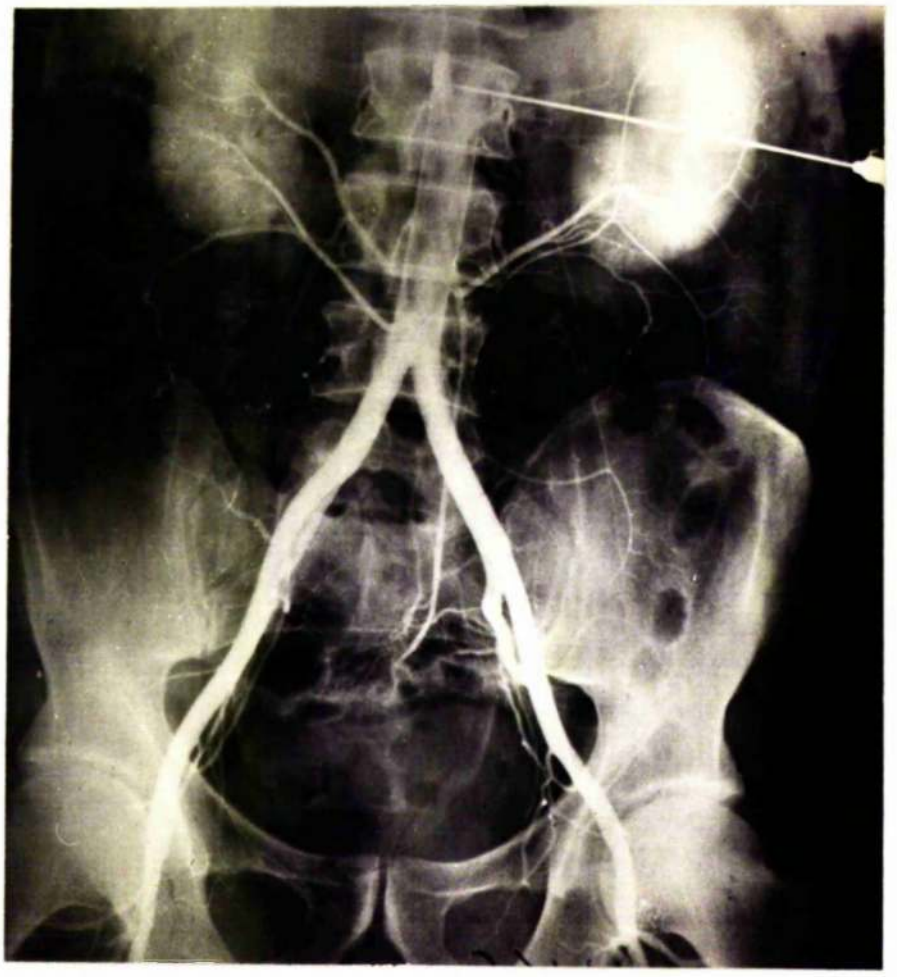


Fig. 10 : Normal Aortogram.

Much of the dye has left the aorta and the kidneys are in the nephrogram phase. A small dense shadow at the tip of the needle indicates a small intra-mural extravasation.

The aorta is broad and regular in outline with three lumbar arteries and the inferior mesenteric artery visualised. The common iliac arteries are of large calibre and smooth in outline and the common iliac bifurcations are visible with the right one occurring at a higher level than the left.

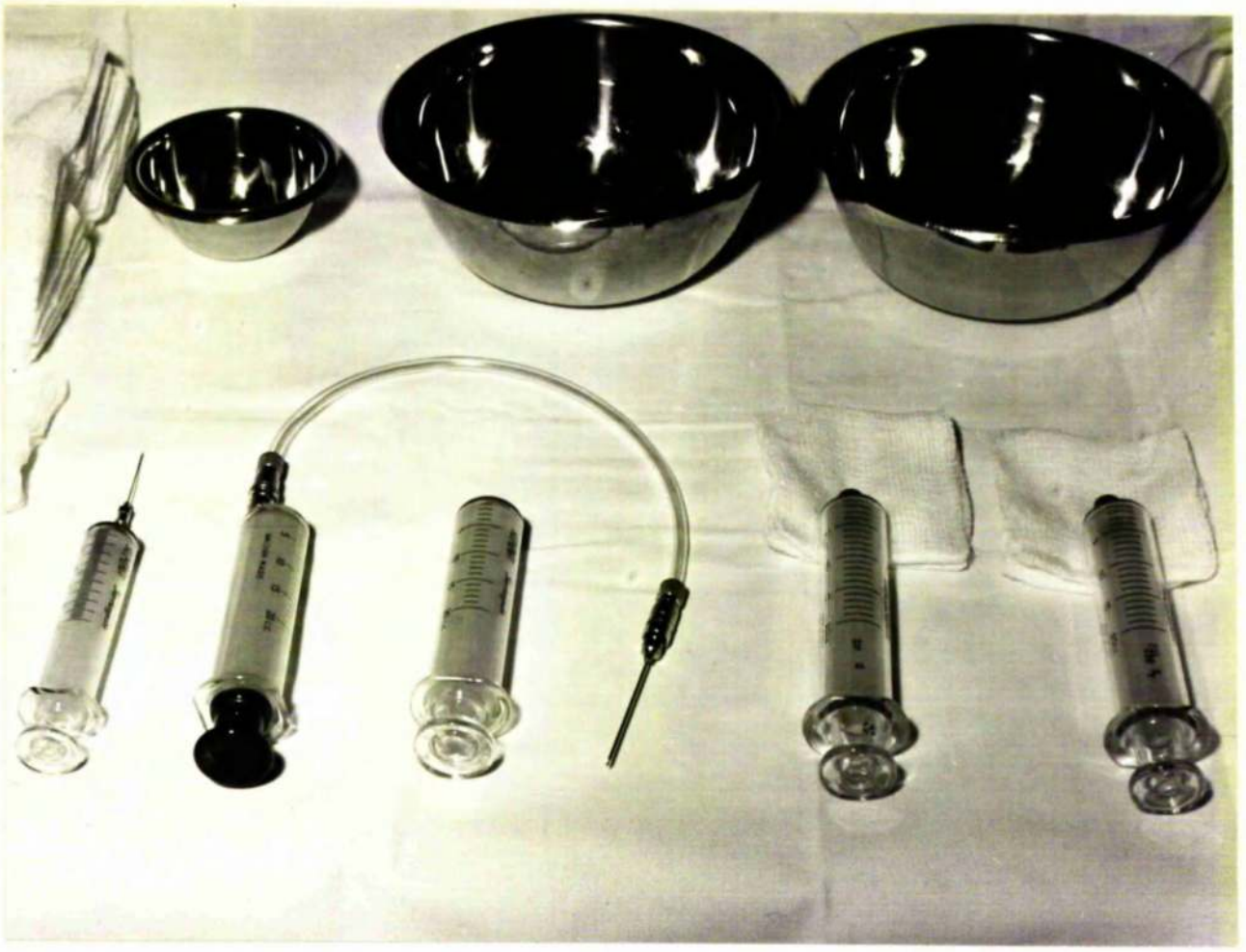


Fig. 11 : Equipment for Femoral Arteriography.

A No.1 serum needle is attached to polythene tubing with Luer-Lok fittings and the syringe is attached to the other end of the tubing by similar fittings.

The 10 ml. syringe and hypodermic needle at the left of the photograph is filled with local anaesthetic and the next two syringes are filled with saline. The two syringes at the right placed on swabs are filled with Urografin.

The two large stainless steel bowls are used for saline and the small bowl for surgical spirit.

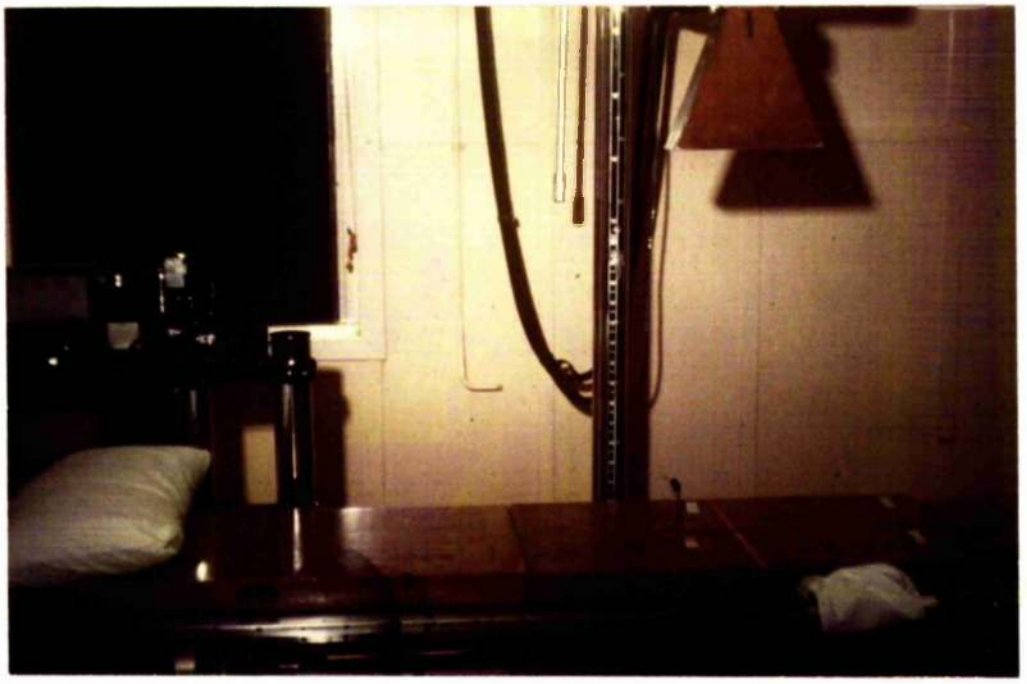


Fig. 12 : Technique of Femoral Arteriography - 1.

The two cassette tunnels are placed side by side at the lower end of the table, and a dummy plywood support at the top end allows the patient to lie flat with one leg on each cassette tunnel. The three trays are seen in position for examining the left leg.

The tube is screened by a plywood and lead 'cone' which restricts the spread of x-rays to the area of a cassette tunnel and is normally centred on the knee.

Fig. 13 : Technique of Femoral Arteriography - 2.

The right groin has been swabbed with spirit and draped with sterile towels. The surface markings of the anterior superior iliac spine, the inguinal ligament, and the pubic spine are indicated. Two parallel vertical lines overlie the femoral artery and a needle has been placed in position to indicate the angle at which the artery is approached.

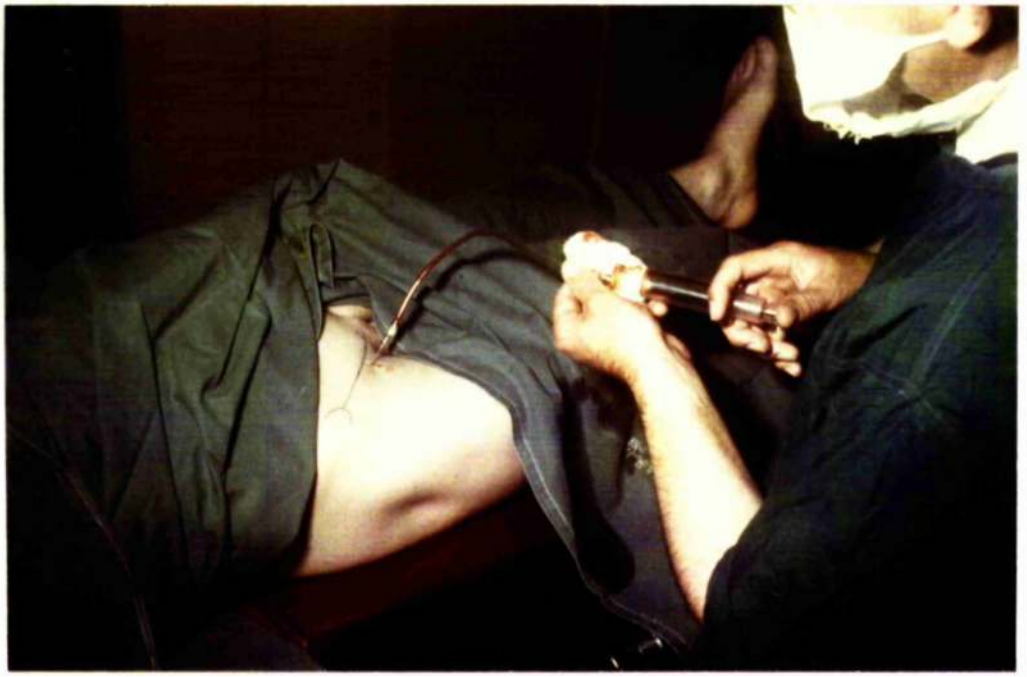


Fig. 14 : Technique of Femoral Arteriography - 3.

The femoral artery has been successfully located and entered. The syringe containing saline has been attached to the plastic tubing and injection of saline is being performed forcibly to confirm that the position of the needle is satisfactory.

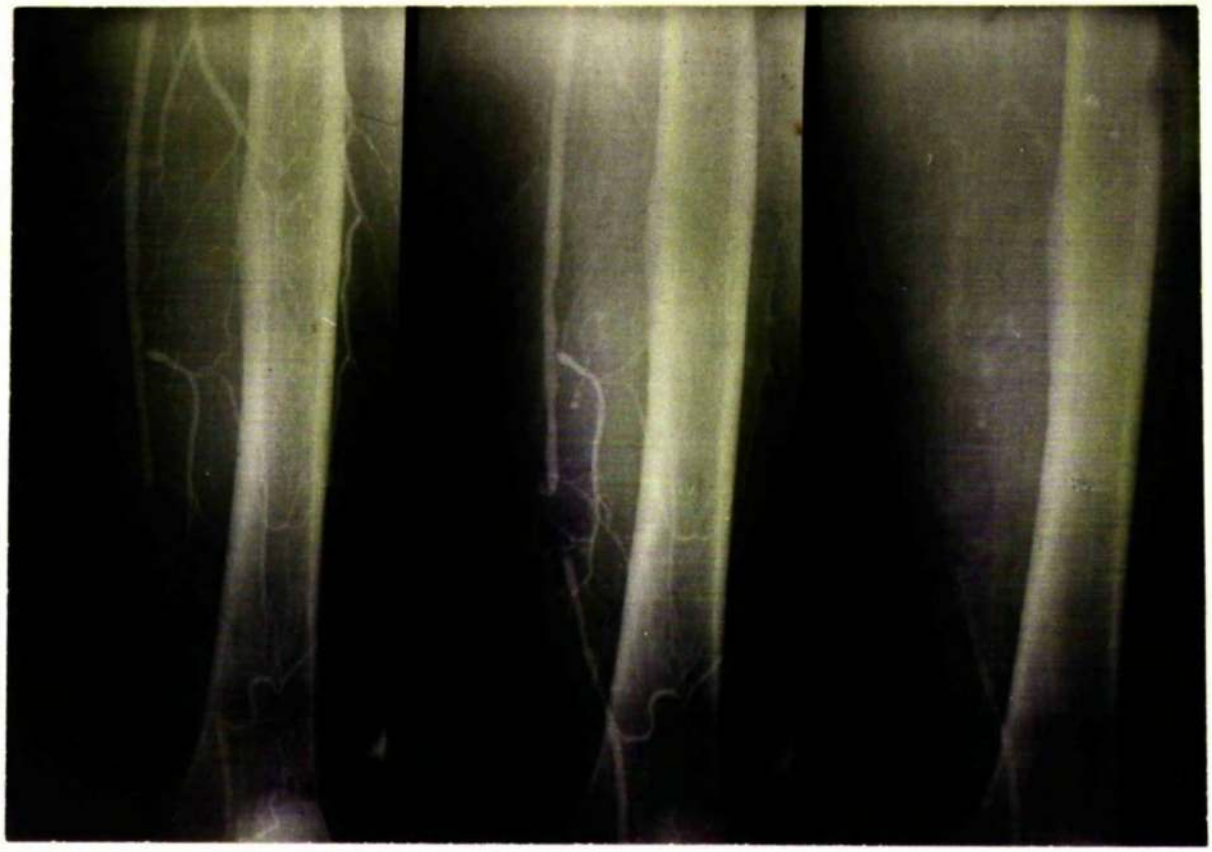


Fig. 15 : Timing in Femoral Arteriography - 1.

The three thigh films of a femoral arteriogram are illustrated in a patient with a short adductor occlusion.

The 1st film shows the superficial femoral artery down to the level of the occlusion with the profunda branches visualised. The patent channel distally is now outlined by dye.

The 2nd film shows details of a short adductor occlusion and the popliteal artery distal to the occlusion is outlined in greater detail. The profunda artery is no longer visible and there is diffuse opacification of the upper part of the thigh.

In the 3rd film, the dye has left the superficial femoral artery and has passed distally. There are faint double shadows in the upper part of the thigh due to venous filling.

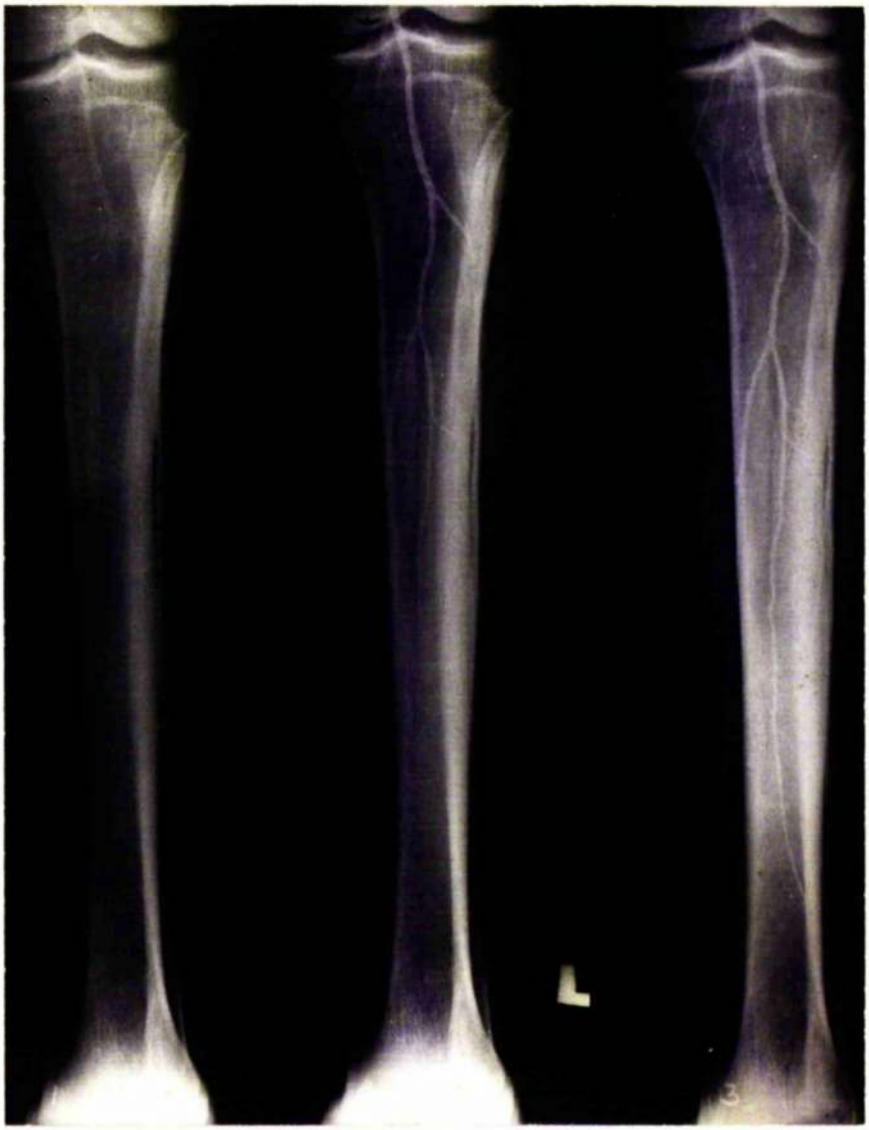


Fig. 16 : Timing in Femoral Arteriography - 2.

These are the lower leg films corresponding to the thigh films in Fig. 15.

In the 1st film, the dye has just reached the popliteal bifurcation and in the 2nd film, the origins of the three lower leg vessels are seen.

In the 3rd film, the posterior tibial and peroneal arteries are visualised down to the lower third of the leg but the anterior tibial artery is superimposed on the cortex of the tibia and cannot be traced downwards.

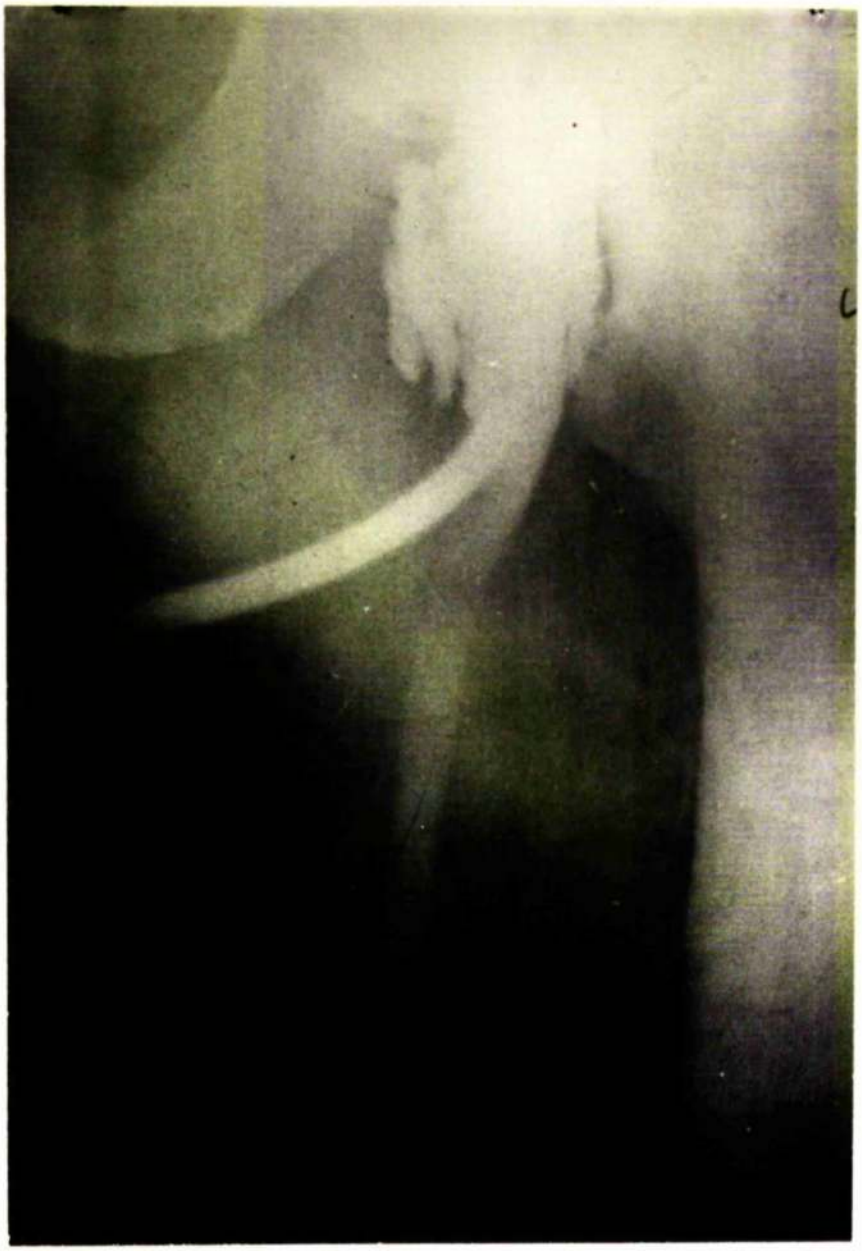


Fig. 17 : Extravasation of Dye.

There is a dense irregular shadow in the groin due to peri-arterial extravasation. A small quantity of dye has entered the artery and gives a faint outline of the vessel.

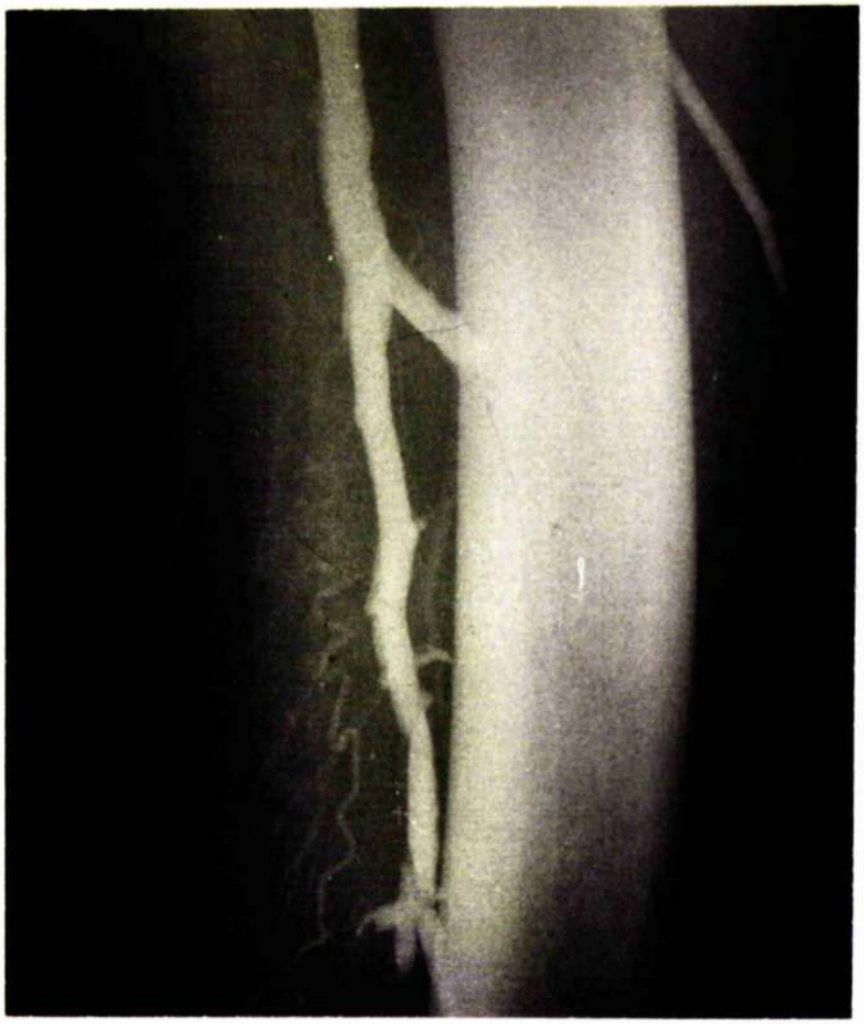


Fig. 18 : Extravasation of Dye.

This is an unusual picture of the profunda artery which has been outlined by dye which has passed down in the adventitial coat to produce a peculiar irregular outline. A small amount of dye appears to have entered the lumen as indicated by the dye in a tortuous collateral vessel medial to the profunda artery.

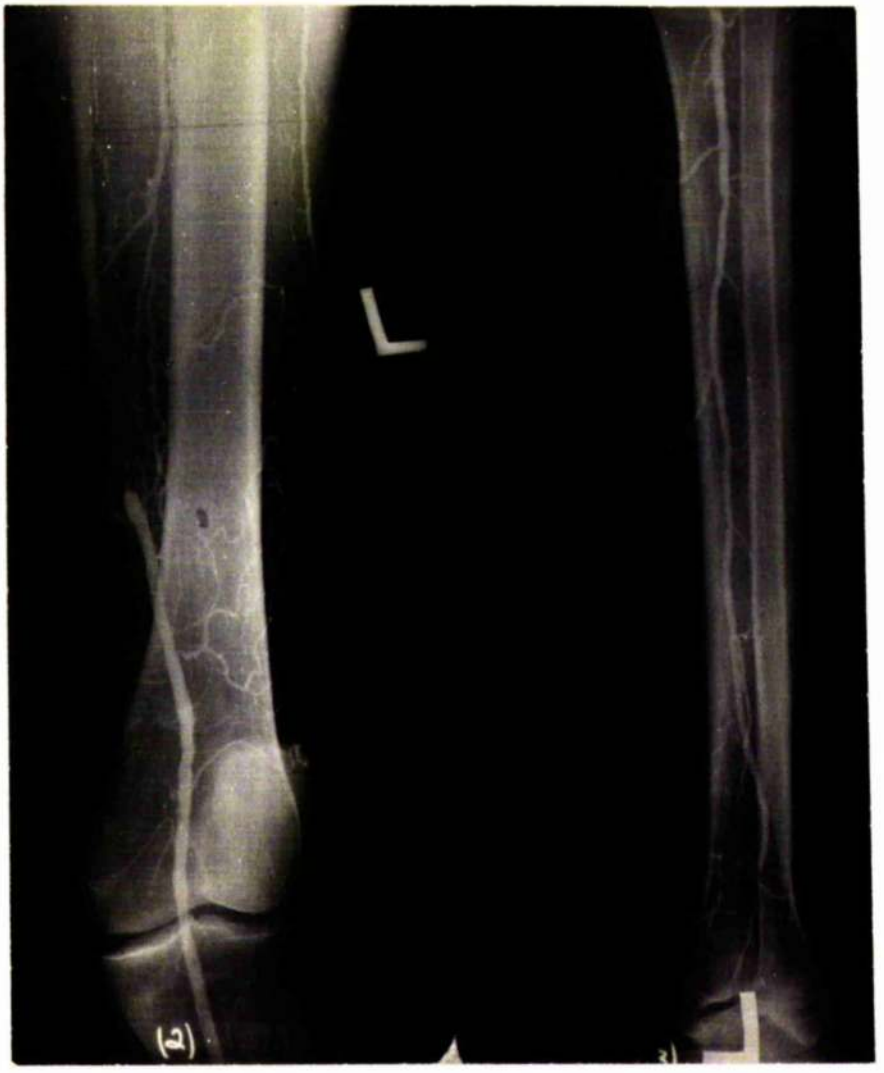


Fig. 19 : Short Adductor Occlusion : Peroneal Leg.

There is a short adductor occlusion with excellent reconstitution of the popliteal artery which shows some irregularity. Numerous small tortuous collateral vessels are present.

In the lower leg film, the peroneal artery is large and breaks up into anastomotic branches above the level of the ankle joint. The anterior and posterior tibial arteries are occluded and are not visualised.

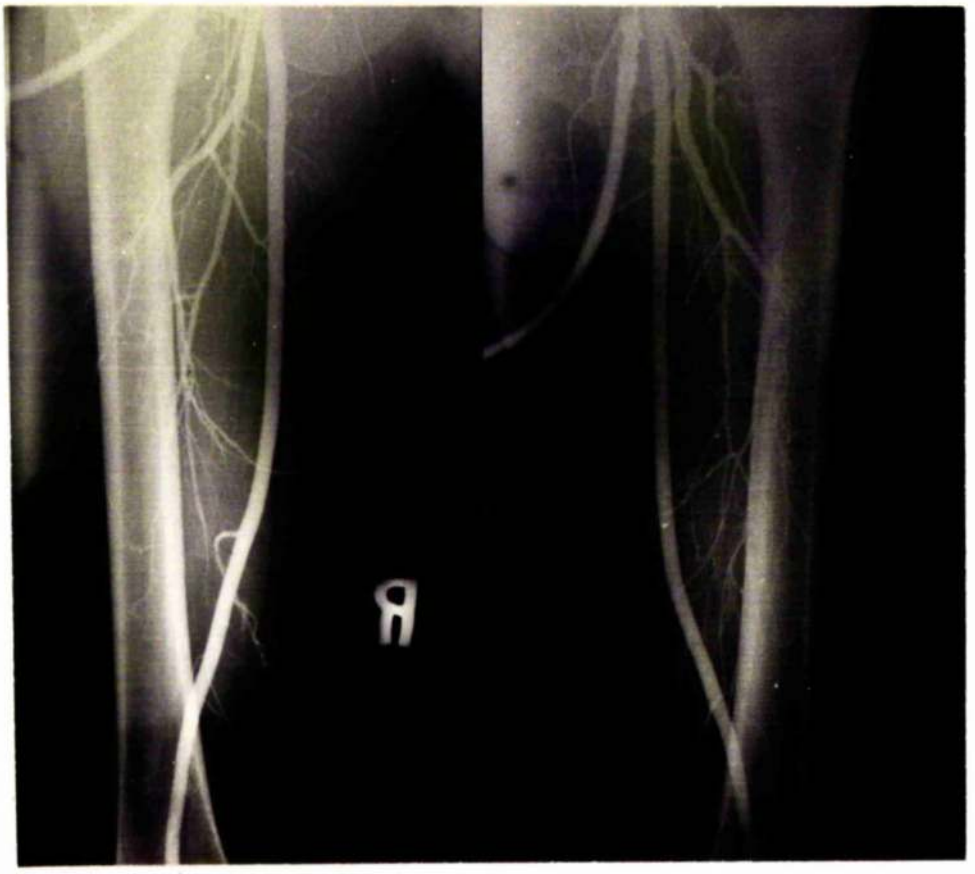


Fig. 20 : Normal Femoral Arteriogram.

The superficial femoral and upper part of the popliteal arteries of both legs outline normally and are of good calibre. The branches are few and small in size.

The profunda femoris is well demonstrated and numerous branches are visible especially on the left side. The lateral circumflex artery is well demonstrated and in the right leg it is larger than the continuation of the profunda itself.

The curved shadow passing to the left of each photograph is the plastic tubing filled with dye.

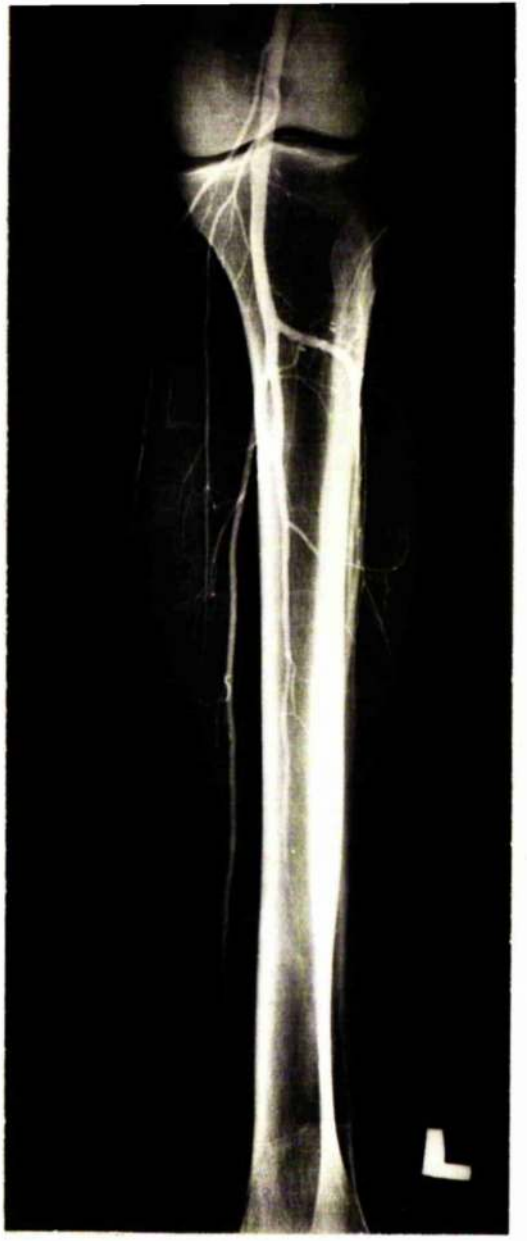


Fig. 21 : Normal Femoral Arteriogram - Lower Leg Film.

The lower part of the popliteal artery and the lower leg vessels are well outlined and are of uniform calibre.

The sural arteries arise from the popliteal artery above the level of the knee joint and pass downwards and medially to the calf muscles.

The anterior tibial artery takes origin a few cms. below the knee joint and its recurrent branch can be seen passing up to the head of the fibula. The posterior tibial and peroneal arteries are of equal calibre.

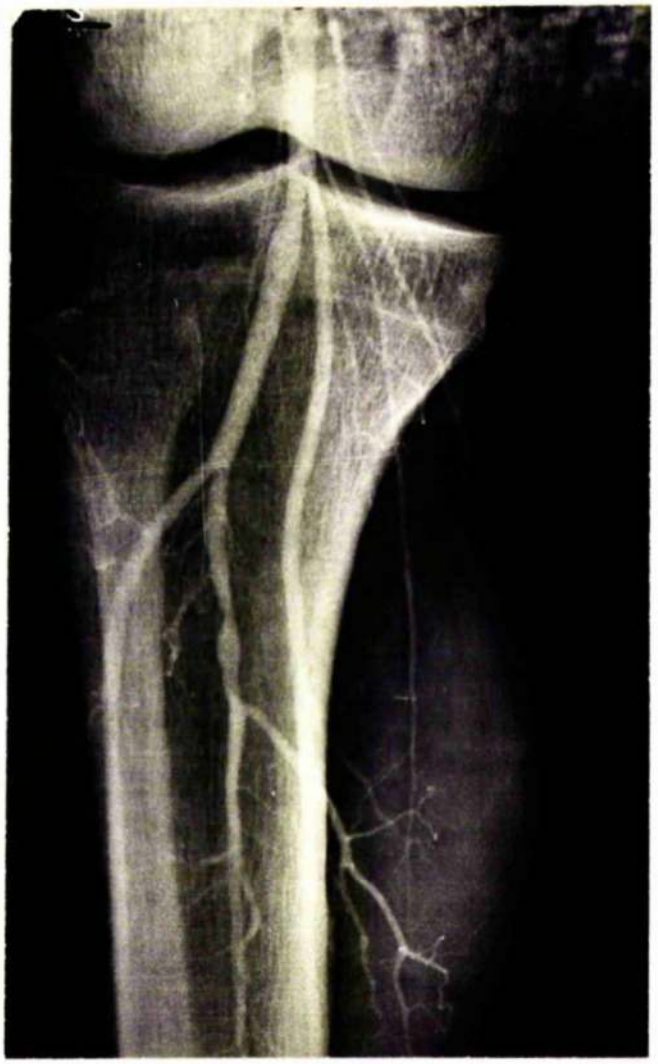


Fig. 22 : High Popliteal Bifurcation.

In this patient the posterior tibial artery arises from the popliteal artery at the level of the knee joint and passes downwards and medially to resume its normal course.

The anterior tibial and peroneal arteries arise a level equivalent to that of the normal popliteal bifurcation.

The popliteal artery is narrowed just below the origin of the posterior tibial artery, and the proximal part of the peroneal artery is irregular in outline.

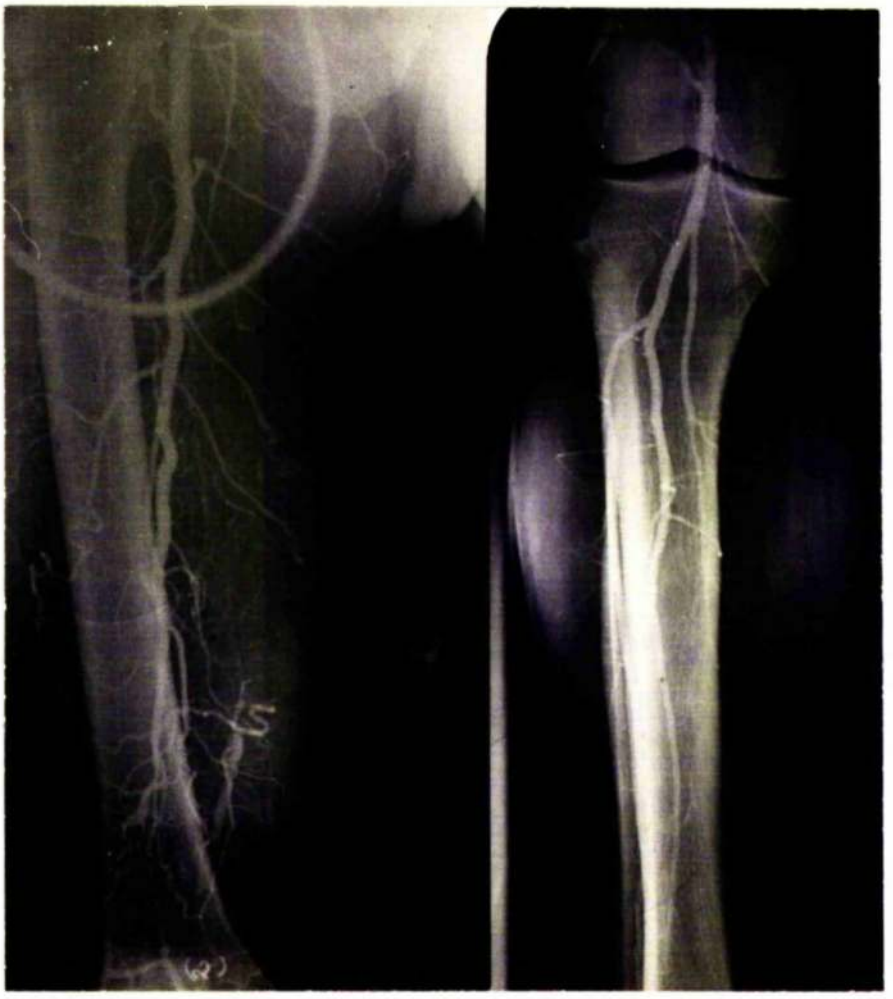


Fig. 23 : Hypertrophy of Profunda Femoris : High Popliteal Bifurcation.

W.J., a man of 53 years was first seen in 1959 when he complained of bilateral claudication at a distance of 1 mile of 3 years duration. Following a Coronary Thrombosis in January, 1959, his walking distance was reduced to ¼ mile.

The right femoral arteriogram shows a long femoro-popliteal occlusion with reconstitution of a short intermediate segment. Collateral channels are numerous and the profunda femoris appears to be much larger than normal.

The lower leg vessels are of reasonable calibre. The posterior tibial artery arises at a high level and the anterior tibial issues from a peroneal artery, which by virtue of its size and direction appears to be the main lower leg artery.

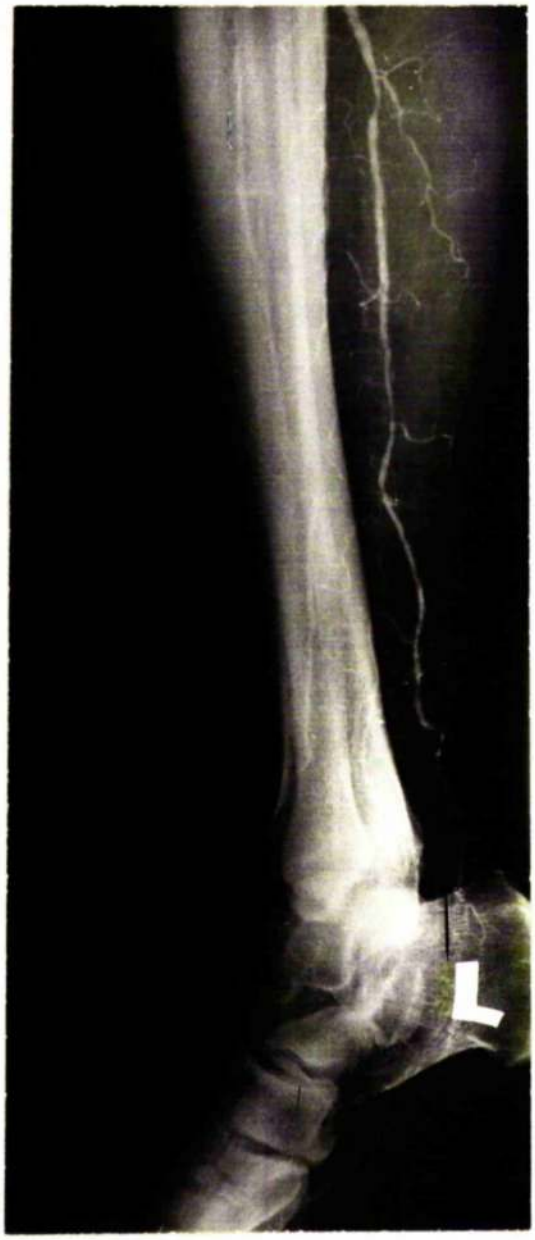


Fig. 24 : Lateral Arteriogram of Lower Leg.

The posterior tibial artery is projected clear of tibial shadows and can be seen to pass behind the medial malleolus into the foot. It is grossly atherosclerotic and irregular.

The peroneal artery is superimposed on the shadows of the tibia and fibula and is difficult to see. The anterior tibial artery is not visualised.

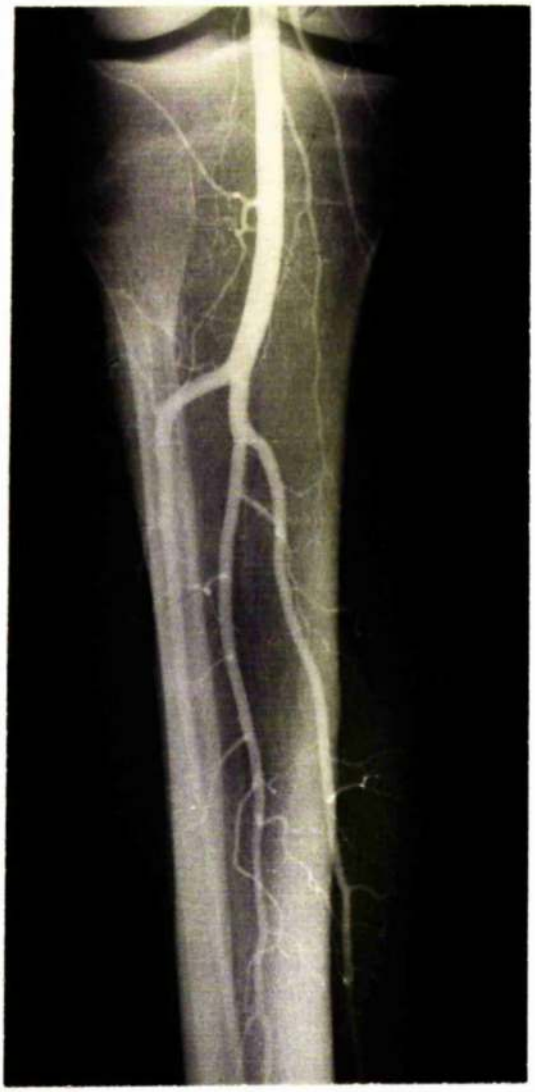


Fig. 25 : Normal Arteriogram.

The popliteal artery is smooth and of good calibre. The anterior tibial artery originates in the manner of a lateral branch and the main stem of the posterior tibial artery is short.

The peroneal artery is the direct continuation of the main trunk and the posterior tibial artery arises in the manner of a lateral branch.

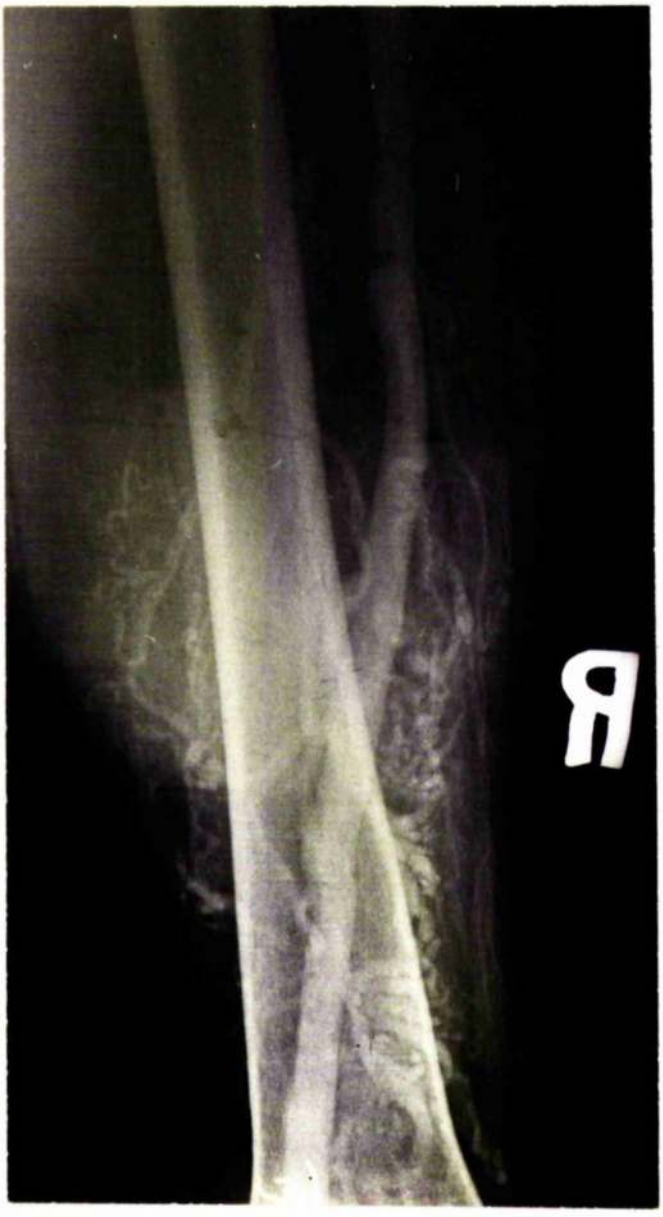


Fig. 26 : Venous Filling during Arteriography - 1.

This is a very striking photograph of dye in the venous system of the thigh found in the third film of a patient with lower leg occlusion. The outline of the femoral vein is well seen and the branches are seen to be double in most cases i.e. venae comitantes.

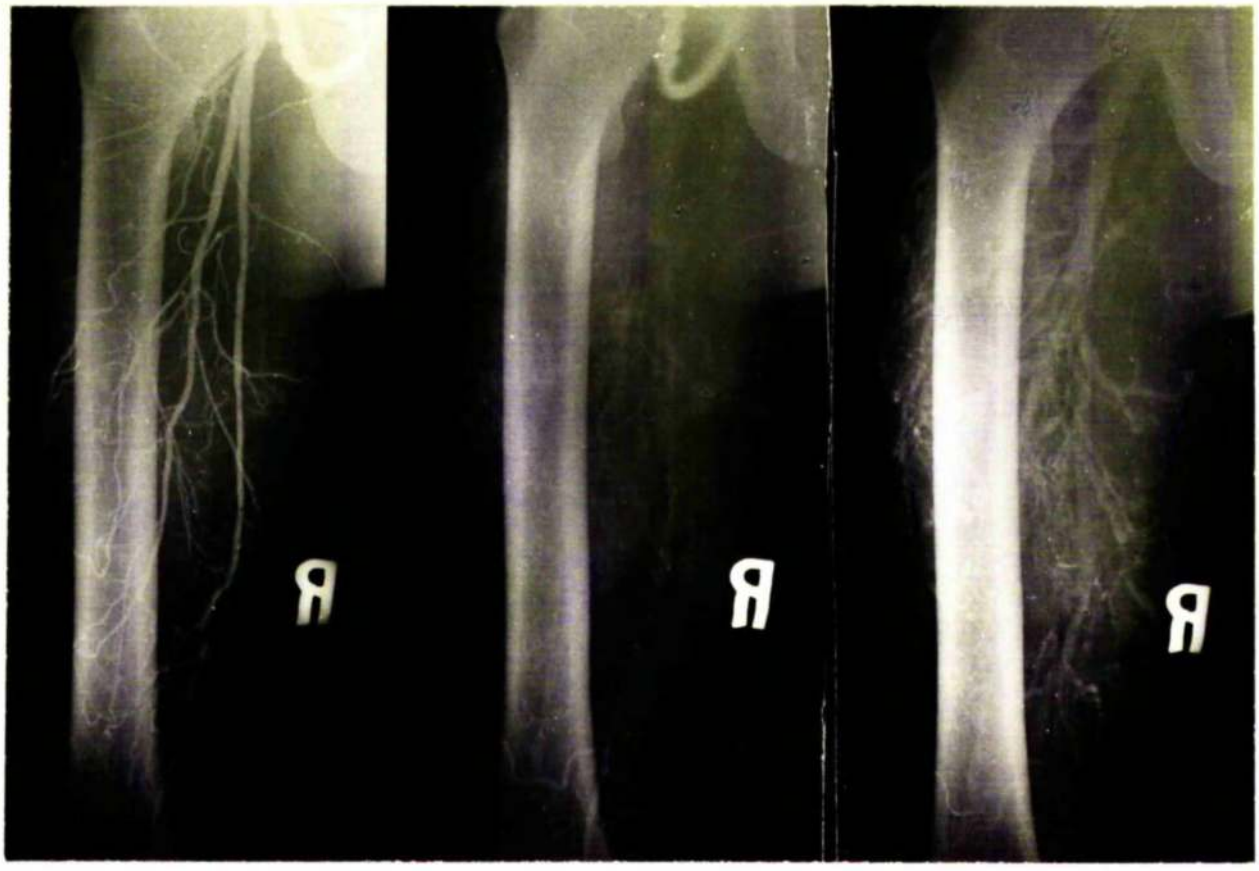


Fig. 27 : Venous Filling during Arteriography - 2.

These are the three films of a patient complaining of claudication. The first film outlines the arteries of the thigh and demonstrates a short adductor occlusion with a large collateral above the block.

In the second film, the dye has almost left the arteries and there is general opacification of the thigh.

The third film shows extensive venous filling. The femoral vein is easily seen at the top of the photograph and the small venous channels are again seen to be double in many places.

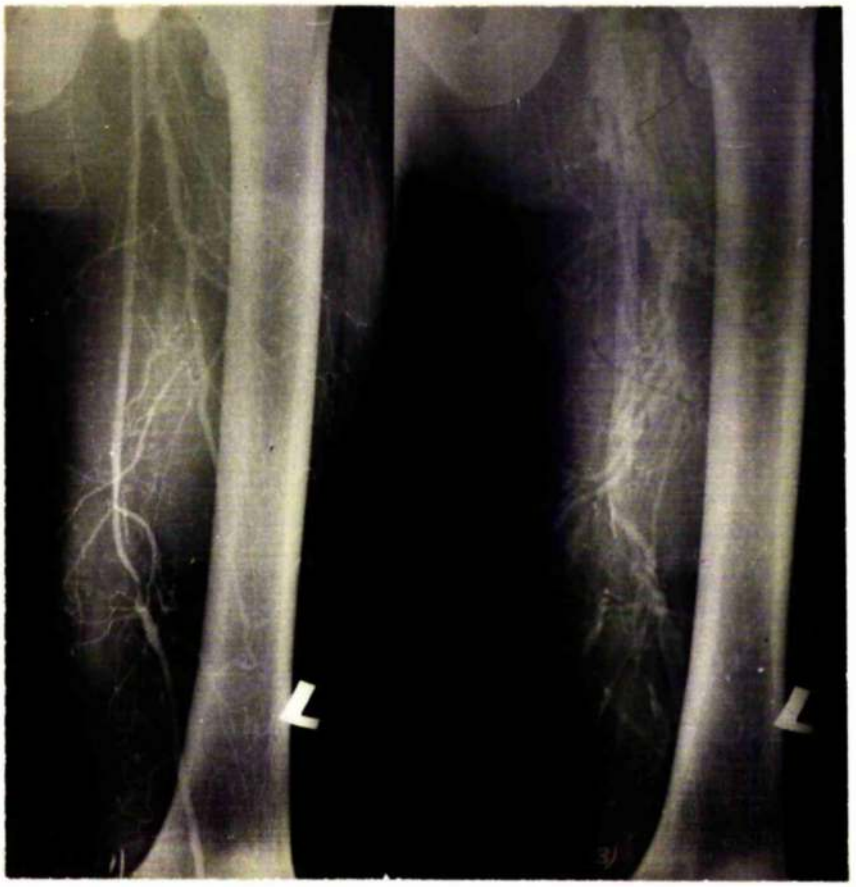


Fig. 28 : Venous Filling during Arteriography - 3.

These are the first and third films of the left femoral arteriogram of the same patient illustrated in Fig.27.

The first film shows a short adductor occlusion at a site corresponding to that of the right thigh, and collateral development is good.

The third film shows widespread venous filling with numerous small double branches. The venous pattern can be seen to follow the arterial pattern very closely.

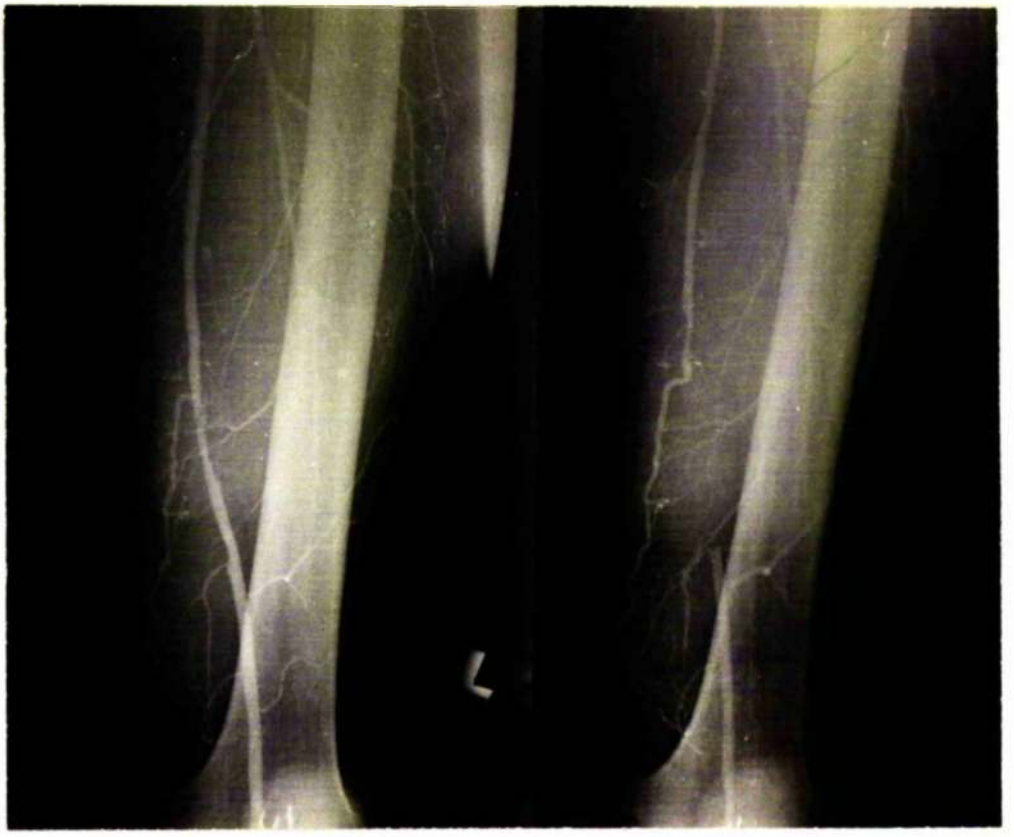


Fig. 29 : The Development of Thrombosis in the Adductor Region.

J.C., a diabetic patient aged 42 years, was first seen in 1959 when he complained of right sided claudication after walking 200 yards. Progressive deterioration occurred and his left femoral artery thrombosed in 1960.

In the first arteriogram (10.2.59), irregularity of the femoral artery due to intimal thickening can be seen with plaque formation occurring below the branch seen in the middle of the vessel.

The second arteriogram (12.1.60) shows that thrombosis has occurred and has extended as far as the branches visualised on the first arteriogram. The proximal part of the artery is more narrowed than before due to diffuse intimal thickening. The development of a collateral network between the limiting and re-entrant collaterals is more pronounced than in the first arteriogram.

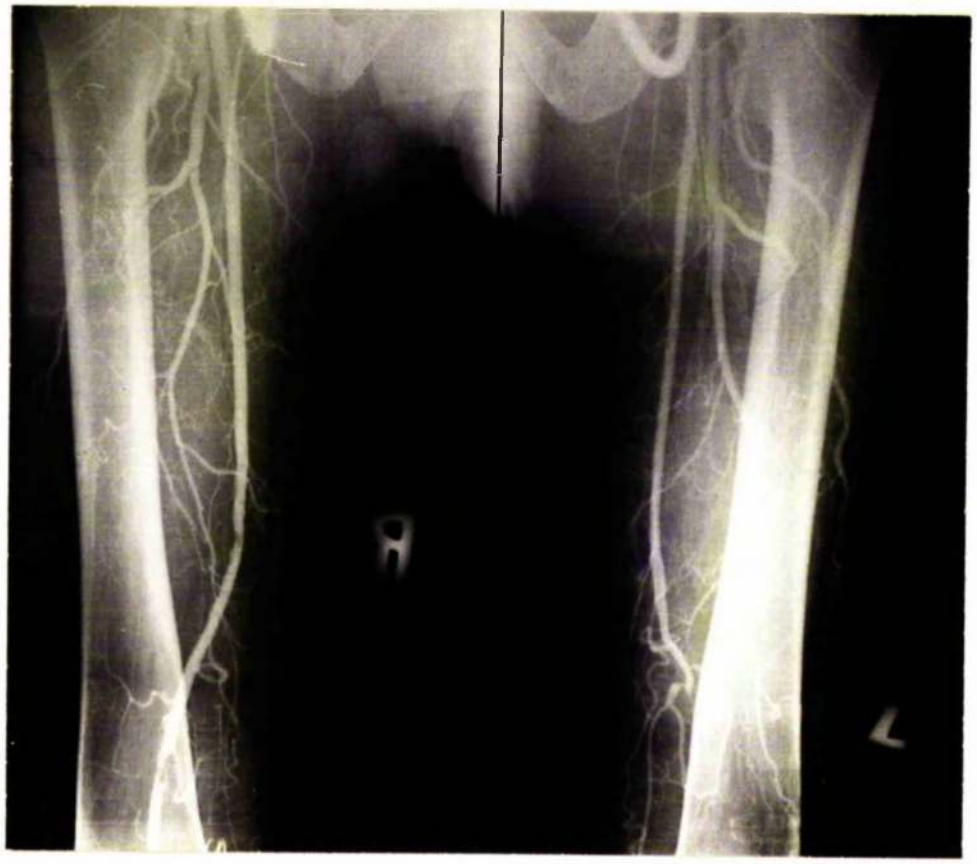


Fig. 30 : Adductor and Popliteal Plaques.

H.W., a man of 53 years, was first seen in 1959 when he complained of left sided claudication at 100 yards distance.

In the arteriogram of the right leg, an adductor plaque is seen which has produced some narrowing of the vessel and a reduction in the density of dye. Another plaque is present in the popliteal artery where stenosis has developed.

The atherosclerotic process has been more severe in the left leg and an occlusion of the popliteal artery has occurred. It is possible that, in the course of time, a similar lesion will develop in the right leg due to the severe popliteal stenosis present.

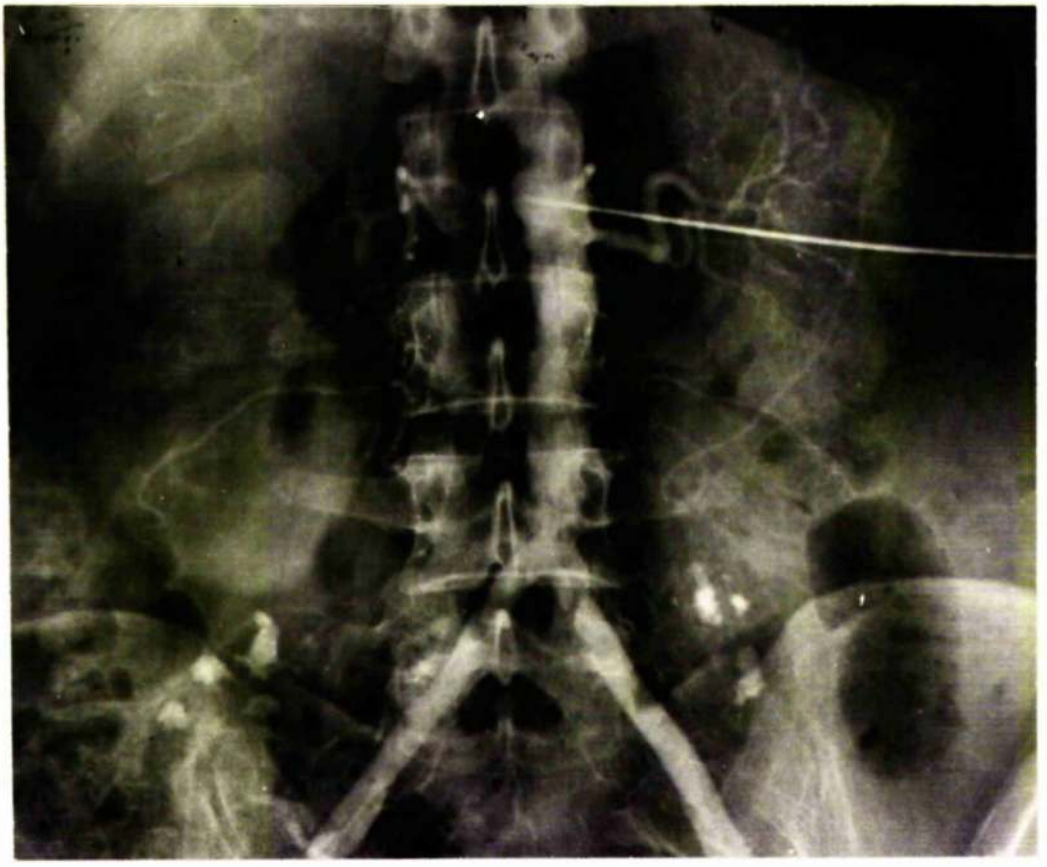


Fig. 31 : Aortic Bifurcation Stenosis.

The lower abdominal aorta is grossly atherosclerotic in this female patient aged 47 years, and there is marked stenosis at the aortic bifurcation which has not constricted the orifices of the common iliac arteries. The inferior mesenteric and lumbar arteries have been obliterated by the disease and are not visualised.

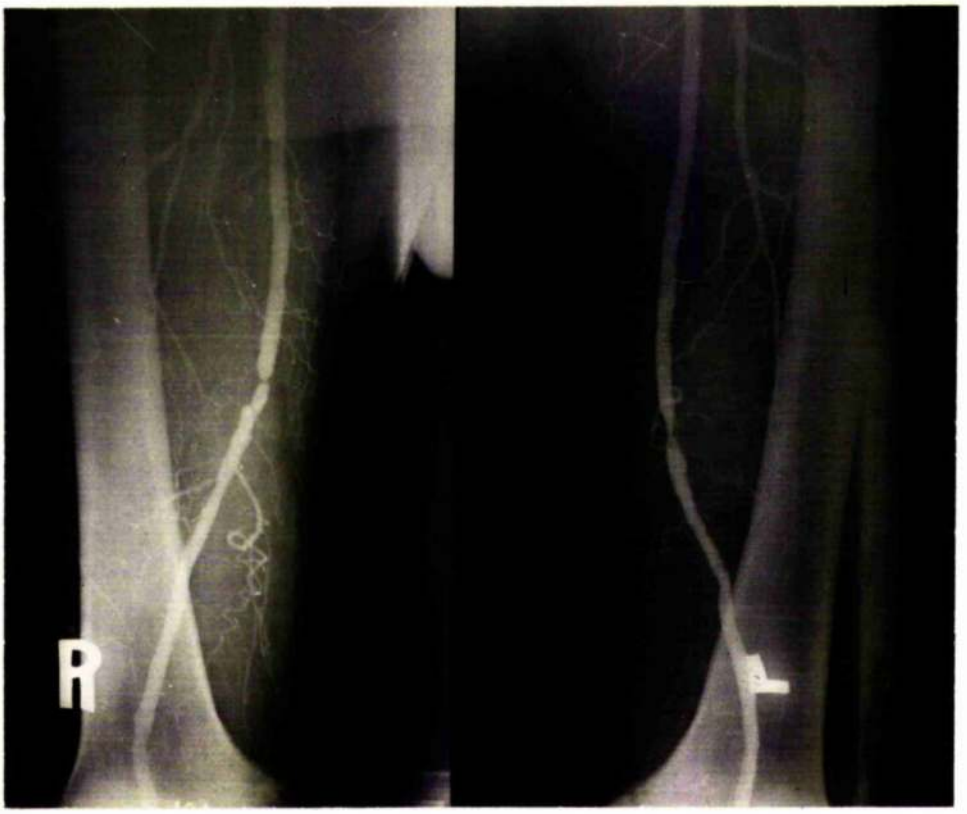


Fig. 32 : Bilateral Incipient Adductor Lesions.

W.W., an elderly man of 74 years, was first seen in 1958 when he complained of bilateral claudication at 100 yards.

Both femoral arteries are severely narrowed in the adductor region due to plaques. Although occlusion appears imminent on either side, both vessels remained patent until his death four years later.

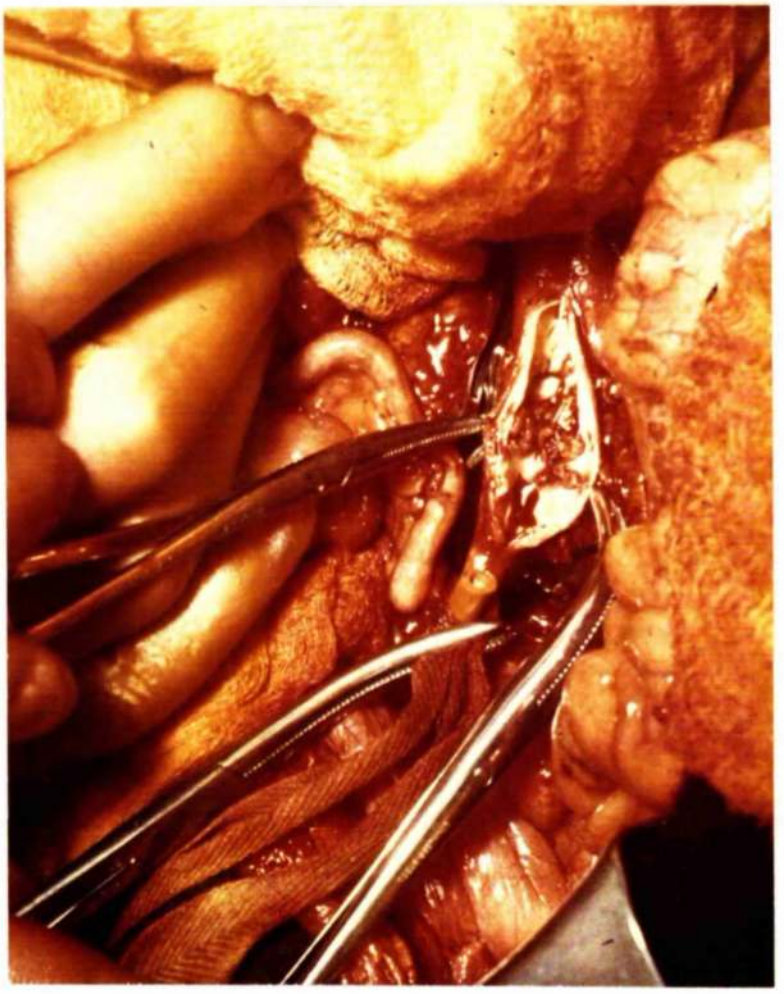


Fig. 33 : Fibrin Deposition in Atheroma.

The right common iliac artery (illustrated in Fig.89) has been incised longitudinally. The marked thickening of the intima can be seen in the wall held by the small artery forceps and the stenosis seen on aortography is due to the deposition of fibrin on an atherosclerotic plaque on the posterior wall of the middle of the artery.

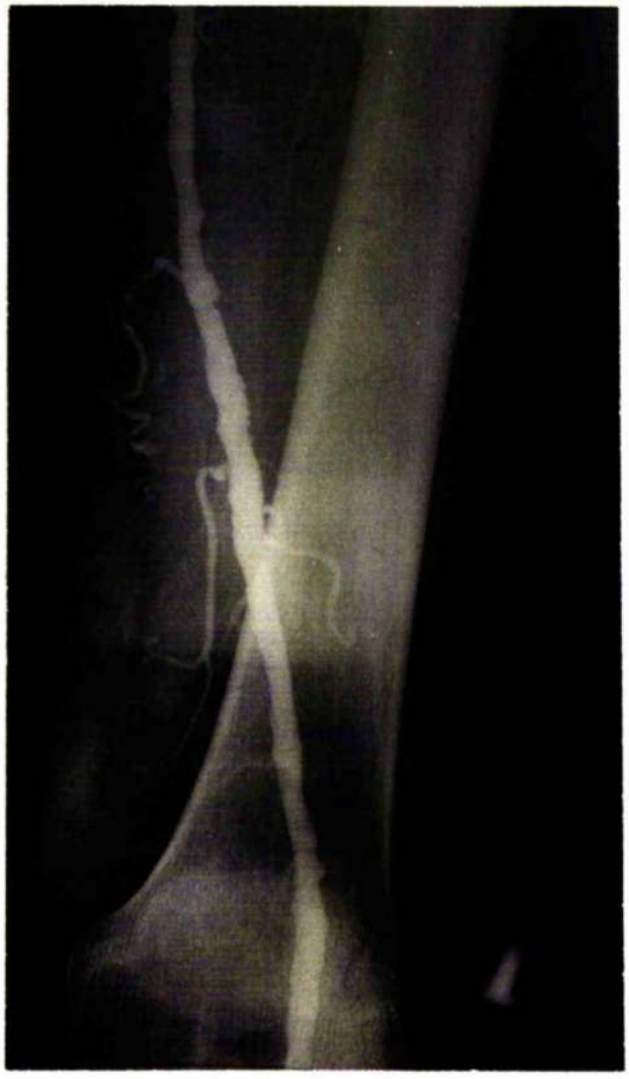


Fig. 34 : Irregularity of Arterial Outline.

A.G., an elderly man of 68 years was investigated in 1958 for right sided claudication. An arteriogram of the left femoral artery performed at the same time showed marked irregularity of outline due to atherosclerosis but no evidence of occlusion.

Thrombosis developed in this vessel two years later leading to a total femoro-popliteal occlusion and gangrene which necessitated amputation of the left leg.

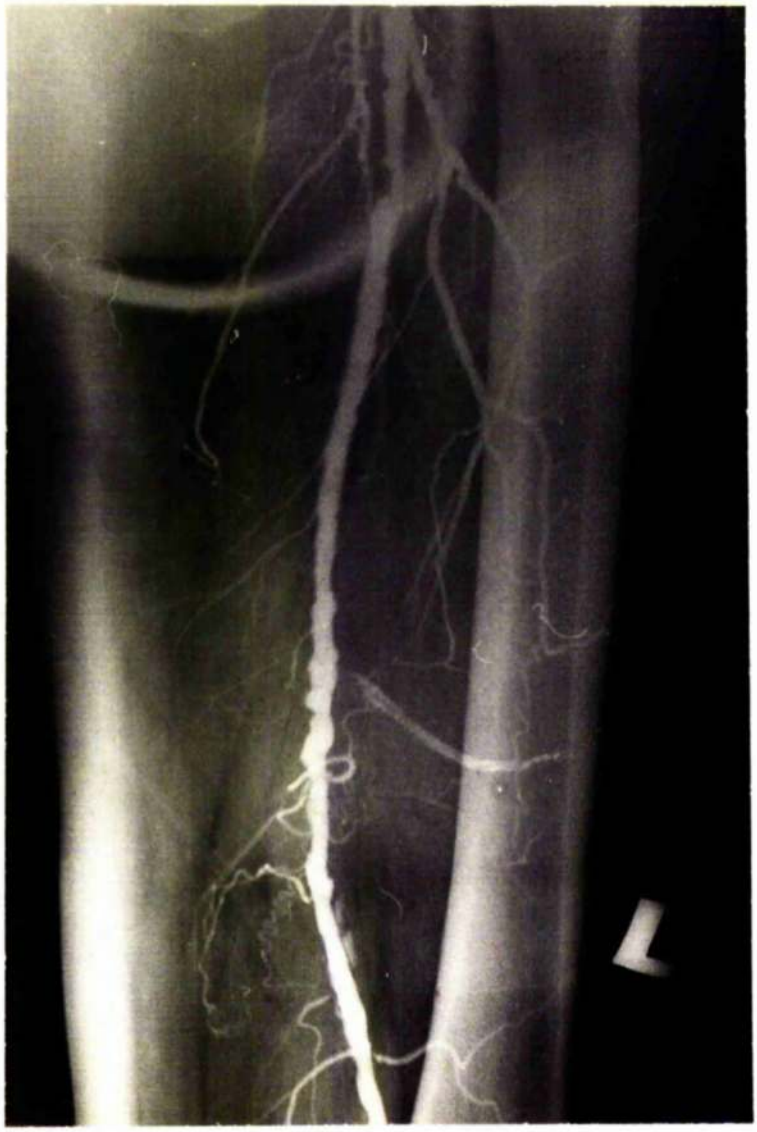


Fig. 35 : 'Beading' of Superficial Femoral Artery.

The left femoral arteriogram shows gross irregularity of outline of the femoral and popliteal arteries which is more severe than in Fig.34. The middle of the artery presents a 'beaded' appearance and there is evidence of calcification in the arterial wall at the level of the marker. Collateral vessels are also developing at this level.

Atherosclerotic changes in the profunda femoris artery are also visible.

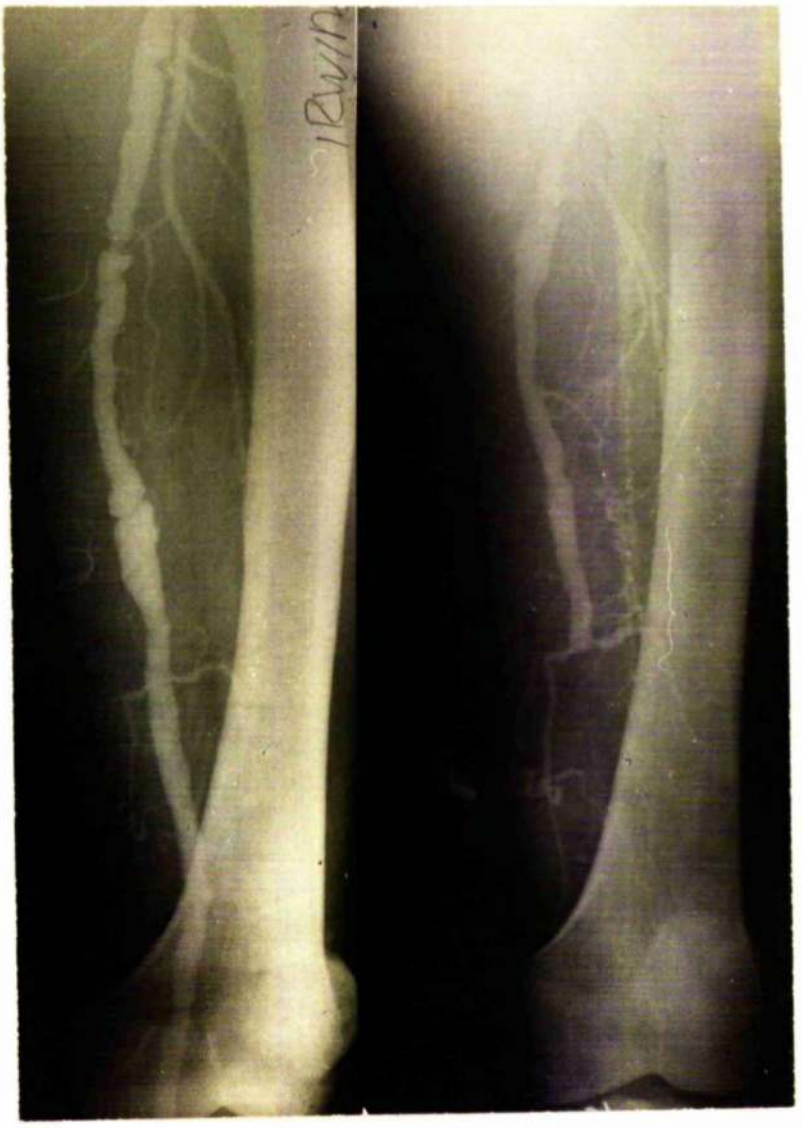


Fig. 36 : Total Popliteal Occlusion.

M.I., aged 45 years, was first seen in 1960 with bilateral claudication at 300 yards distance. Bilateral femoral arteriography showed gross dilatation and irregularity of both femoral arteries.

The left femoral artery is illustrated here, and narrowing of the lumen in several places is evident. In 1961, he developed the occlusion illustrated in the right hand photograph. Ischaemic rest pain was relieved by phenol injection of the left lumbar sympathetic chain but he remained severely handicapped by claudication until his death from coronary thrombosis later that year.

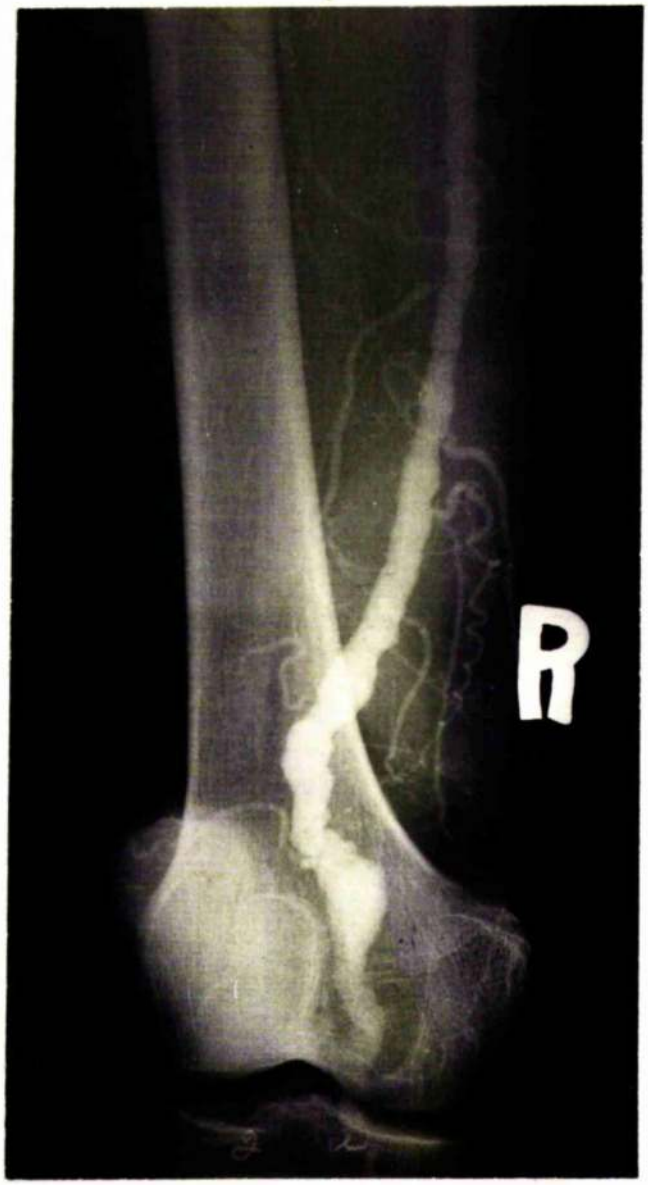


Fig. 37 : Multiple Popliteal Dilatation.

This arteriogram of J.B., aged 61 years shows widespread irregularity and dilatation of the popliteal artery. The degree of dilatation present is aneurysmal in type although no definite aneurysm is present.

This patient suffered from severe bilateral claudication at 50 yards distance due to a left total popliteal occlusion and a right low popliteal occlusion involving the bifurcation. With the severe changes illustrated, it is not surprising that treatment failed to alleviate his claudication.

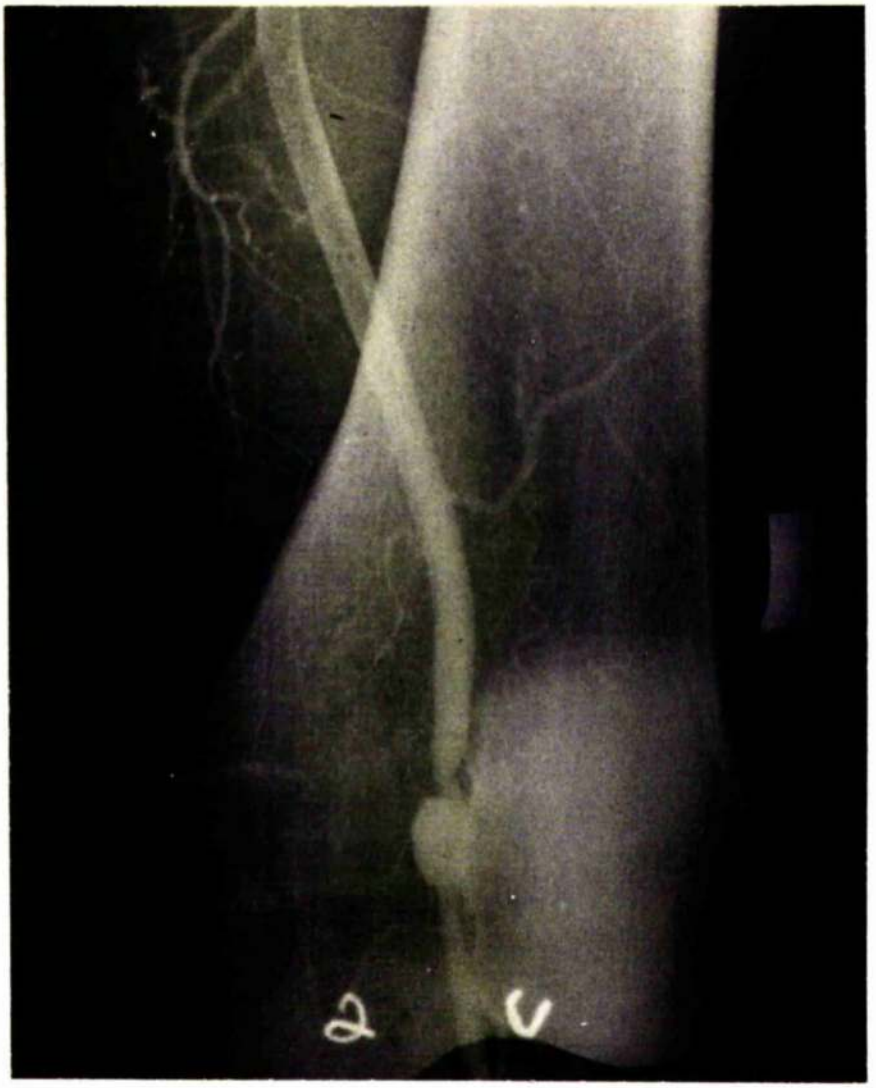


Fig. 38 : Popliteal Aneurysm.

This arteriogram shows a small saccular aneurysm of the left popliteal artery, the outline of the rest of the artery being smooth and regular.

It is interesting that this man of 37 years had experienced right sided claudication for 8 years and had total femoro-popliteal and anterior tibial occlusions in the right leg and an aneurysm in the left. The changes in the occluded leg were those of atherosclerosis but he developed migrating phlebitis which suggested the possibility of Buerger's disease. The distal lesions typical of this condition were absent and the arterial lesion is considered to be atherosclerosis.

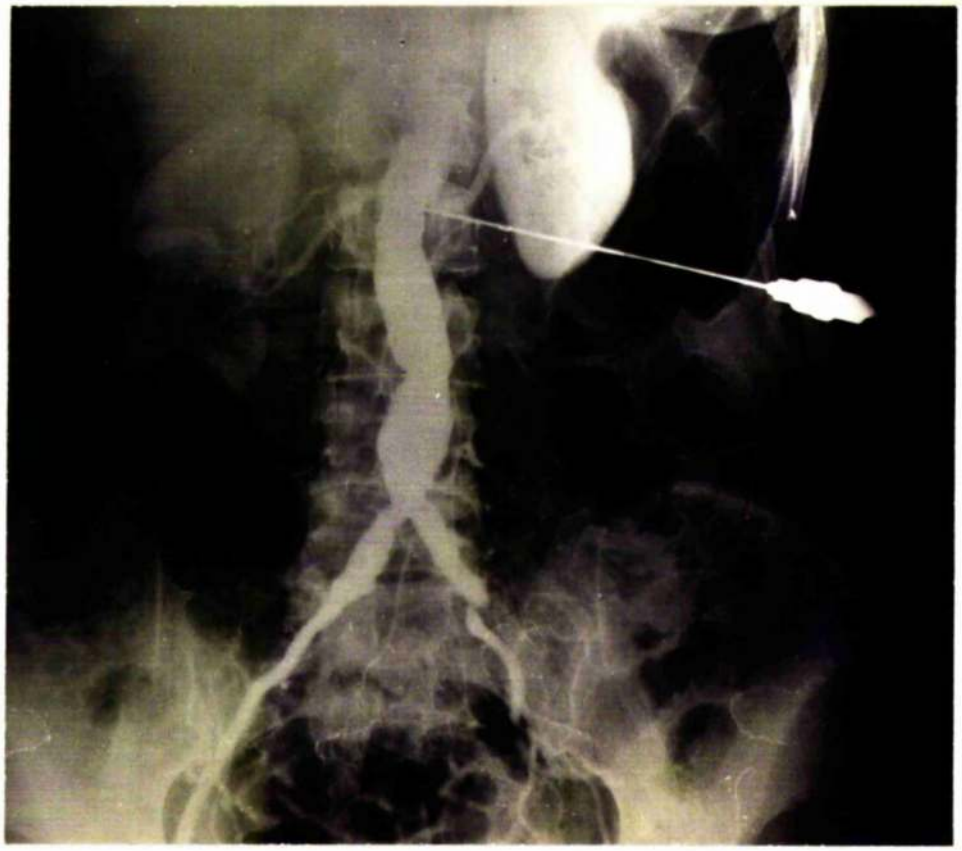


Fig. 39 : Early Aneurysmal Dilatation of the Aorta.

This aortogram of H.W., a 61 year old woman shows a left external iliac occlusion. The aorta is atherosclerotic and there is early fusiform aneurysmal dilatation of the lower abdominal aorta. This has not progressed since 1960 and surgical excision of the dilated segment has not been necessary.

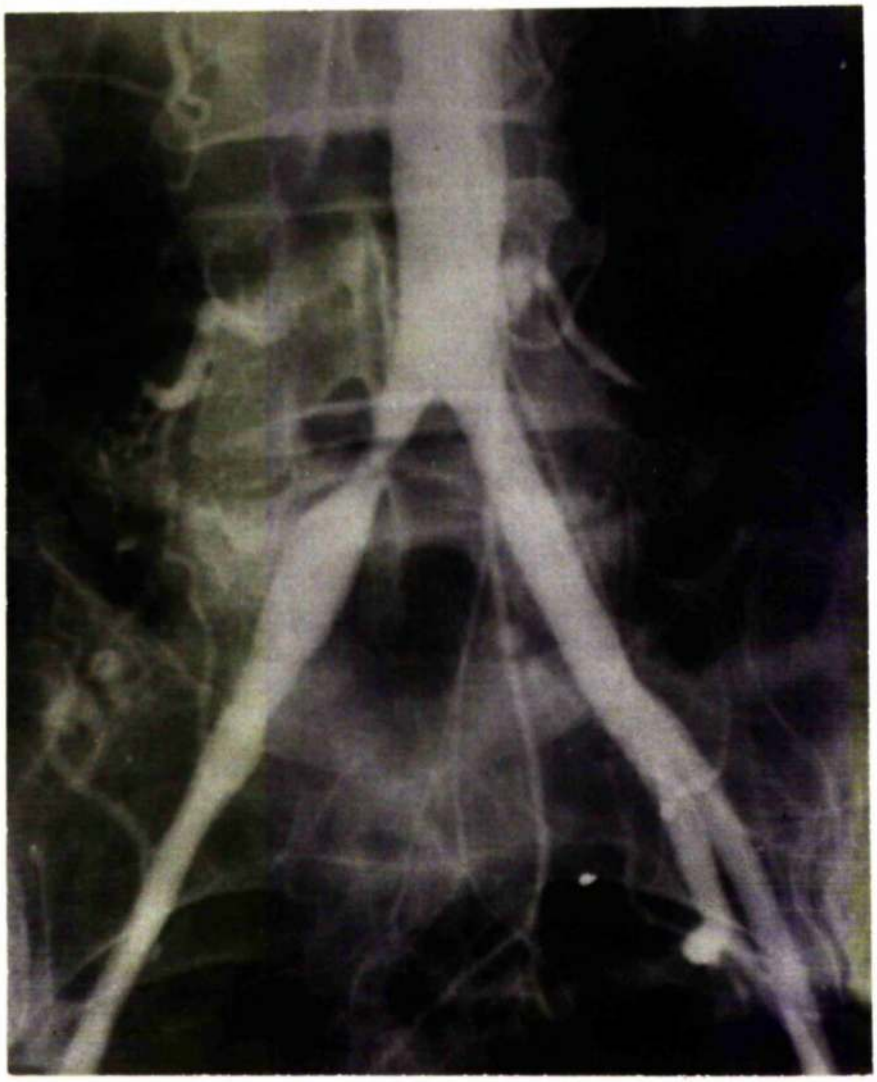


Fig. 40 : Post-Stenotic Dilatation of the Right Common Iliac Artery.

There is severe stenosis of the right common iliac artery affecting the proximal portion of the vessel near the aortic bifurcation. Post-stenotic dilatation is evident and at operation the anterior wall was found to be very thin.

In this man of 52 years, a short bypass teflon graft from the aorta to the dilated artery beyond the stenosis was inserted on 14.6.62. Subsequent convalescence was uneventful and the patient is now symptom free.

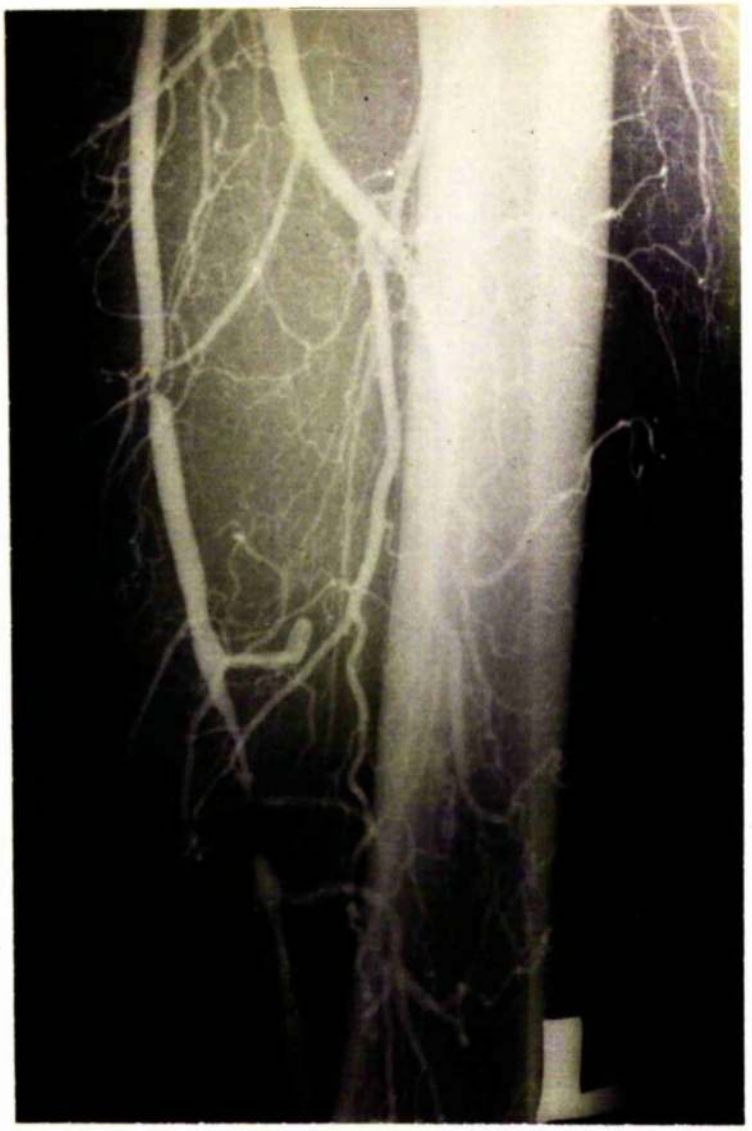


Fig. 41 : Persistence of Post-Stenotic Dilatation.

This left femoral arteriogram shows a short adductor occlusion with dilatation of the reconstituted main vessel distal to the block. This dilated area is due to a post-stenotic dilatation which has persisted after occlusion. The limiting collateral is narrowed at its origin and further extension in a proximal direction to the next collateral appears inevitable in the course of time.

The upper portion of the vessel shows an area of severe narrowing and numerous collateral vessels have arisen from the profunda femoris. These collaterals appear to curve towards the main vessel as they descend to make connection with the re-entrant collaterals.

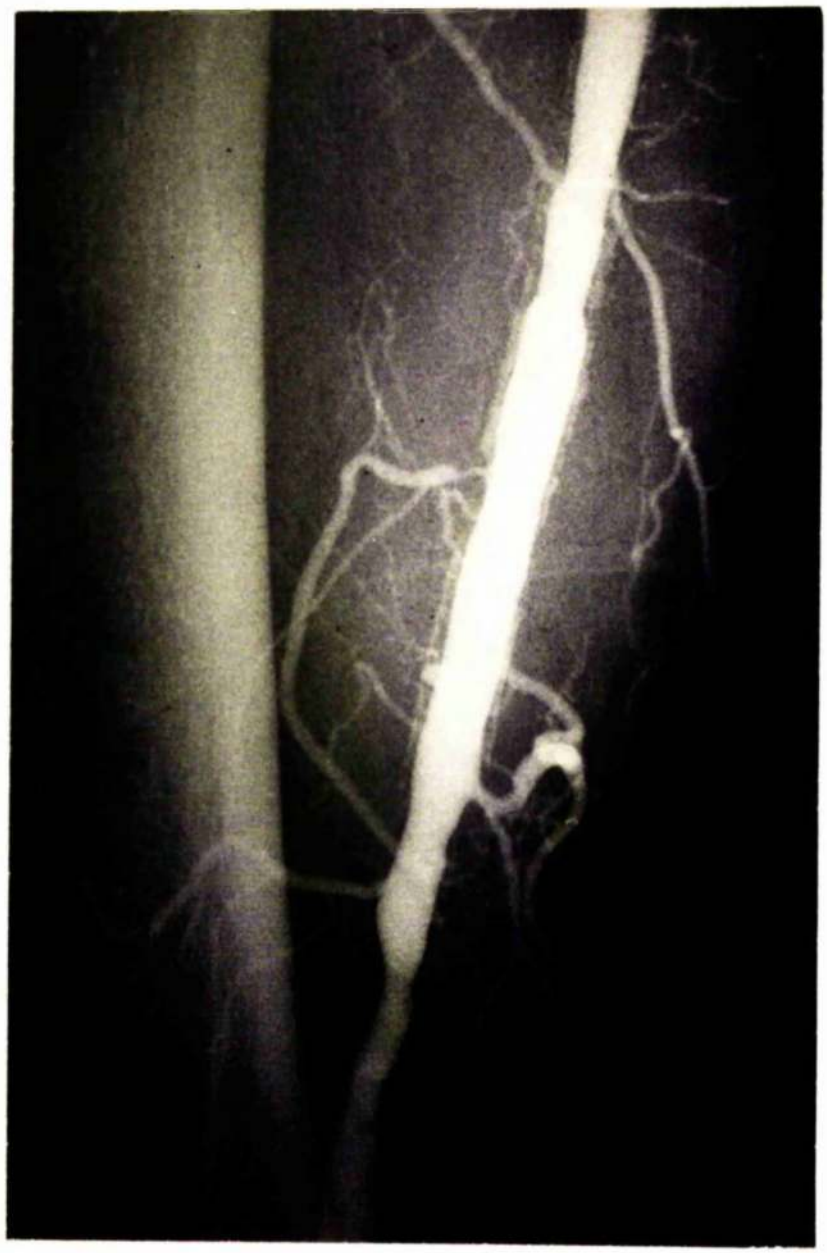


Fig. 42 : Calcification of Arterial Wall.

Several segments of calcified vessel can be seen and the diameter of the lumen is maintained at these levels. Irregularity of outline due to intimal thickening is very marked in the lower part of the artery.

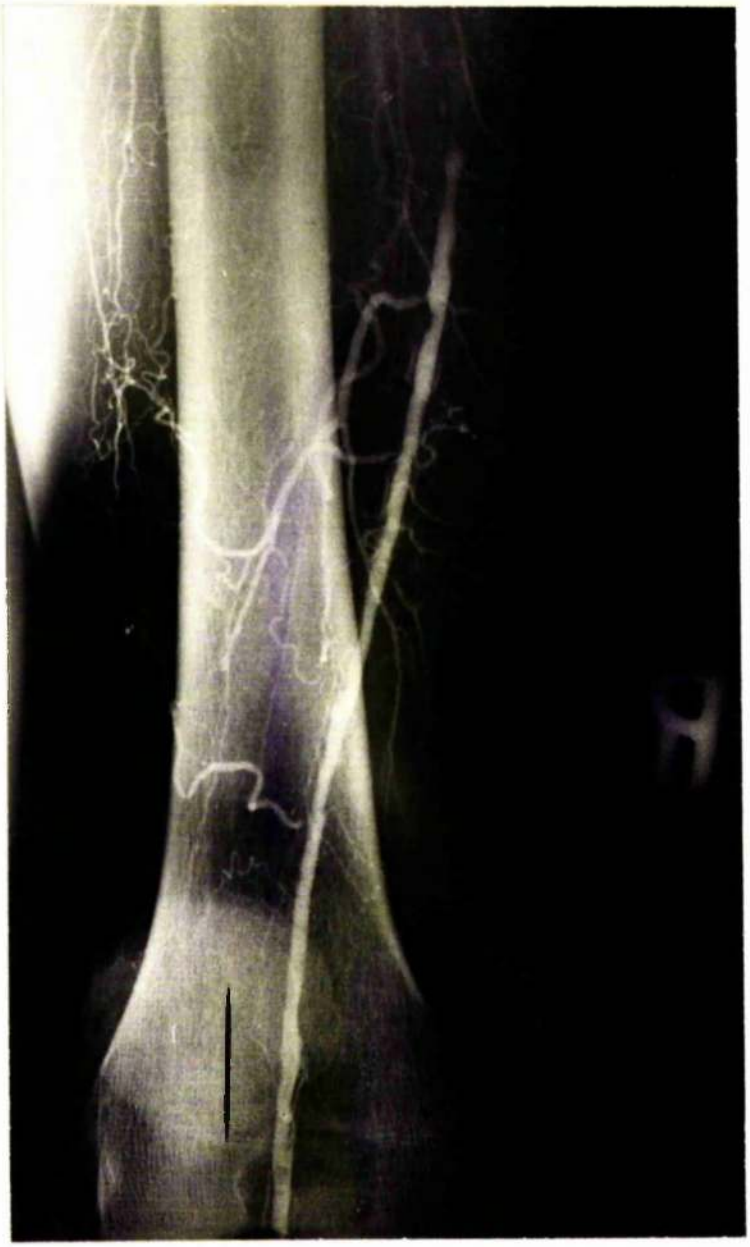


Fig. 43 : Arterial Calcification.

This right femoral arteriogram of a 67 year old man with claudication at 100 yards distance shows occlusion of the proximal segment of the superficial femoral artery. The lumen is irregular throughout but the diameter is maintained at two small areas of calcification near the upper end of the patent portion of artery.

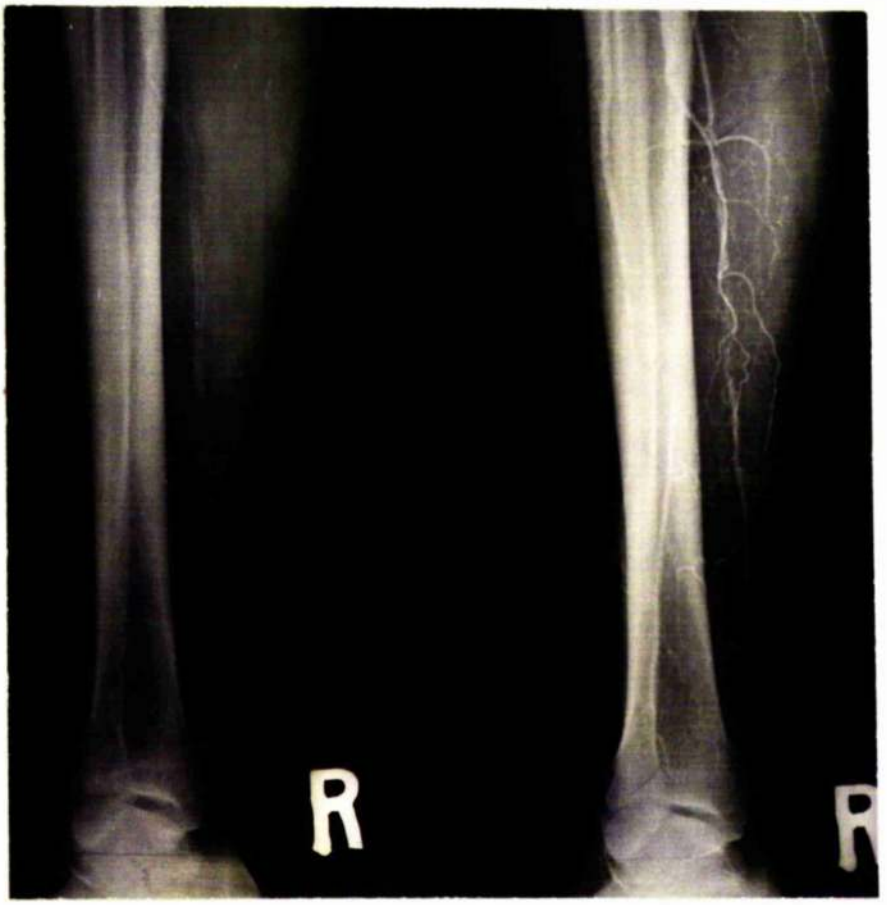


Fig. 44 : Calcification of the Posterior Tibial Artery.

These are the 2nd and 3rd films of a lower leg series. In the second film illustrated on the left some dye is visible in the anterior tibial artery. The posterior tibial artery is calcified and retains an outline suggesting calcification of the media.

In the third film illustrated on the right, dye is seen in the three lower leg arteries but the calibre of the posterior tibial is poor and there appears to be an occlusion at its middle.

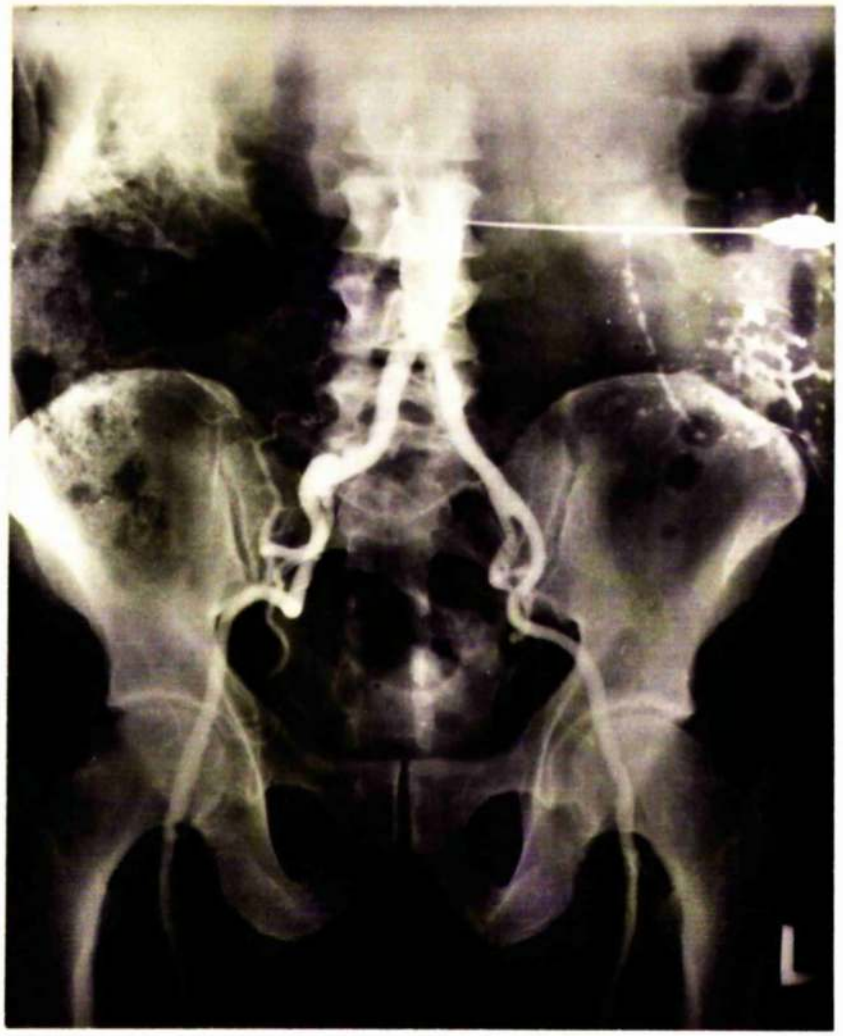


Fig. 45 : Plaque Formation at the Aortic Bifurcation.

This man of 59 years was seen in 1962 when he complained of bilateral claudication at 50 yards distance which dated from a myocardial infarct in 1960. Both femoral pulses were reduced in volume and aortography showed a lesion at the aortic bifurcation which produced narrowing of the orifices of both common iliac arteries. Both external iliac arteries are elongated and tortuous but were found to be relatively healthy at operation although the disease process was severe and extensive in the aorta and iliac arteries.

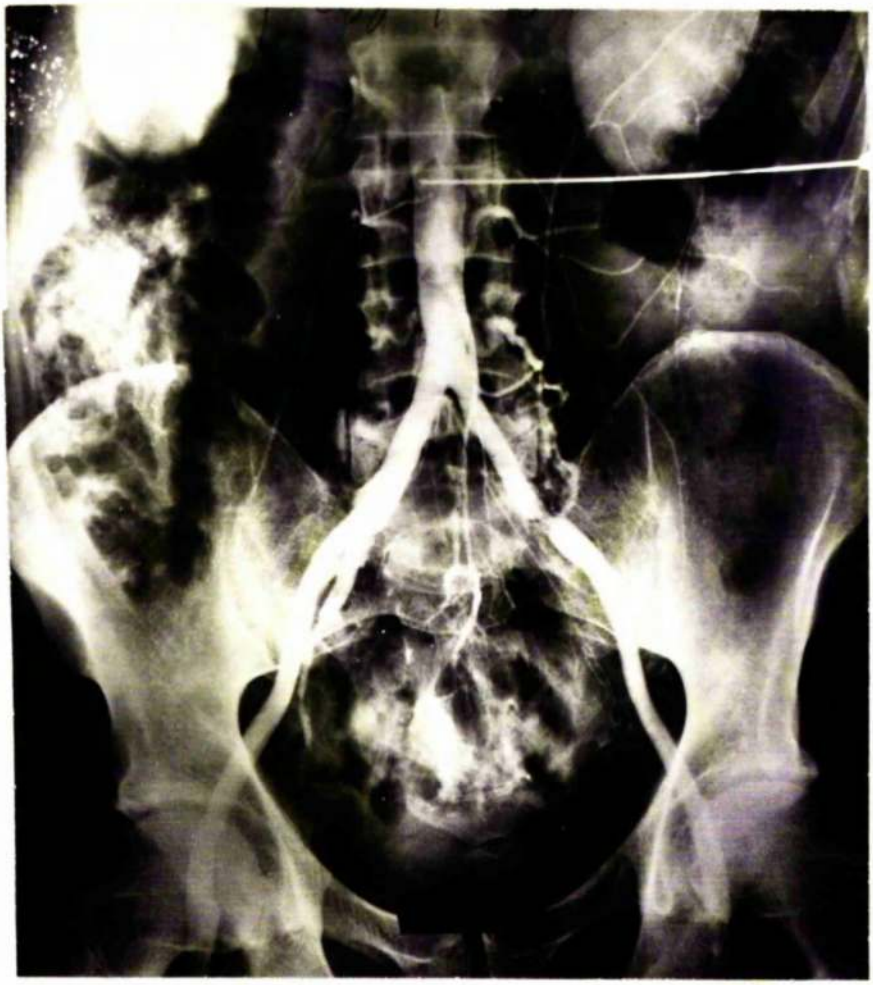


Fig. 46 : Plaque Formation at the Common Iliac Bifurcation.

There is an area of severe stenosis in the left common iliac artery at a level which corresponds with the common iliac bifurcation of the other side. The left internal iliac artery is occluded and this is probably secondary to plaque formation at the common iliac bifurcation. Further progression is liable to cause common and external iliac occlusion on this side.

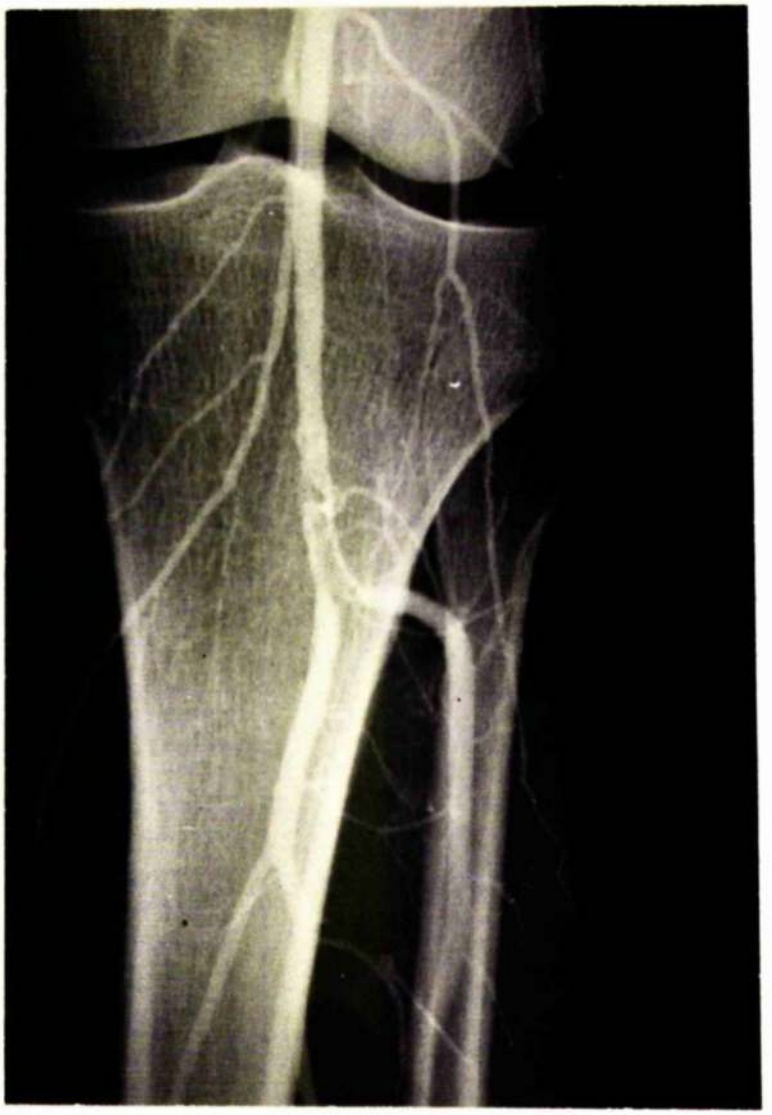


Fig. 47 : Plaque Formation at the Popliteal Bifurcation.

The popliteal and tibial vessels in this arteriogram are of reasonable calibre and outline except in the lowest part of the popliteal artery. The dye column in the lowest 2 cms. is reduced in density by a large atherosclerotic plaque at the popliteal bifurcation which is encroaching on the lumen from the posterior wall.

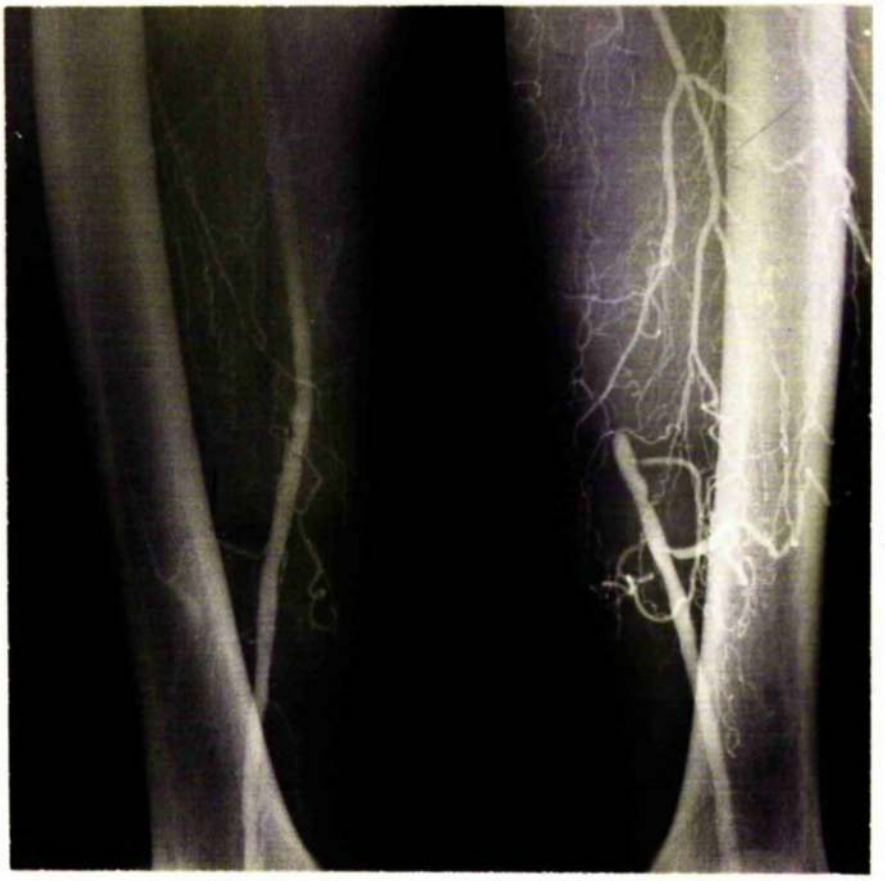


Fig. 48 : Narrowing at Orifices of Major Branches.

In the right femoral arteriogram there is some irregularity in the adductor region due to plaque formation and calcification is seen in the medial wall of the artery opposite the origin of a lateral branch.

The left arteriogram shows a long superficial femoral occlusion with an excellent collateral network derived from the profunda artery. The two collateral vessels show evidence of narrowing at their junction with the main channel.

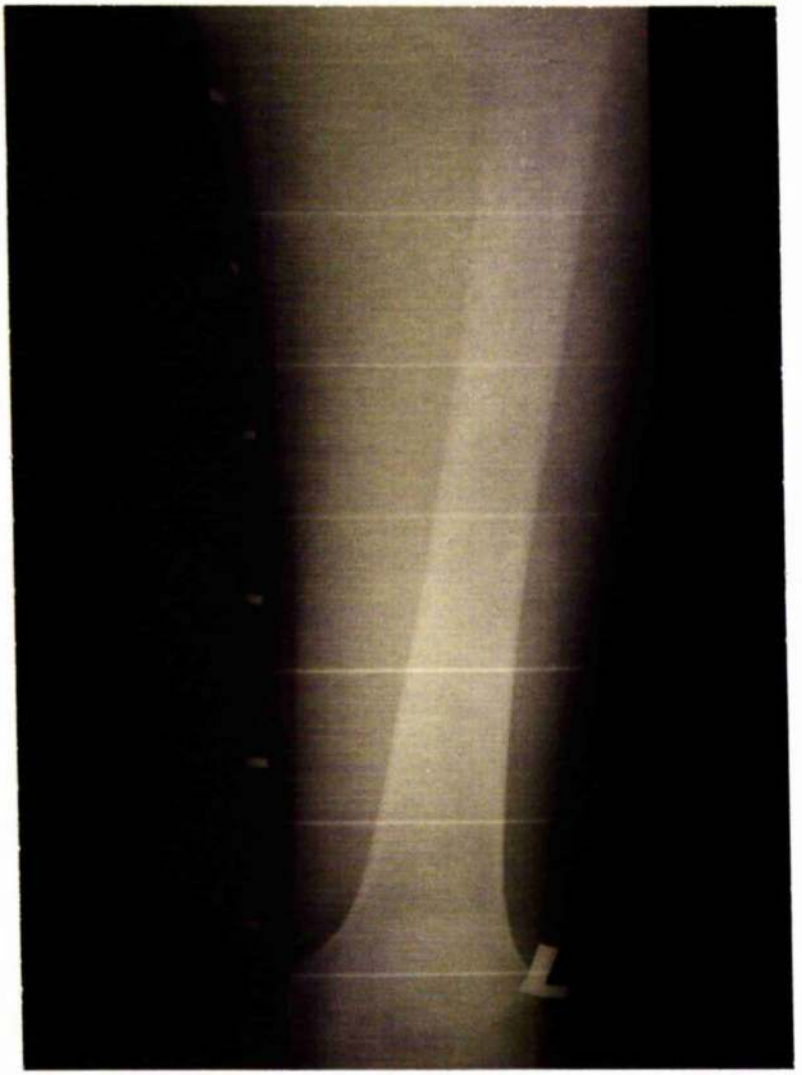


Fig. 49 : Magnification on to Film.

The unscreened film in the cassette tunnel was ruled with a graphite pencil at 5 cm. intervals and lead markers (seen on the left of the picture) were placed at 5 cm. intervals on the thigh at a level corresponding to that of the femoral and popliteal arteries. Calculation of the difference in distance between the lead markers and the transverse lines ruled on film indicate a magnification of 4% in the lower part of the thigh and 8% in the upper thigh.

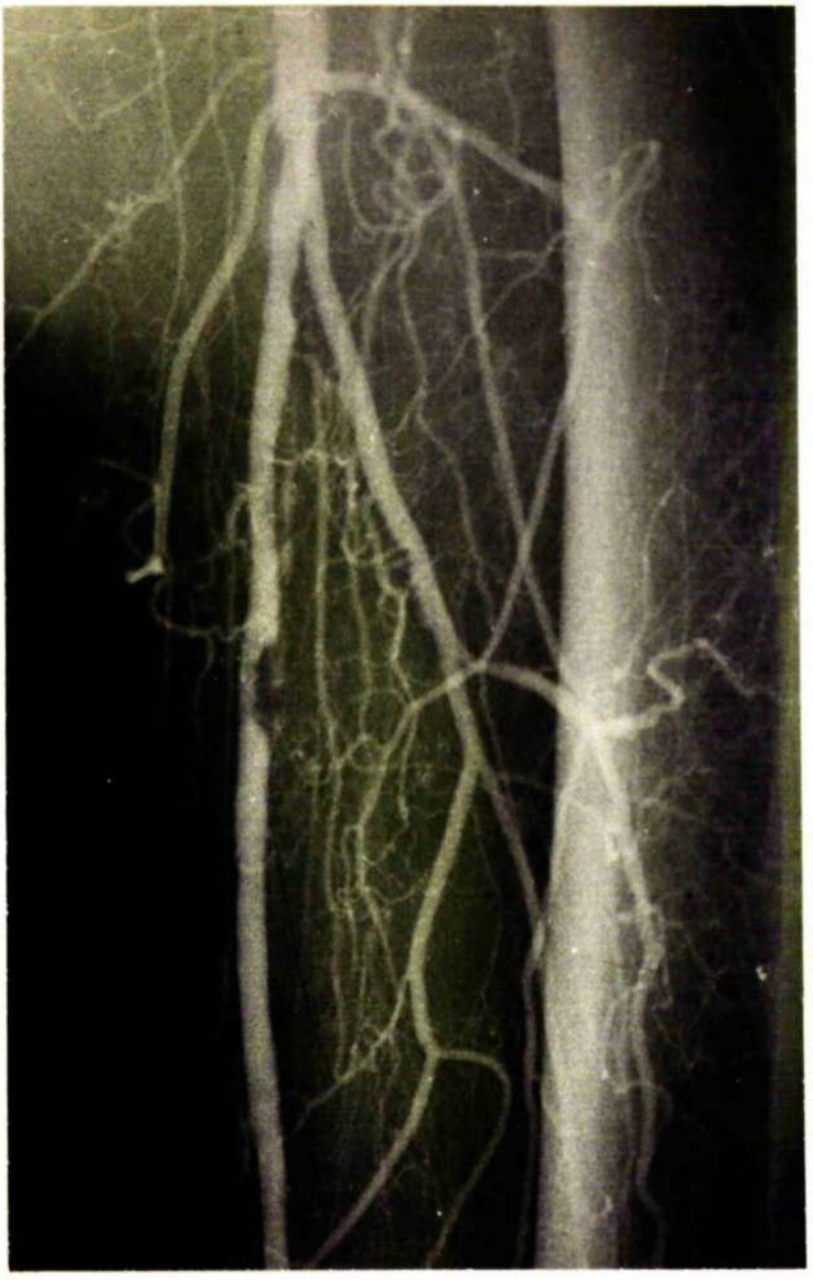


Fig. 50 : Plaque Formation in the Proximal Part of the Superficial Femoral Artery.

This femoral arteriogram provides an unusually clear picture of the common femoral junction which shows a moderate degree of narrowing due to plaque formation. The upper segment of the superficial femoral artery is narrowed and irregular in outline and calcium deposits are visible in the lateral wall.

A large plaque has constricted the vessel and greatly reduced the volume of flow but there are no large collateral vessels arising from the superficial femoral artery proximal to the lesion.

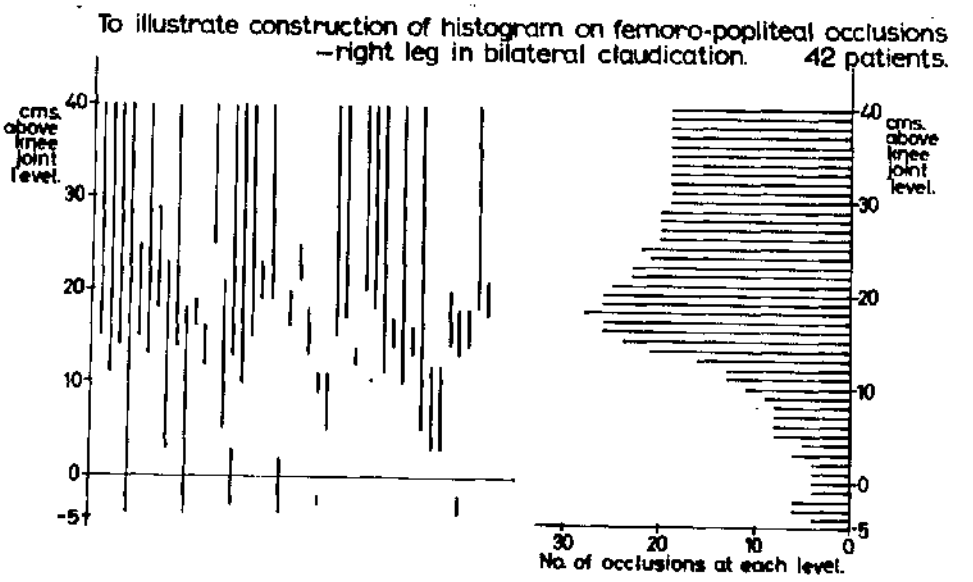


Fig. 51 : Method of Histogram Construction.

In the left hand side of this figure, the occlusions have been represented as vertical lines drawn against the scale on the left of the figure. Most of the long occlusions extend up to the 40 cm. level (the common femoral junction). The short occlusions are more variable but are most numerous at the middle of the scale - the adductor region.

The histogram on the right hand side is constructed by counting the number of vessels occluded at each level (e.g. 26 vessels are occluded at the 20 cm. level) and drawing a horizontal line of appropriate length at the corresponding level of the scale. Each line represents the number of vessels occluded at each level and the histogram indicates the rate of arterial occlusion in the femoral and popliteal arteries.

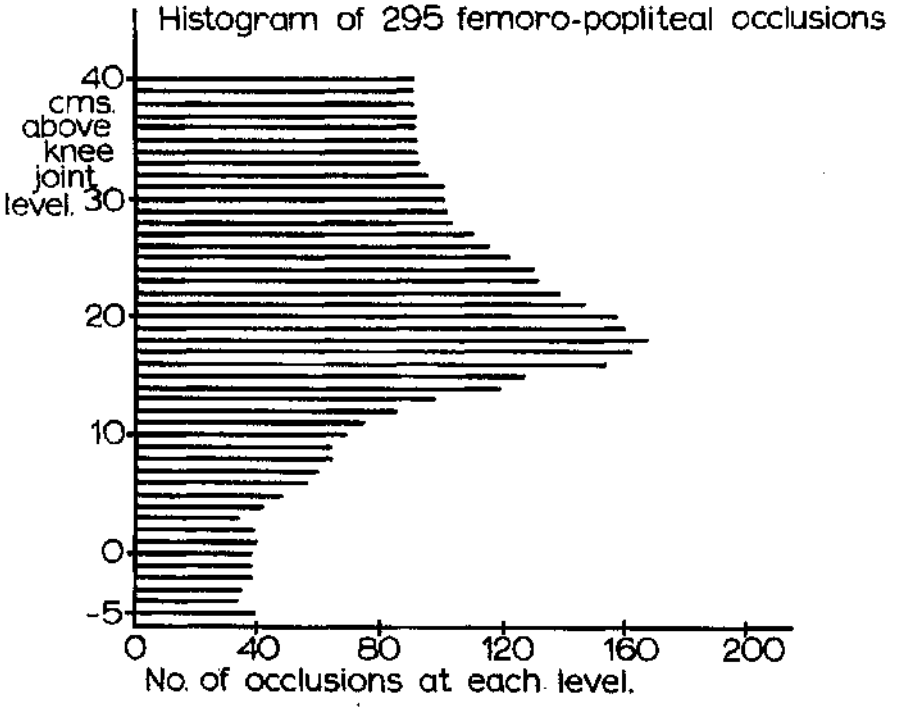


Fig. 52 : Histogram of 295 Femoro-Popliteal Occlusions.

This is the histogram of all the 295 femoro-popliteal occlusions found in the 528 femoral arteriograms of 264 patients. The appearances are similar to that seen in Fig.51.

The histogram is broad at the top due to the large number of long occlusions extending up to the common femoral junction and there is a minor degree of variation in the 30-40 cm. segment. The rate of occlusion increases lower down and the large adductor peak is smooth and extends from 27 to 14 cms., being maximal at 18 cms.

Below 14 cms. the rate of occlusion falls rapidly but the curve is not so smooth and it tends to straighten out at the level of the knee joint.

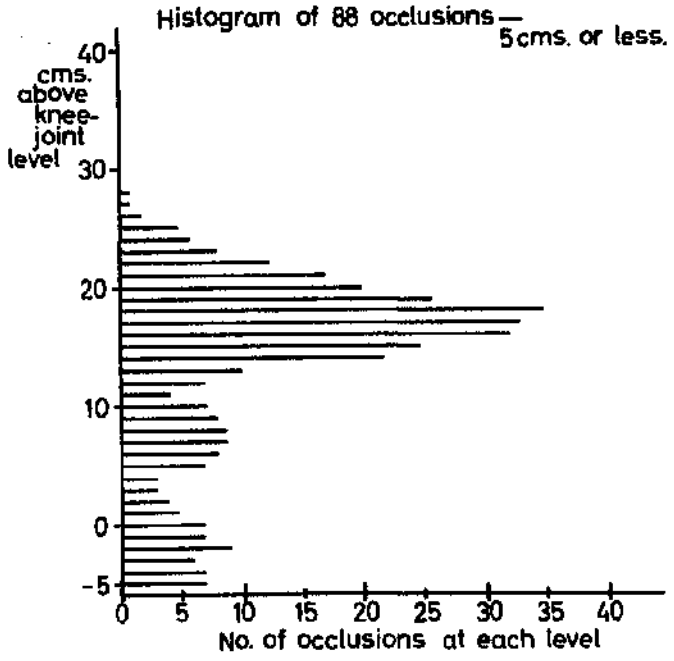


Fig. 53 : Histogram of Occlusions Measuring 5cms. or less.

This histogram of 88 femoro-popliteal occlusions measuring from one to five cms. in length shows a large peak which extends from 14 cms. to 21 cms. (approximately) and is maximal at 18 cms.

The second feature is the appearance of subsidiary peaks from 5 to 10 cms. and from -5 to 0 cms.

There is evidence of a lower rate of occlusion between the peaks and the absence of any observations above 28 cms. indicates that short occlusions have not been seen in this upper segment in the 264 patients investigated.

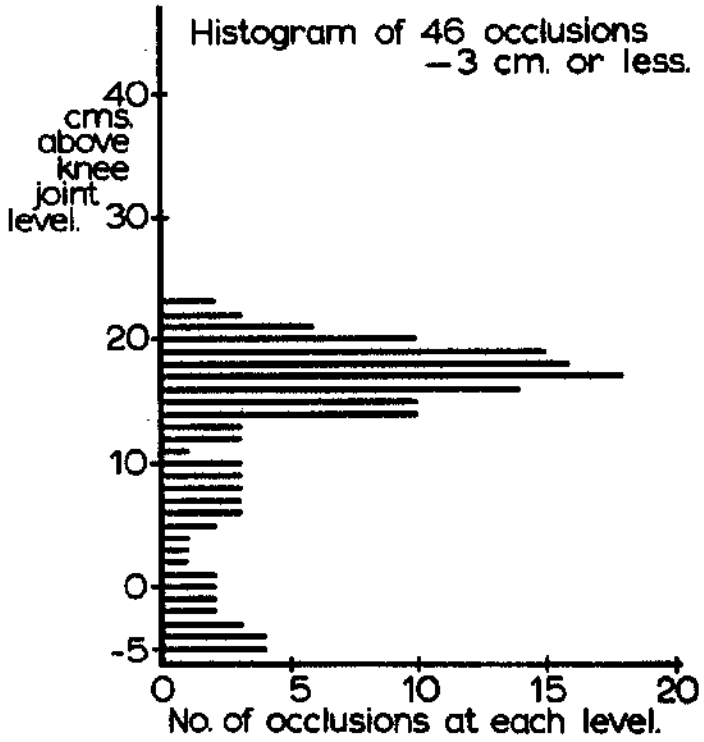


Fig. 54 : Histogram of Occlusions Measuring 3cms.or less.

This histogram is similar to the previous histogram (Fig.53). Three peaks are again visible. The principal peak extends from 14 to 21 cms. and is maximal at 17 cms. The middle peak extends from 5 - 10 cms. and the lowest peak from -5 to 1cm.

In this figure there are no observations recorded above the 23 cm. level, and the lowest peak tends to increase in magnitude as it approaches -5 cms. (the popliteal bifurcation).

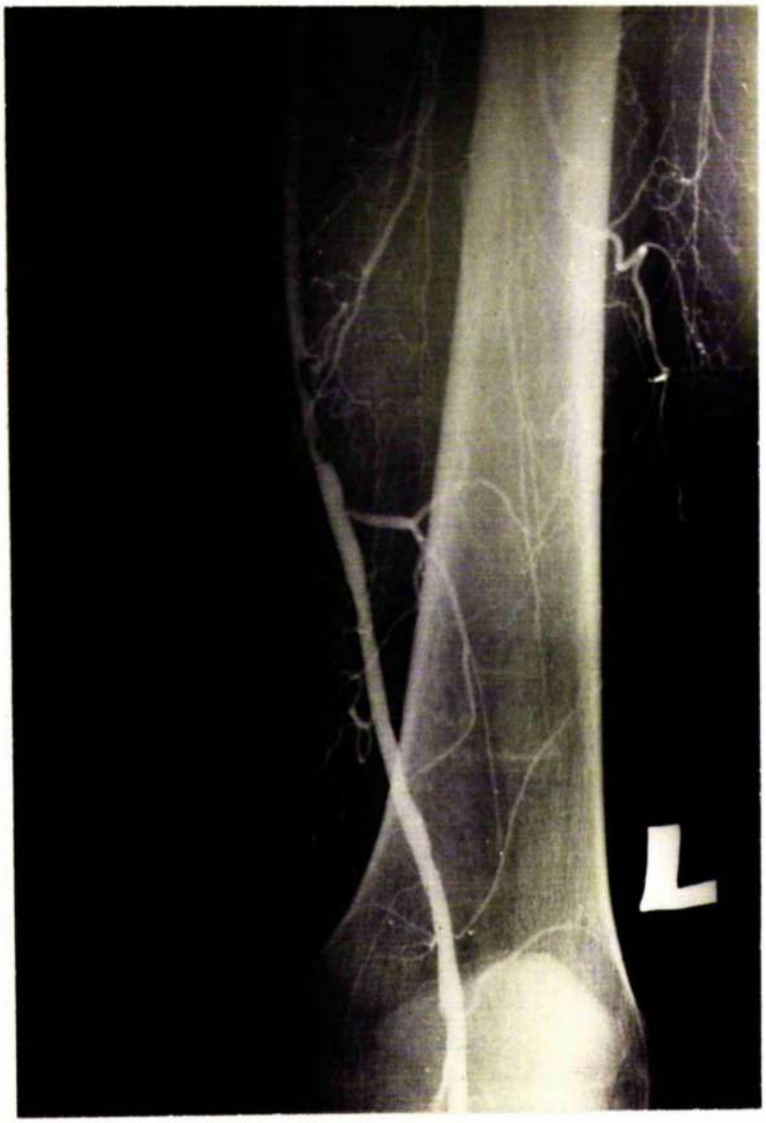


Fig. 55 : Incipient Arterial Occlusion.

This superficial femoral artery shows gross narrowing in the adductor region due to a large atherosclerotic plaque. Collaterals are already developing and it seems probable that occlusion will eventually occur.

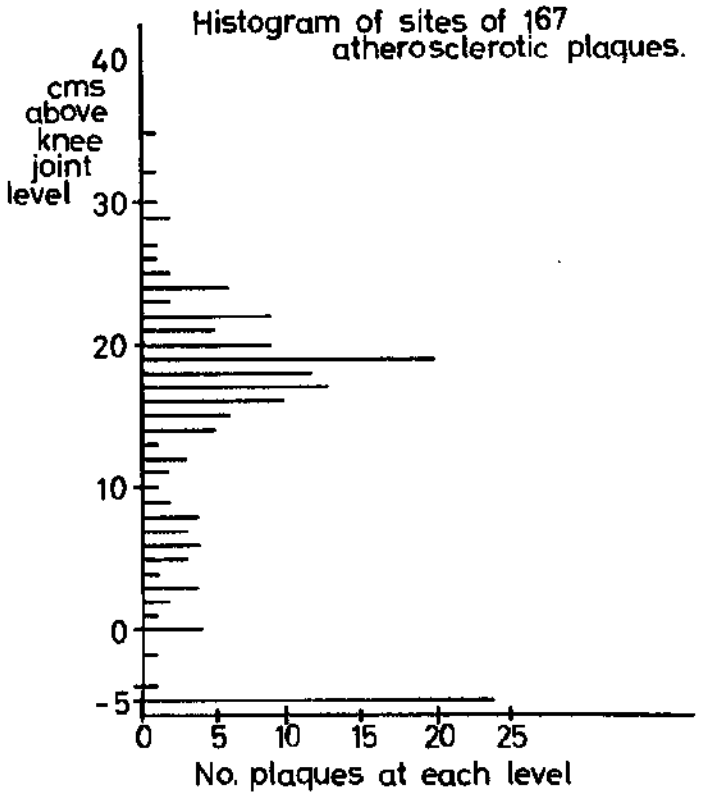


Fig. 56 : The Distribution of 167 Atherosclerotic Plaques in the Femoral and Popliteal Arteries.

This histogram illustrates the distribution of 167 atherosclerotic plaques causing severe narrowing of the femoral and popliteal arteries. The observations are widely scattered, especially in the popliteal artery from 0 to 12 cms., and the most prominent features are the large peak in the adductor region (14-24 cms.) with a maximum incidence at 19 cms., and the peak at the popliteal bifurcation (-5 cms.)

There are relatively few observations in the upper part of the superficial femoral artery (30 to 40cms) and in the popliteal artery below the level of the knee joint (0 to -5 cms.)

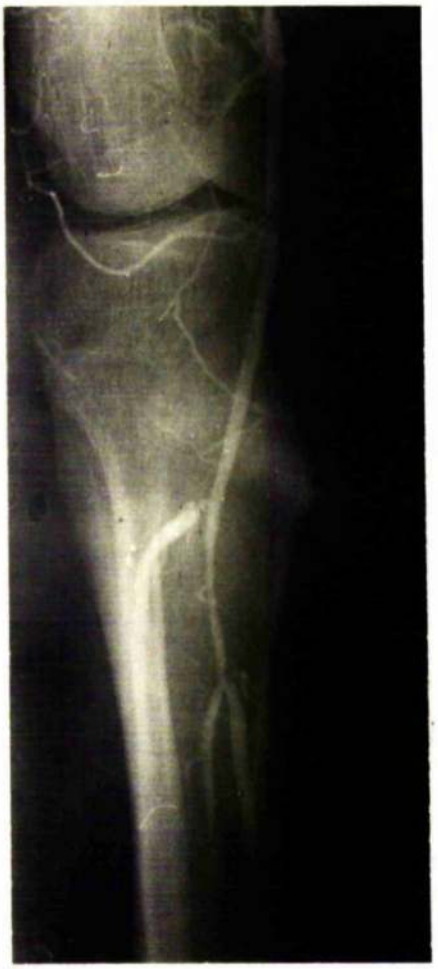


Fig. 57 : Plaque Formation at the Popliteal and Posterior Tibial Bifurcations.

This arteriogram shows atherosclerotic lesions at the popliteal and posterior tibial bifurcations. The anterior tibial artery is grossly narrowed at its origin and shows some post-stenotic dilatation, whereas the adjacent popliteal artery is of reasonable calibre and smooth in outline.

It seems probable that anterior tibial occlusion will occur in the course of time and the popliteal artery will remain patent.

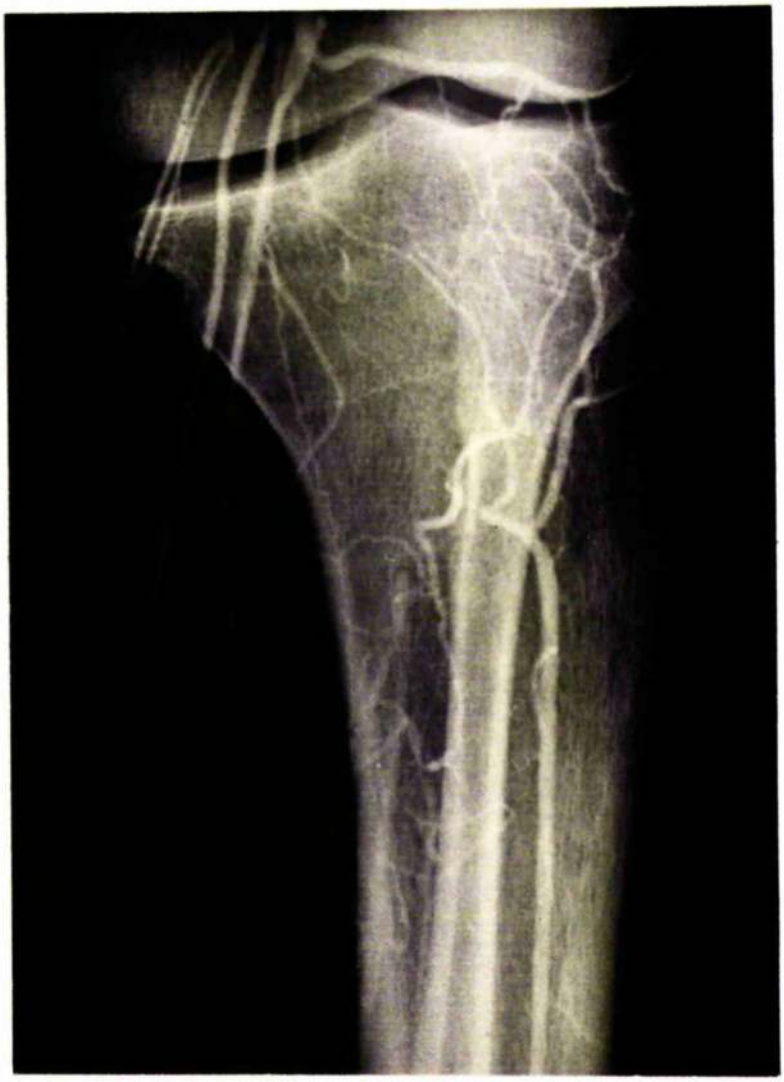


Fig. 58 : Low Popliteal Occlusion.

In this arteriogram, the popliteal artery is occluded from the origin of the sural arteries immediately above knee joint level to the bifurcation. The sural arteries (two in number) and the medial inferior genicular artery appear to form a leash of three vessels coursing downwards and medially.

The anterior tibial artery is reconstituted through its recurrent branch but is occluded just at its origin as is the posterior tibial artery. The posterior tibial and peroneal arteries show only a relatively faint shadow.

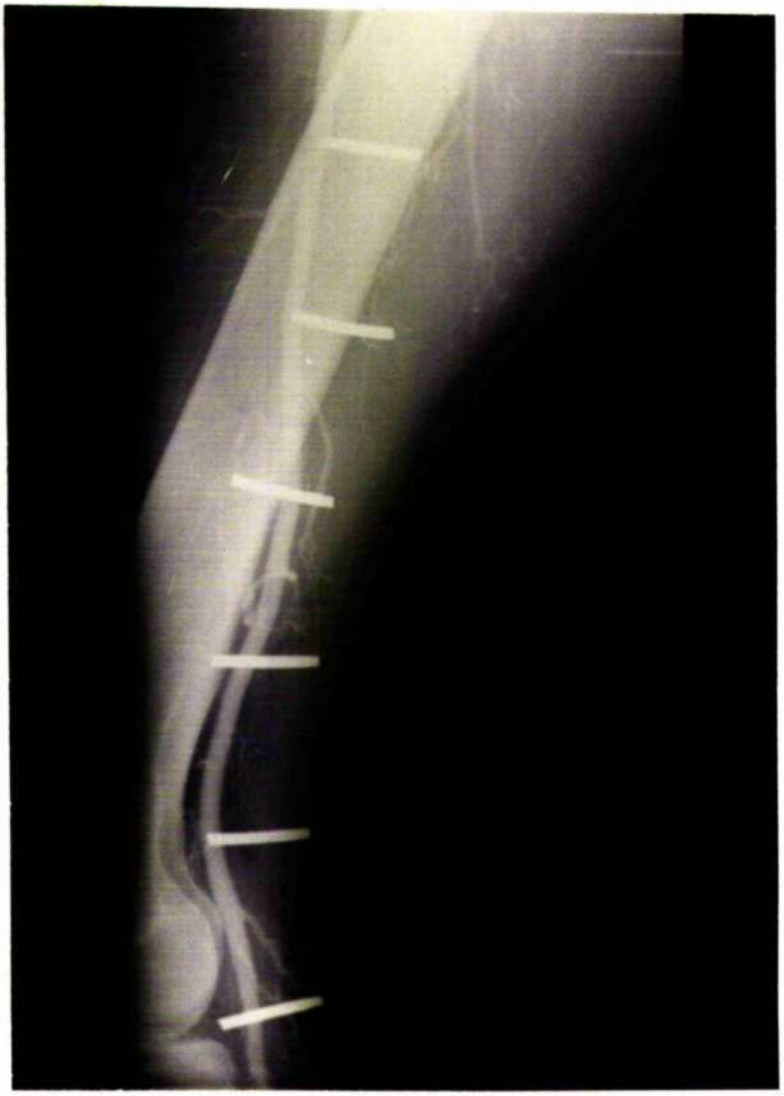


Fig. 59 : Lateral View of the Femoral Artery.

In this arteriogram, lead markers have been placed at 5 cm. intervals on the thigh, and the lowest is at the level of the knee joint. At 25 cms. from the knee joint, the arterial shadow crosses the anterior cortex of the femur and passes backwards to become the popliteal artery. Between 10 and 20 cms. there is a shallow S-shaped curve and it is probable that the adductor opening lies within this segment.

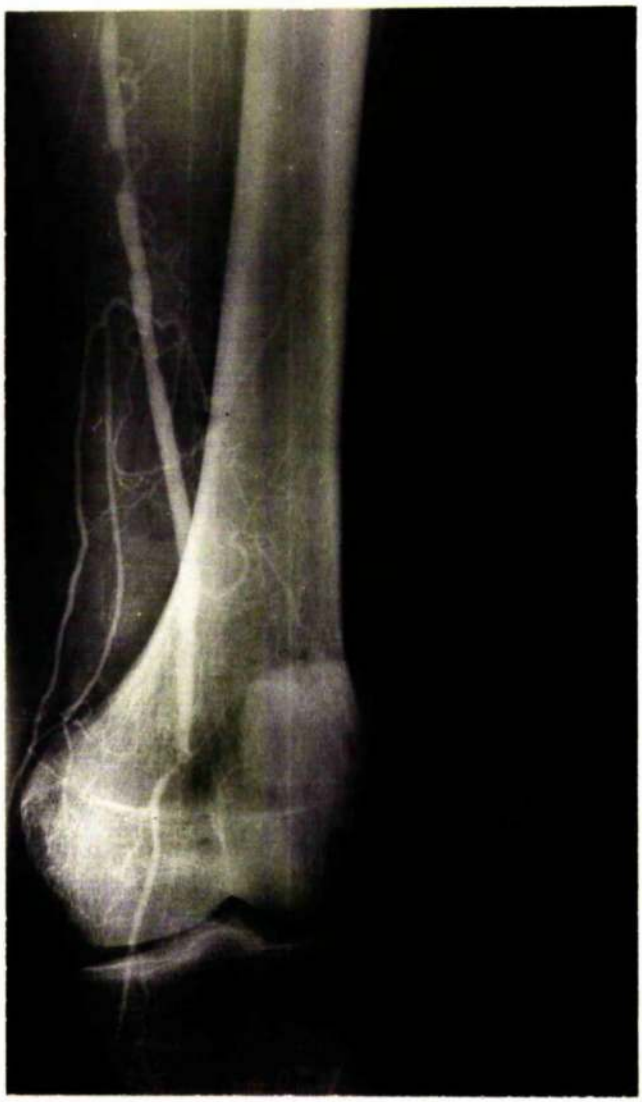


Fig. 60 : Low Popliteal Occlusion.

In this arteriogram, the popliteal artery is occluded up to the level of one of the sural arteries which is narrowed at its origin. Extension of the occlusion to the origin of the genicular artery is possible.

The saphenous and descending genicular arteries are prominent as they course downwards to provide a collateral circulation and the adductor opening probably lies just below the origin of these branches.

Above this level, there is a large plaque producing severe narrowing of the superficial femoral artery in the adductor region.

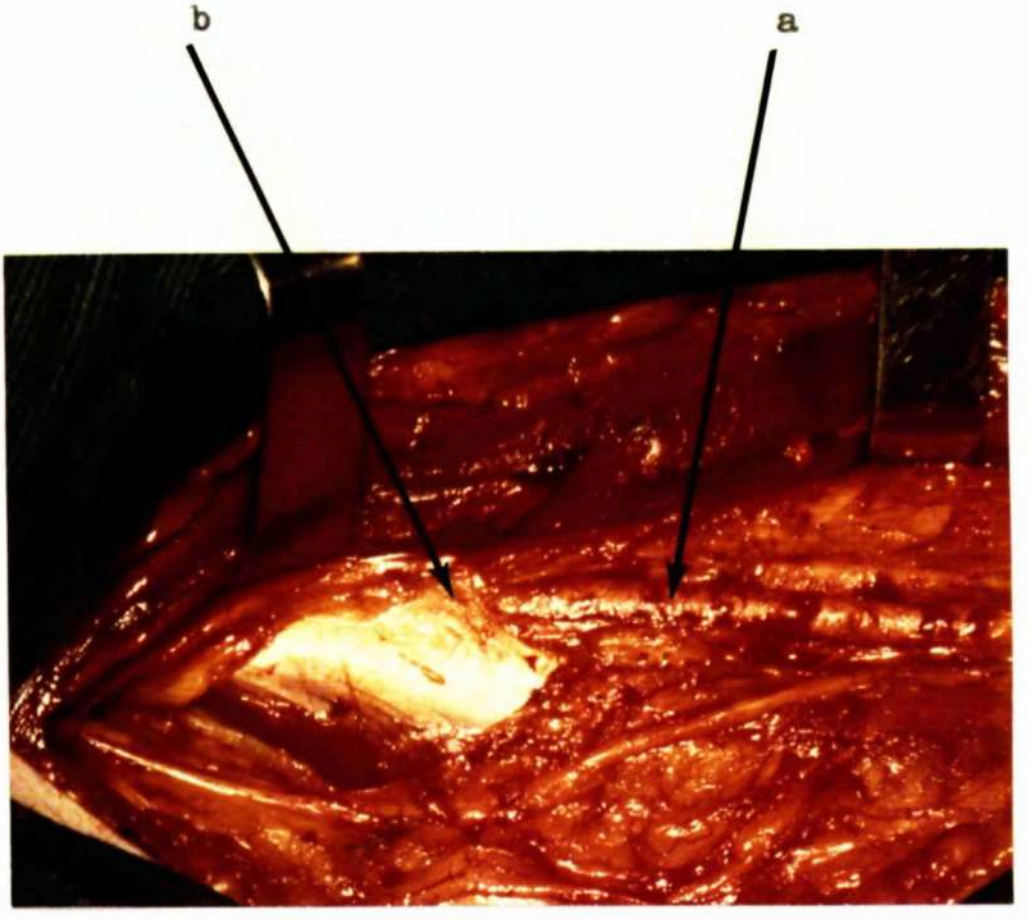


Fig. 61 : The Relationship of the Adductor Tendon to the Femoral Artery.

In this operation photograph, the glistening fibrous adductor tendon is prominent. The superficial femoral artery (a) is seen to pass through the adductor opening and, at this level, there is a firm triangular portion of tendon (b) whose free margin is in contact with the artery.

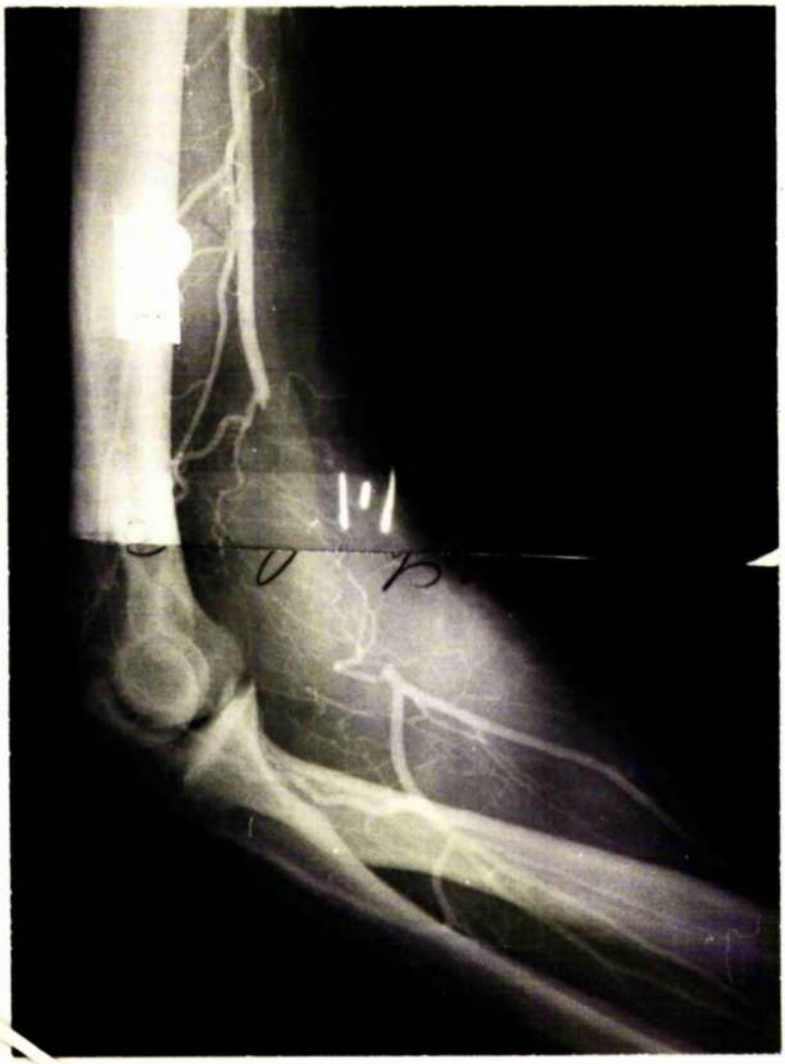


Fig. 62 : Traumatic occlusion of the Brachial Artery.

J.McM., aged 37 years, sustained a lateral dislocation of the right elbow with median and ulnar palsies. After reduction, the radial artery was impalpable but the arm was viable.

The arteriogram shows occlusion of the brachial artery at the level of the elbow joint with reconstitution at its bifurcation.

Following recovery of the nerve lesions, he returned to work but experienced claudication of the forearm muscles during heavy work. This was partially relieved by phenol block of the upper dorsal sympathetic chain and he has been able to continue working as a labourer.

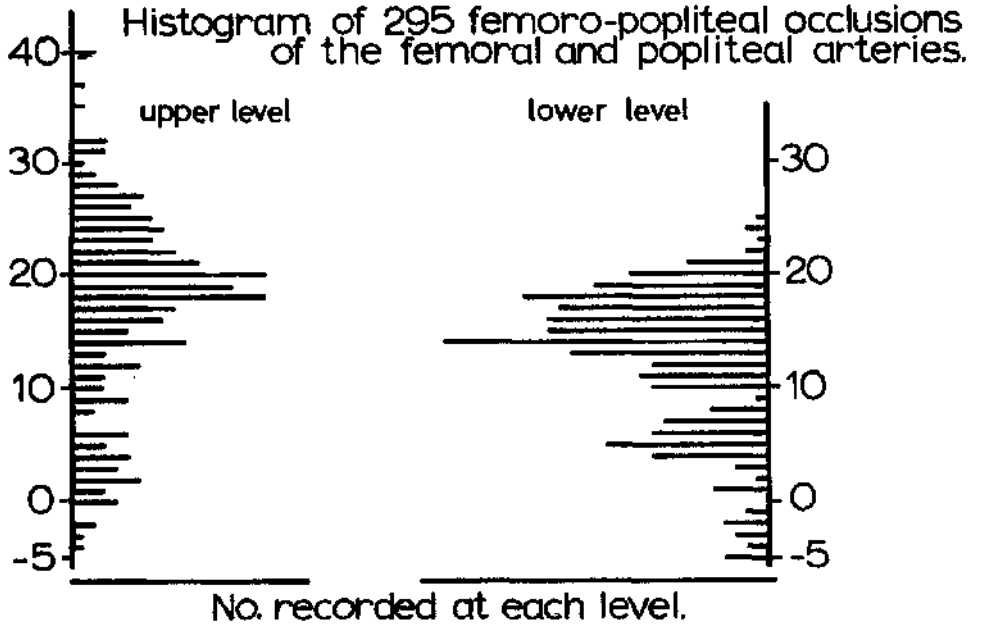


Fig. 63 : Histogram of the Upper and Lower Level of Occlusions.

In the left hand side of this figure, the upper level of the 295 femoro-popliteal occlusions has been plotted. There is a peak in the adductor region and a 'skip' area above the sural peak at 0 to 6 cms. Observations from 33 to 40 cms. and from -1 to -4 cms. are few.

The lower level of occlusion is recorded on the right. The adductor peak is naturally lower and extends into the upper part of the popliteal artery. The principal feature is the large number of observations at 14 cms. due to the descending genicular artery. The sural peak from 4 to 8 cms. is also very prominent.

When these two histograms are summated, Fig.64 is obtained.

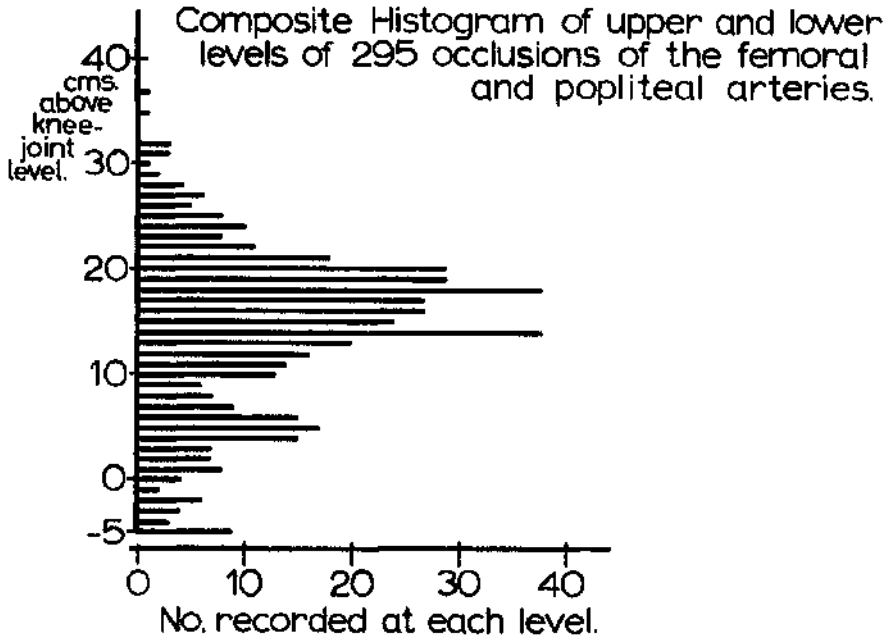


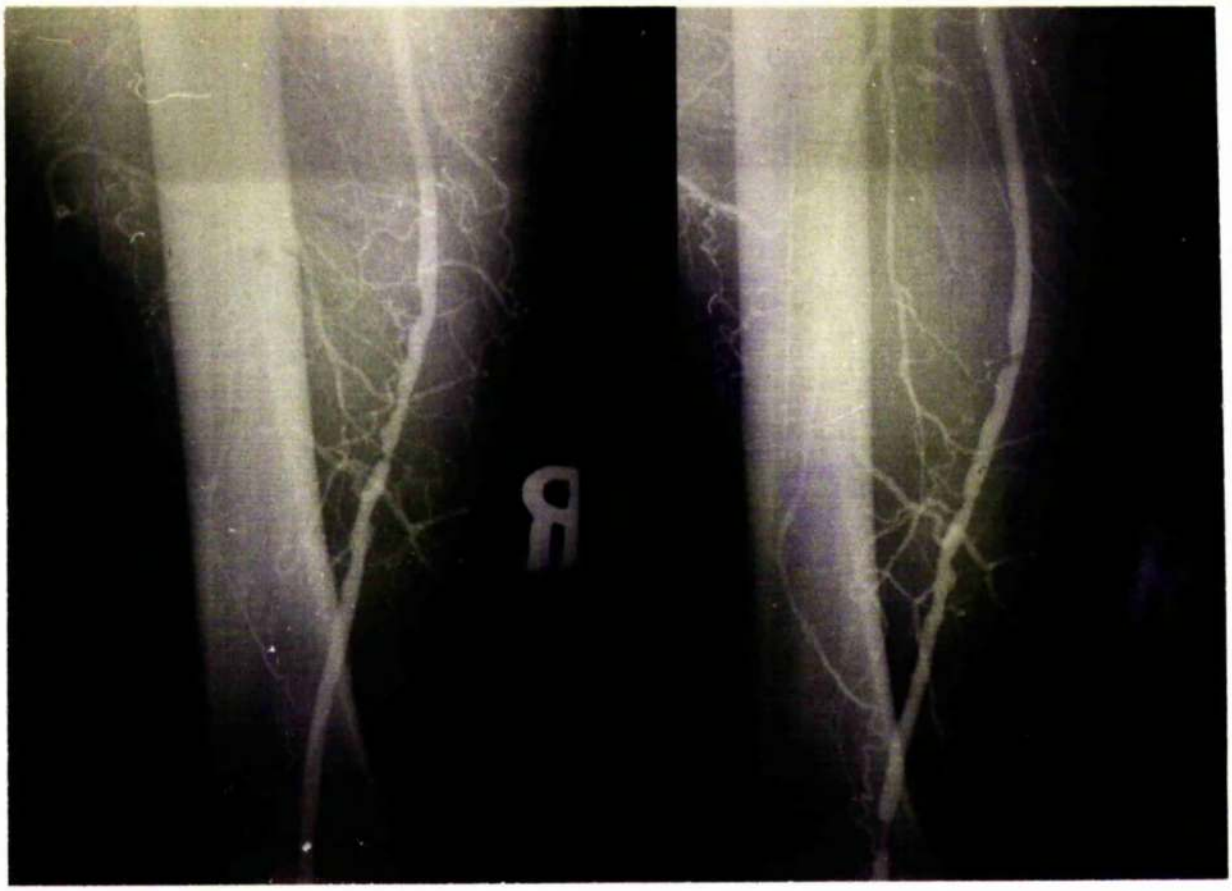
Fig. 64 : Distribution of Limiting and Re-entrant Collaterals.

This histogram is a summation of the two histograms illustrated in Fig.63 and shows the distribution of the limiting and re-entrant collaterals.

There is a large adductor peak with a maximum incidence at 18 cms. The large individual peak at 14 cms. appears to be due to the frequency with which the descending genicular artery occurs at this level.

The peak at 4 to 6 cms. is due to the sural vessels.

There is a relative absence of observations between 30 and 40 cms. and between 0 and -4 cms.



1959. 1960.

Fig. 65 : The Subsequent Development of a Popliteal Lesion.

J.A., aged 50 years, was investigated for right sided claudication in 1959 and the arteriogram shows gross atherosclerotic changes in the femoral artery without occlusion. Narrowing of the artery is severe at one point and there was an associated posterior tibial occlusion.

In 1960 his claudication became worse and a further arteriogram shows that he developed stenosis of the popliteal artery. The photograph on the right shows the lesion of the popliteal artery which was not present in 1959.

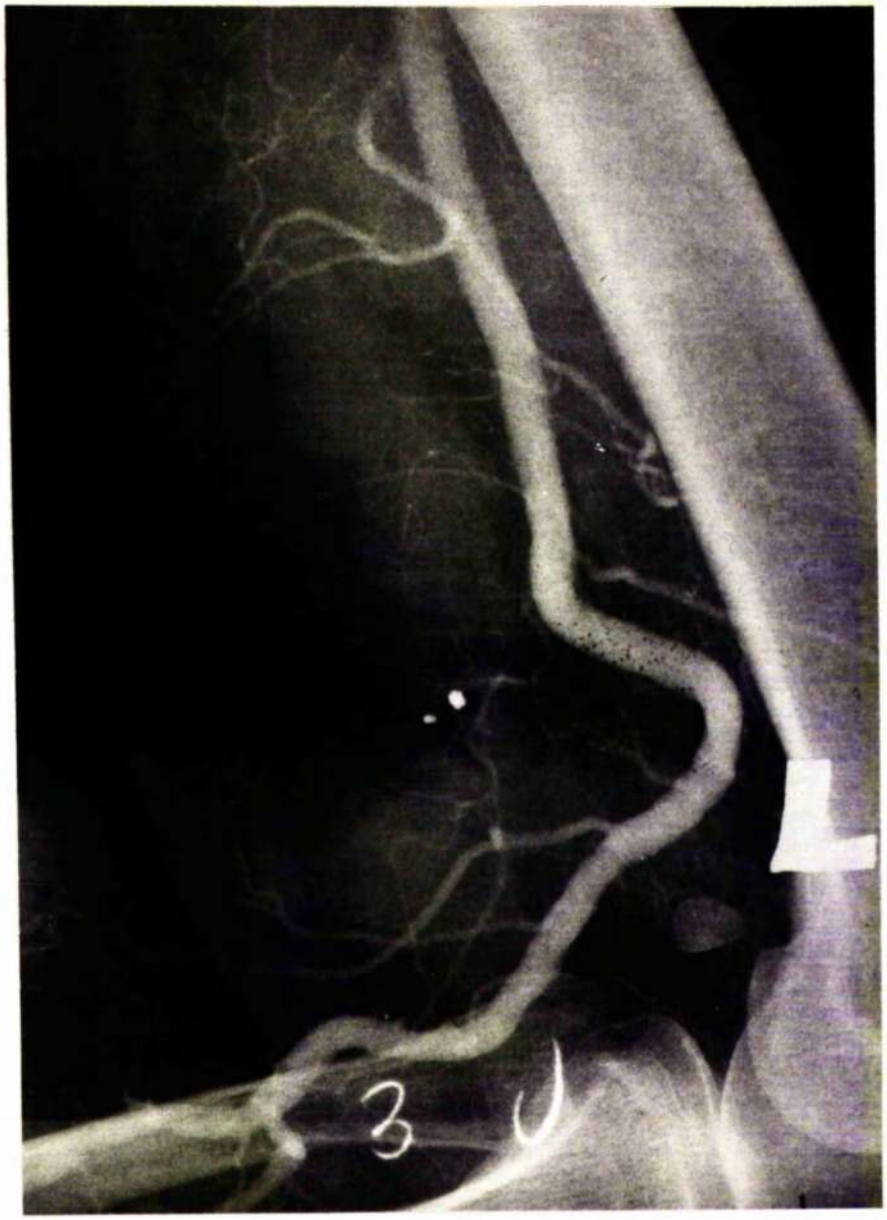


Fig. 66 : Curve Formation in the Popliteal Artery.

In this lateral arteriogram of the popliteal artery with the knee in flexion, the popliteal artery shows several curves due to the elongation and tortuosity occurring in an atherosclerotic vessel.

The proximal acute curve has large branches at the base of each limb of the curve and these may have created relatively fixed points between which the mobile artery is free to develop an acute curve.

From the knee joint to the popliteal bifurcation small secondary curves are present.

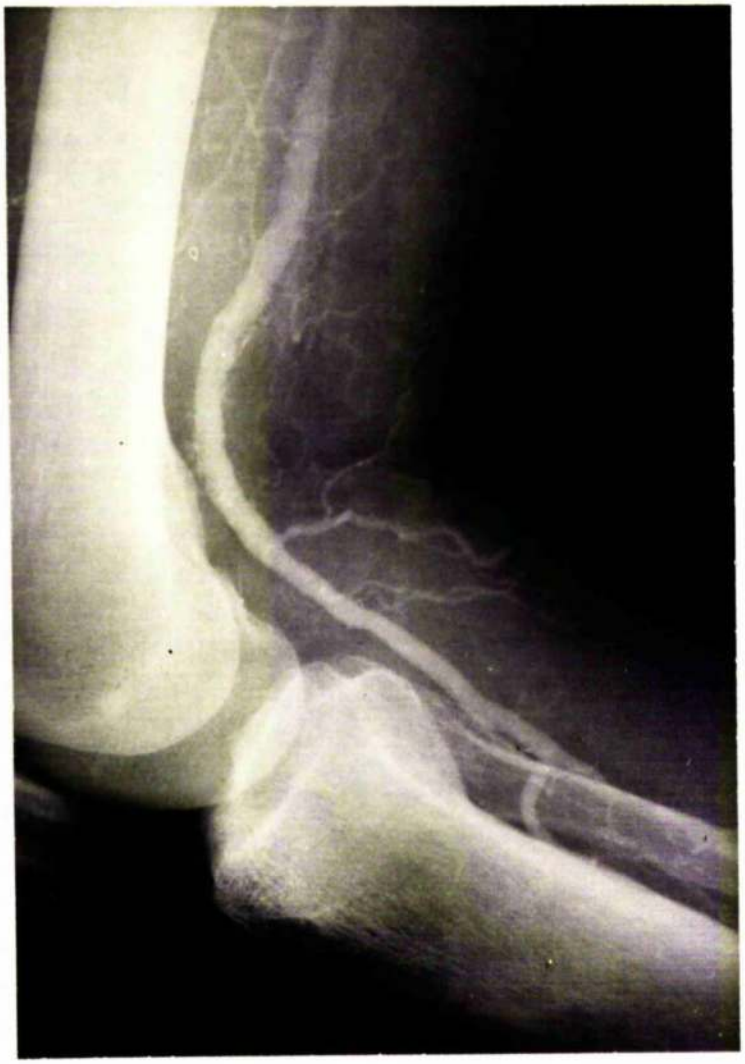


Fig. 67 : Plaque Formation in the Popliteal Artery.

Curve formation in this popliteal artery is not so pronounced as in Fig.66. The sural artery appears to lie at the lower end of the curve.

The artery is moderately atherosclerotic and irregular and this is most marked on the summit of the curve where plaque formation appears to be occurring on the convexity.

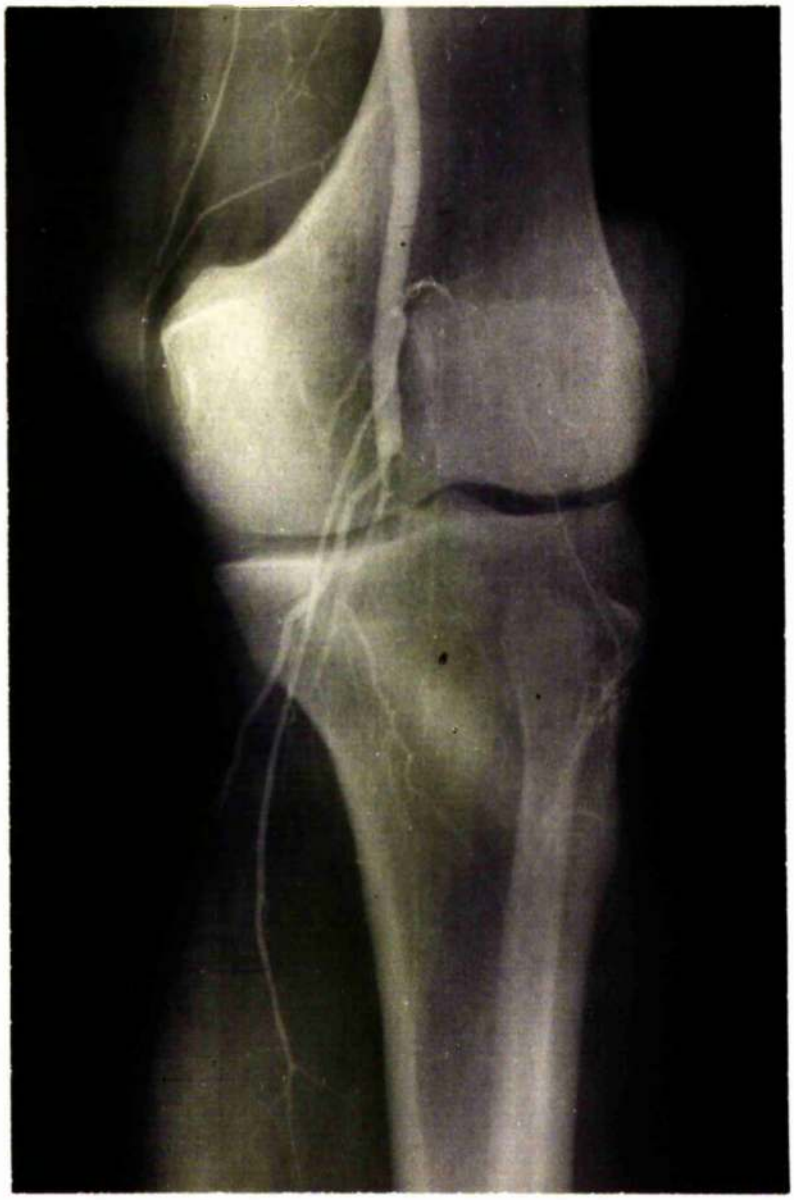


Fig. 68 : Low Popliteal Occlusion.

This arteriogram shows a low popliteal occlusion limited by the sural arteries which arise just above the level of the knee joint. In the A.P view, the sural arteries course downwards and medially as a leash of three vessels, one of which is the medial inferior genicular artery.

No reconstitution of the tibial arteries can be seen on this film.

Fig. 69 : Total Popliteal Occlusion.

W.B., an elderly man of 78 years, was first seen in January, 1960 when he developed gangrene of his left great toe. A femoral arteriogram performed on 24.1.60 showed occlusion of the whole length of the popliteal artery up to the level of the descending genicular artery. A few small collateral vessels could be seen below this level but no reconstitution of the main trunk was seen. A mid-thigh amputation was performed on 29.1.60.

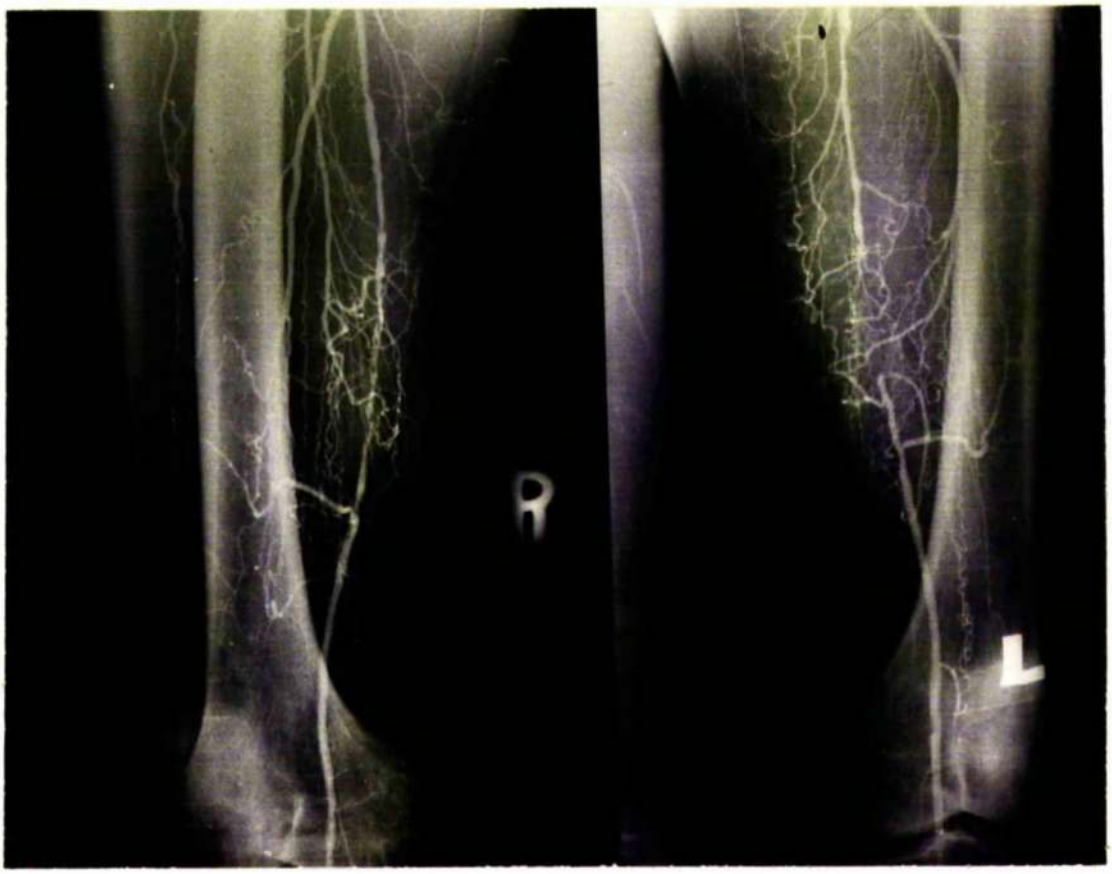


Fig. 70 : The Development of Collateral Vessels.

This bilateral femoral arteriogram shows lesions of the adductor region in both legs.

In the left leg, there is a short occlusion with numerous collaterals and, in the right leg, occlusion is incomplete.

The pattern of collateral development is similar on the two sides and it seems probable that symmetrical occlusions will develop. This patient (a diabetic aged 70 years) had bilateral symmetrical posterior tibial occlusions also.

The collateral vessels developing distally are larger in size than those arising from the artery above the occluded segments.

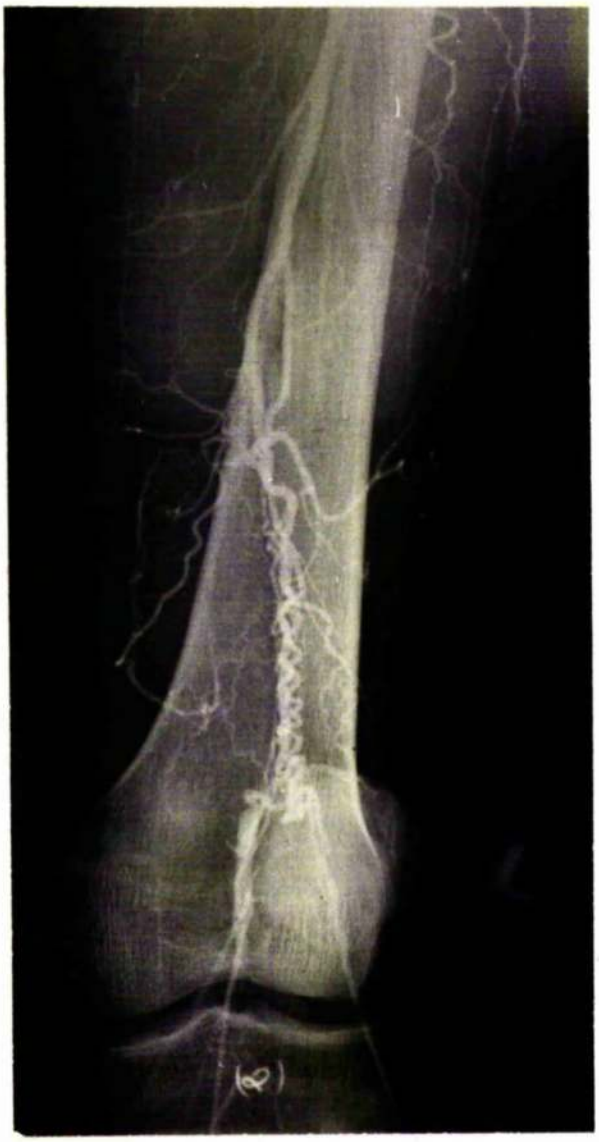


Fig. 71 : Tortuosity of Collateral Vessels.

D.R., aged 44 years, complained of left sided claudication at 50 yards in 1959 and was found to have double femoro-popliteal occlusion. There is a small patent segment of the popliteal artery above the knee joint and the artery was reconstituted at the popliteal bifurcation.

The leash of collateral vessels arising from the lower end of the profunda femoris illustrates the characteristic tortuosity of these vessels which appear to form a spiral in this arteriogram.

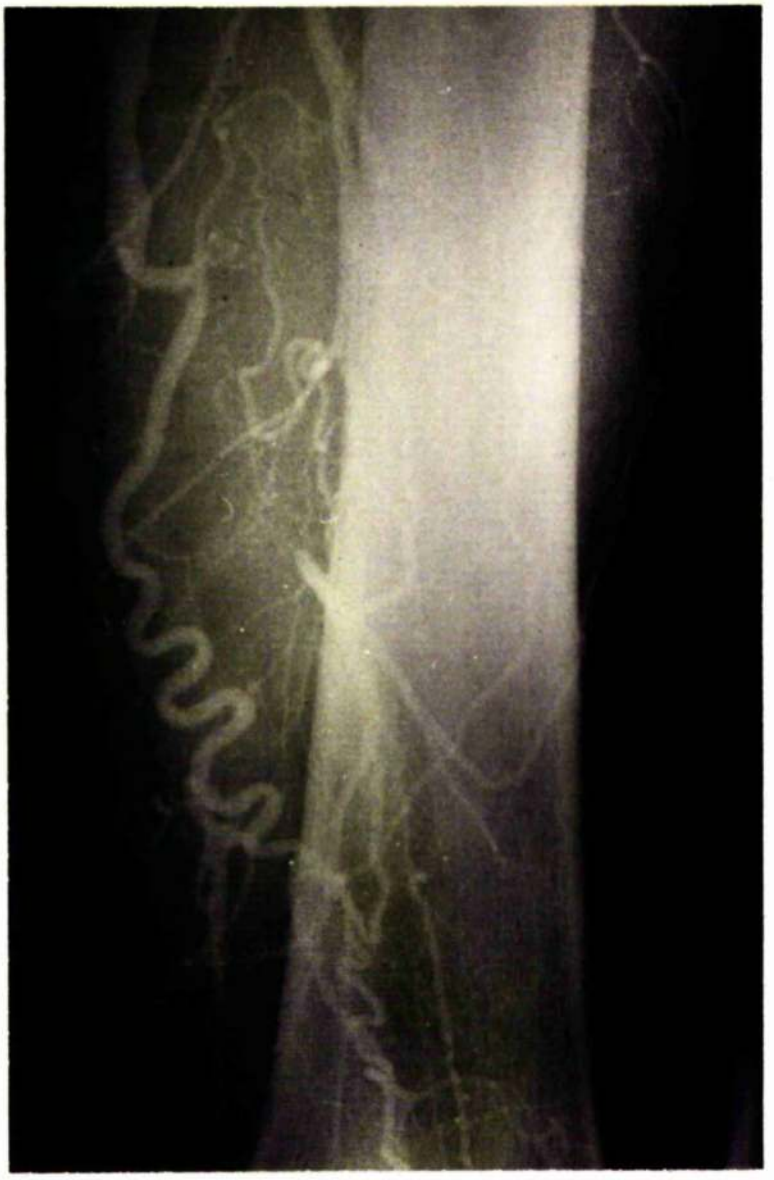


Fig. 72 : Tortuosity of Collateral Vessels.

In this arteriogram, a large tortuous limiting collateral vessel arises from the lower end of the patent superficial femoral artery but no reconstitution of the popliteal artery can be seen. A branch of the popliteal artery overlies the shadow of the femur and is reconstituted from the profunda femoris although its origin has been occluded.

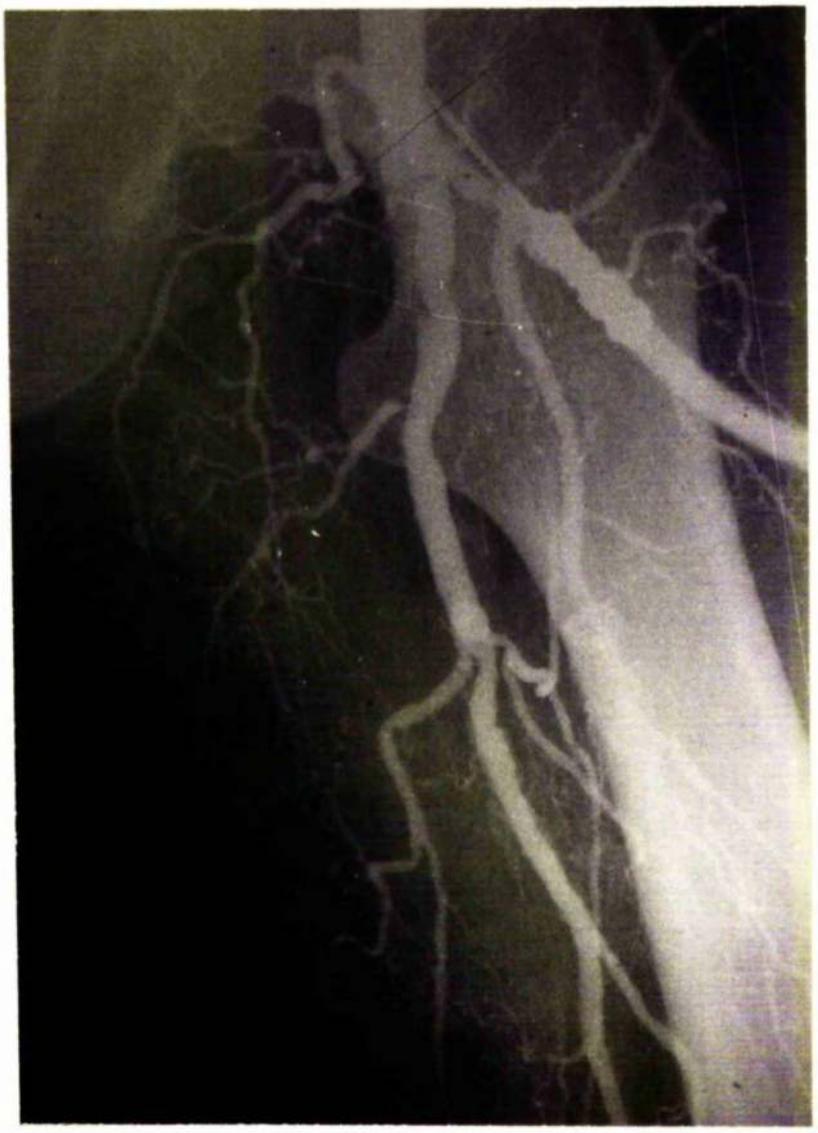


Fig. 73 : Atherosclerosis of the Profunda Femoris Artery.

This elderly patient, S.L., aged 70 years, developed a total femoro-popliteal occlusion with gangrene of the foot necessitating mid-thigh amputation.

The arteriogram shows the common femoral artery pierced by the needle and the superficial femoral artery is occluded. The profunda femoris shows atherosclerotic changes without occlusion, but the narrowing of the main trunk and of the origins of the branches has considerably reduced the collateral flow through the profunda femoris.

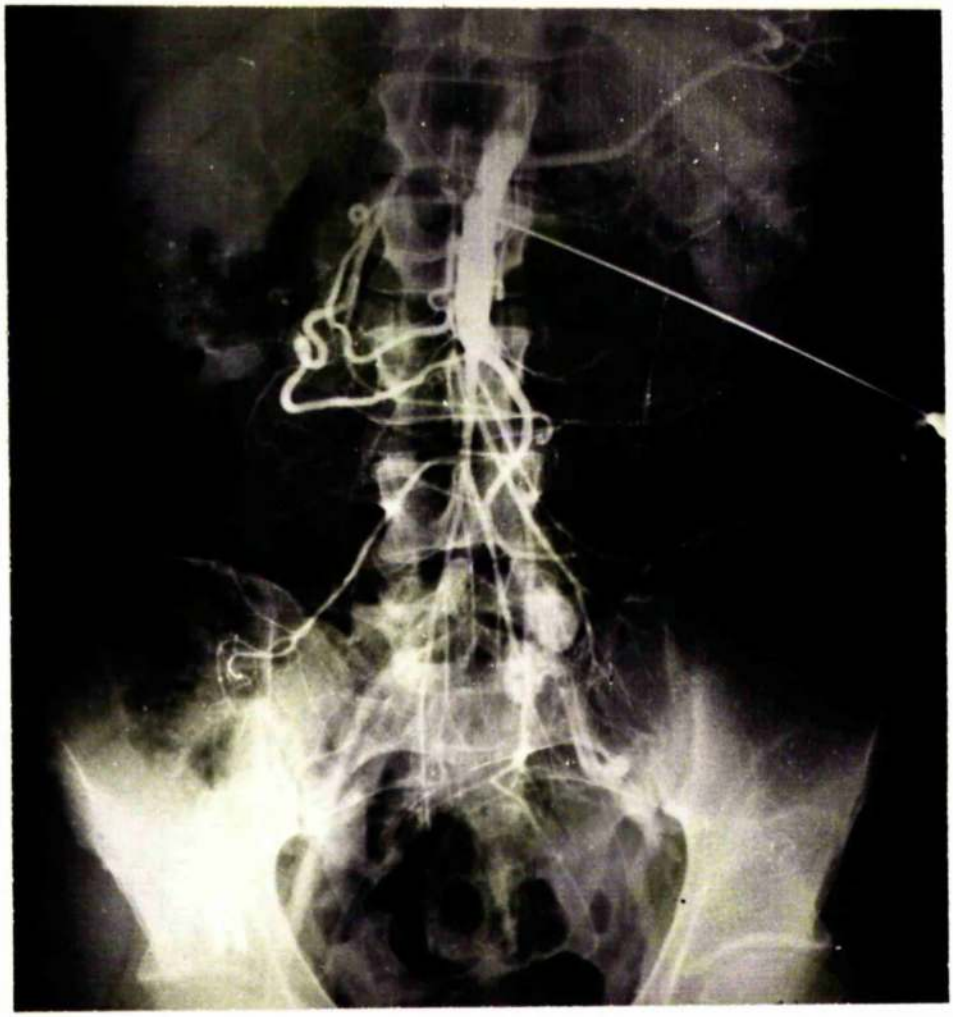


Fig. 74 : Occlusion of the Abdominal Aorta.

This aortogram shows occlusion of the aorta below the inferior mesenteric artery and reconstitution of both iliac arteries can be faintly seen.

This patient, a young male diabetic of 37 years, complained of bilateral claudication at 300 yards and, after phenol injection of the lumbar sympathetic chain, his claudication was relieved except for a slight ache on walking uphill.

The relative absence of symptoms produced by a severe occlusion of this type is probably due to the extensive collateral development possible in a young patient.

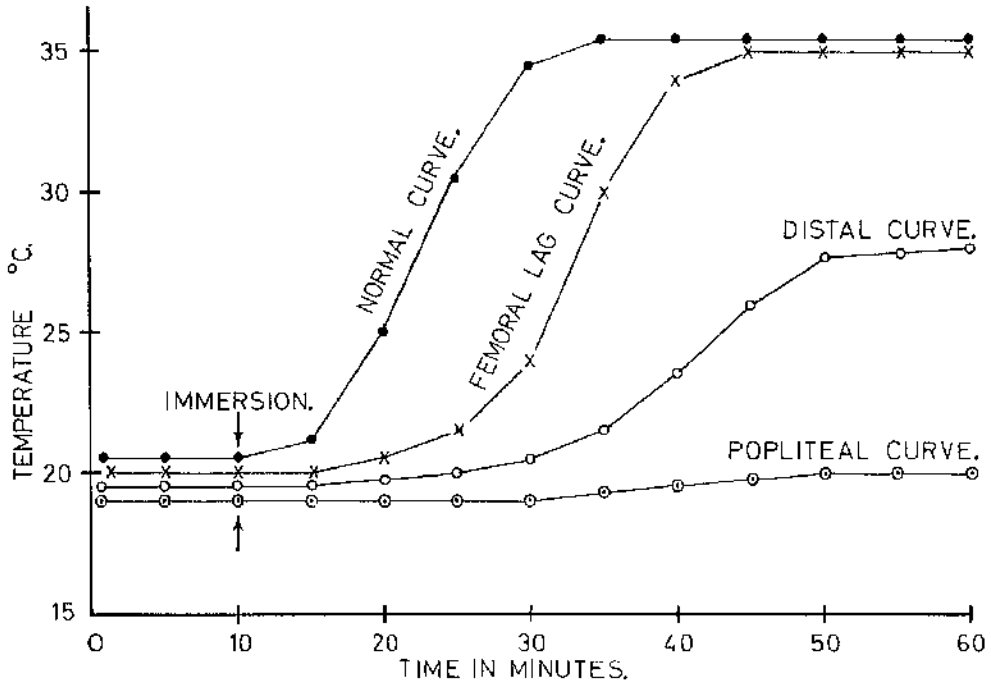


Fig. 75 : Composite Diagram of Vaso-dilation Curves.

Four common types of vaso-dilation curve are illustrated.

In the "normal" curve, the rise in temperature recorded from the great toe is rapid and soon reaches maximum levels.

In the "femoral" leg curve, typical of proximal superficial femoral occlusions and short adductor occlusions, the rise is rapid but delayed and the curve reaches maximum levels eventually.

In the "distal" curve typical of long femoro-popliteal occlusions and those with poor collaterals, the rise is delayed and incomplete.

In the "popliteal" curve, so-called because of its frequent association with extensive popliteal occlusion threatening viability, there is virtually no response to reflex heating indicating that the available collateral circulation is poor.

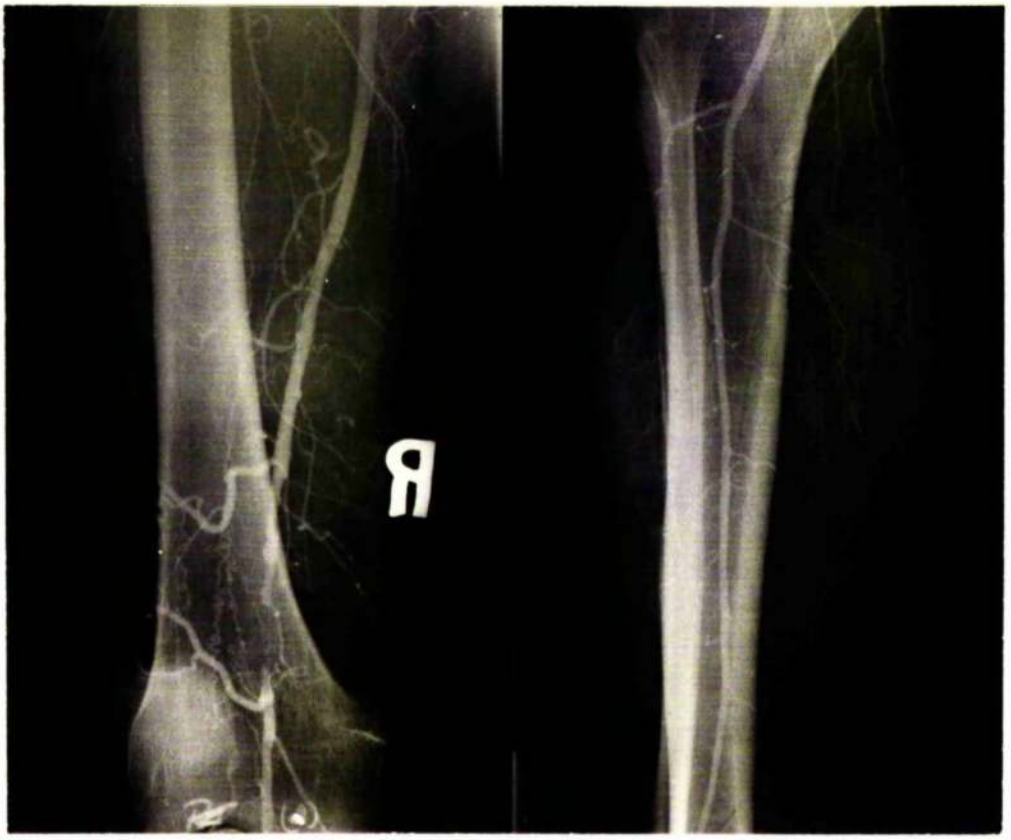


Fig. 76 : Collateral Vessels in a Short Popliteal Occlusion : Posterior Tibial Occlusion.

P.D., aged 58 years, developed right sided claudication in 1954 following a coronary artery thrombosis. When seen in 1959, he experienced claudication at 300 yards and, after phenol injection of the right lumbar sympathetic chain, he was halted by anginal pain at ¼ mile.

The arteriogram shows a short right popliteal occlusion with large proximal and re-entrant collaterals joined by a network of small tortuous vessels. The limiting collateral is small and proximal extension of occlusion to the large collateral at the level of the marker appears possible.

In the lower leg, a large peroneal artery and a small anterior tibial artery are visualised. The posterior tibial artery is occluded.

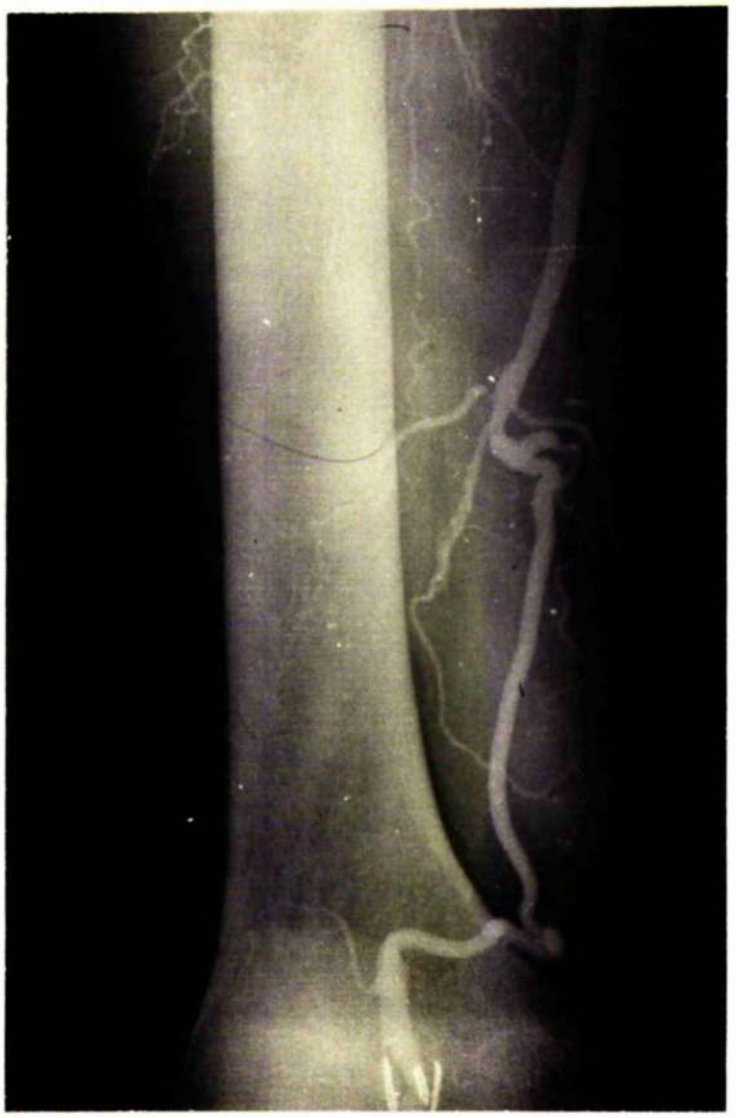


Fig. 77 : The Natural Development of Large Collateral Channels.

J.F., aged 68 years, was seen in 1957 when he complained of claudication of the right leg at 100 yards of 8 years duration.

The arteriogram shows an occlusion of the popliteal artery with a large collateral channel bridging the gap.

In spite of this apparently adequate collateral channel, he obtained only minimal improvement after phenol injection of the right lumbar sympathetic chain.

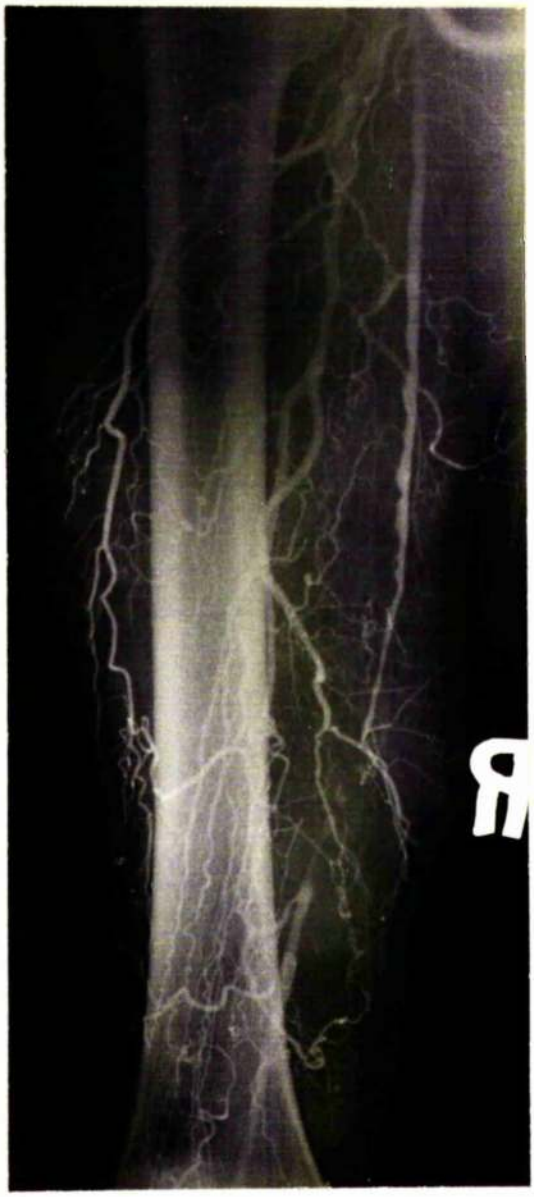
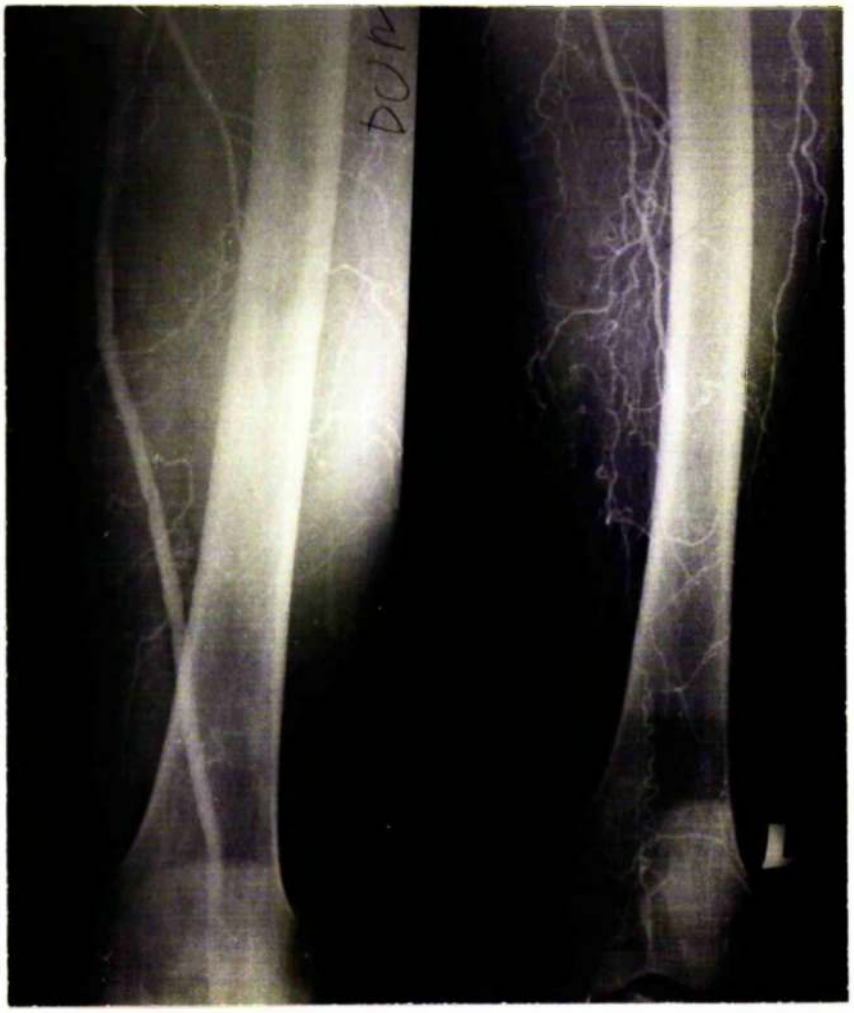


Fig. 78: The Collateral Circulation in Adductor Occlusion.

This arteriogram shows a short adductor occlusion with few collaterals arising from a narrowed irregular superficial femoral artery. Collaterals derived from the profunda femoris are numerous and numerous longitudinal collaterals which are superimposed on the femur travel a considerable distance to establish distal connections.

The descending branch of the lateral femoral circumflex (artery to vastus lateralis : the 'rectus femoris collateral') is prominent.



1957. 1960.

Fig. 79 : The Development of Total Femoro-Popliteal Occlusion.

W.D., aged 67 years, developed claudication of his right leg in 1957 and arteriography of the left leg (25.6.57) showed mild atherosclerotic changes (illustrated on the left).

In 1960, he developed rest pain in his left foot and on arteriography (14.6.60) was found to have developed a left total femoro-popliteal occlusion with no reconstitution of the tibial arteries.

Amputation of the left leg was performed on 7.7.60 and he died from coronary artery thrombosis on 12.10.60.

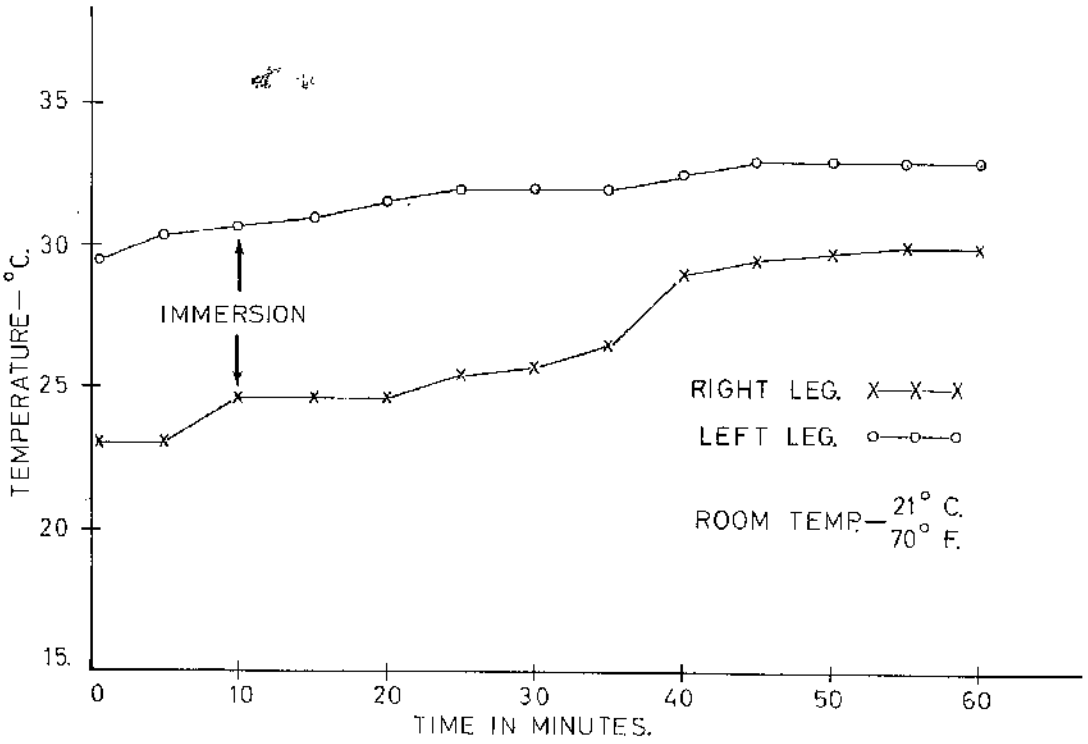


Fig. 80 : The Reflex Vaso-Dilation Curve after Sympathectomy.

Both legs show a slight rise in temperature before immersion.

The curve of the left leg starts high and shows relatively little rise indicating that existing vaso-dilation is almost maximal due to previous lumbar sympathectomy.

The curve of the right leg shows a delayed and incomplete rise ("distal" type of curve - Fig.75) due to a long femoro-popliteal occlusion.

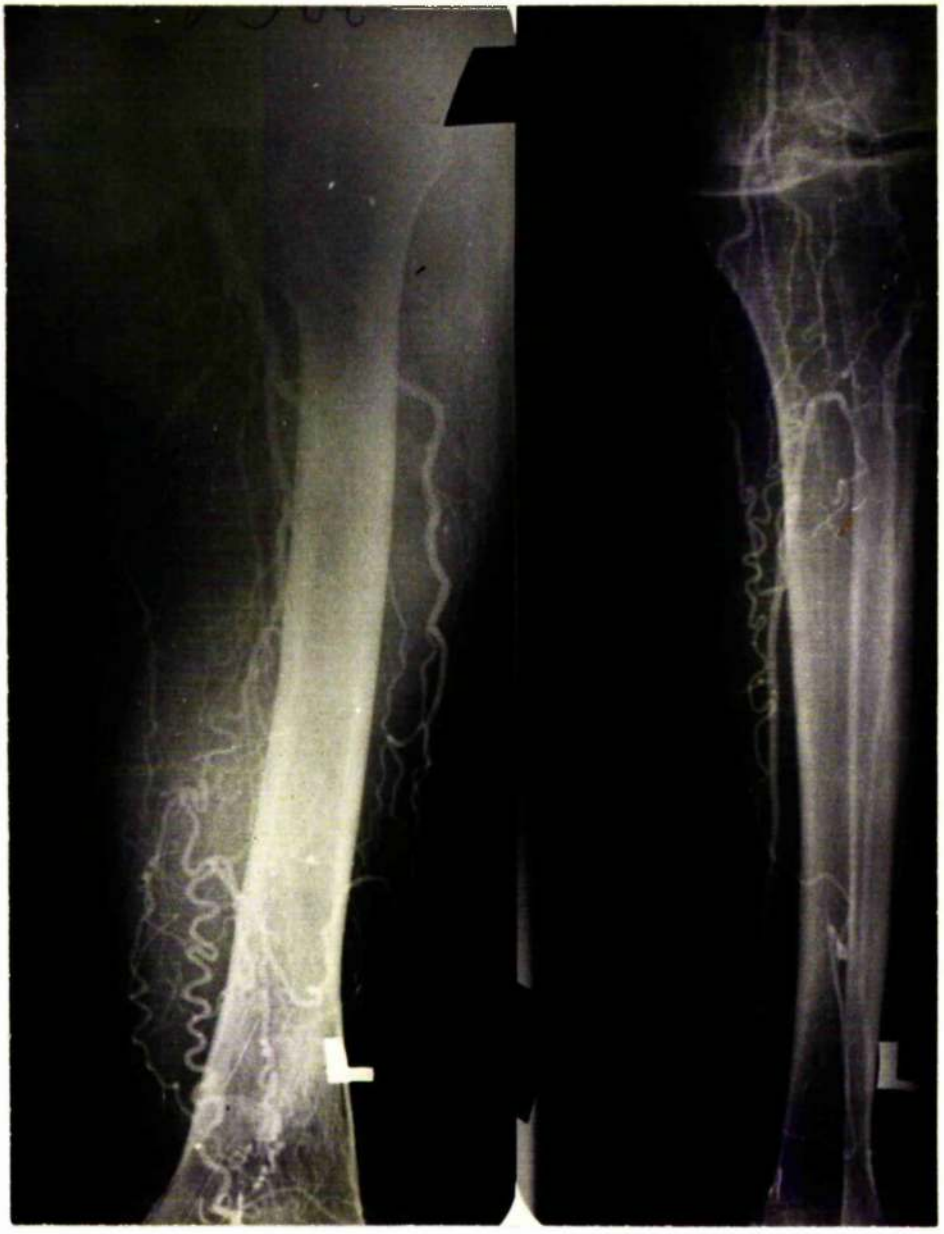


Fig. 81 : Collateral Development after Sympathectomy.

This left femoral arteriogram was performed three years after lumbar sympathectomy and shows a total femoro-popliteal occlusion with an extensive network of collateral vessels. Reconstitution of the posterior tibial artery is excellent but there is occlusion of the upper part of the peroneal artery and the proximal part of the anterior tibial artery.

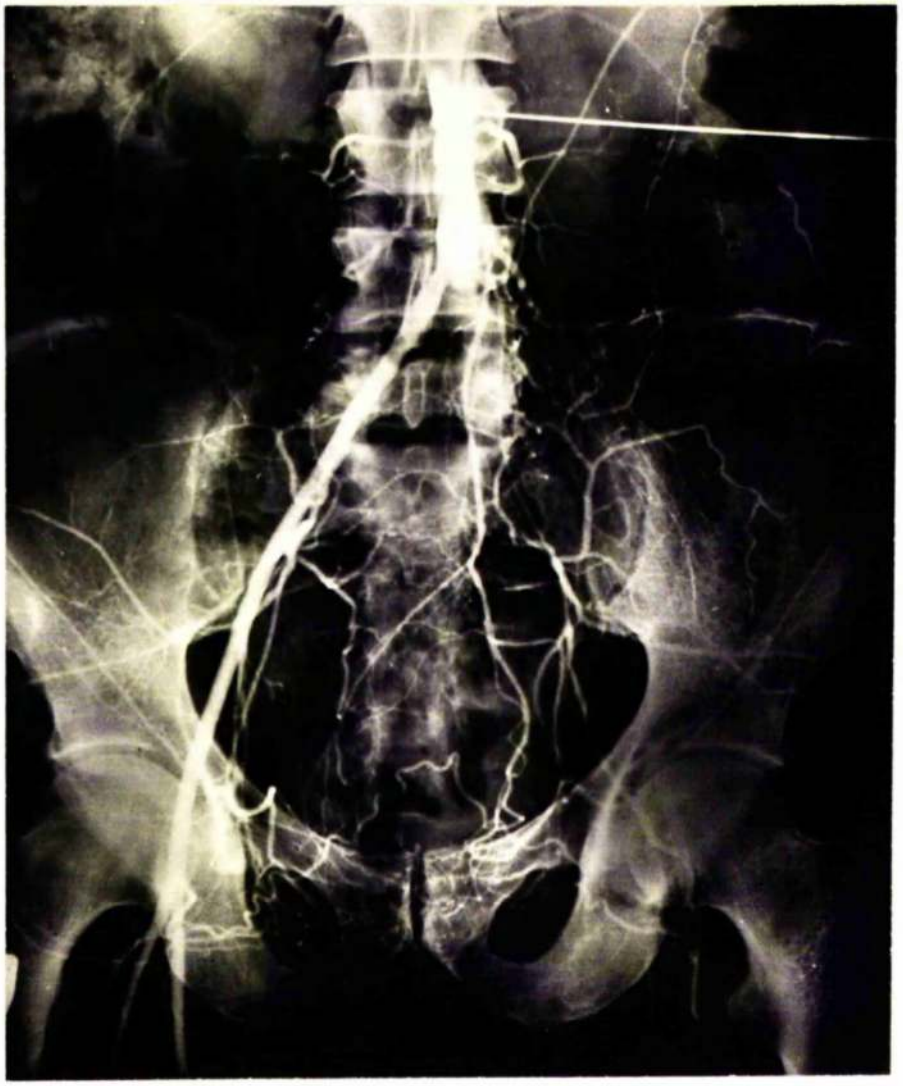


Fig. 82 : Occlusion of the Left Common Iliac Artery.

J.C., a man of 46 years, was seen in 1960 with left sided claudication at 100 yards.

Aortography shows an occlusion of the left common iliac artery, the left external iliac artery being visible on the second film. This common iliac occlusion is presumed to have originated in the common iliac artery itself as there is evidence of a plaque in the middle of the right common iliac artery (at the level of the L. 4-5 disc space.)

A bypass teflon graft was successfully inserted on 6.6.60 from aorta to left external iliac artery but it subsequently thrombosed a few months later.

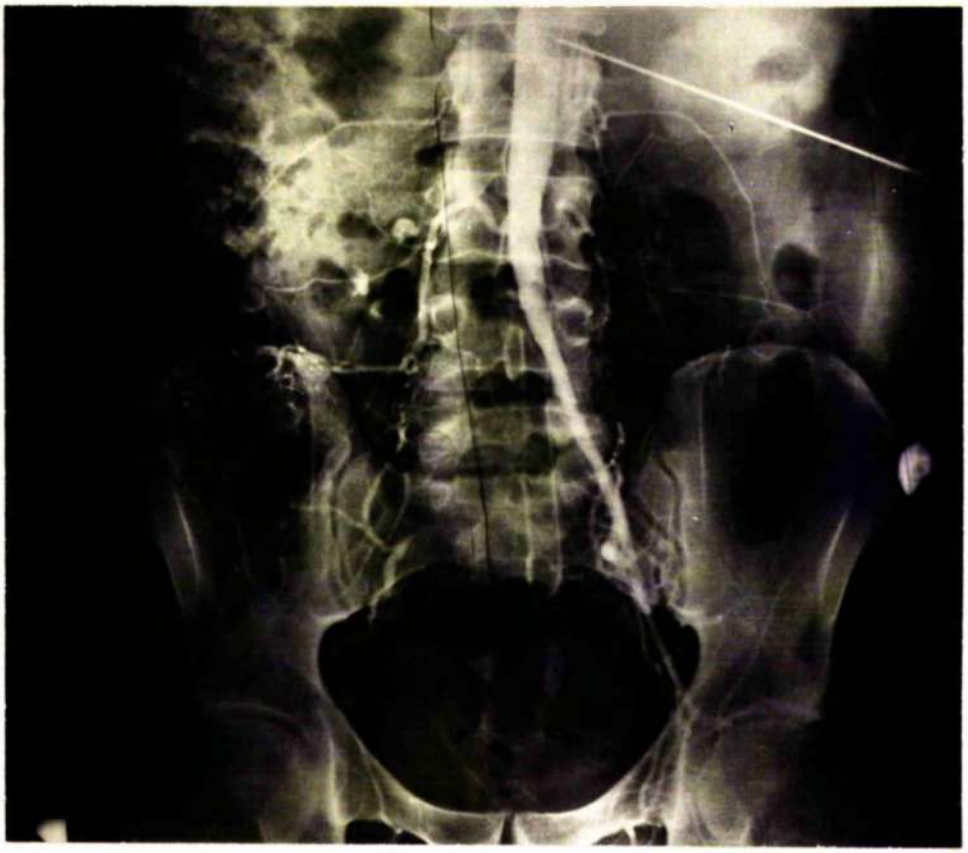


Fig. 83 : Occlusion of the Right Common and External Iliac Arteries : Left Common Femoral Occlusion.

This aortogram shows occlusion of the right common and external iliac arteries and the internal iliac artery is reconstituted through a large ilio-lumbar anastomosis. On the left side, the common femoral artery is occluded and there was an associated left femoro-popliteal occlusion. In view of these multiple lesions, no operative treatment was undertaken.

The left common iliac artery is of normal calibre at its origin and it is presumed that the right common and external iliac occlusion originated at the common iliac bifurcation.

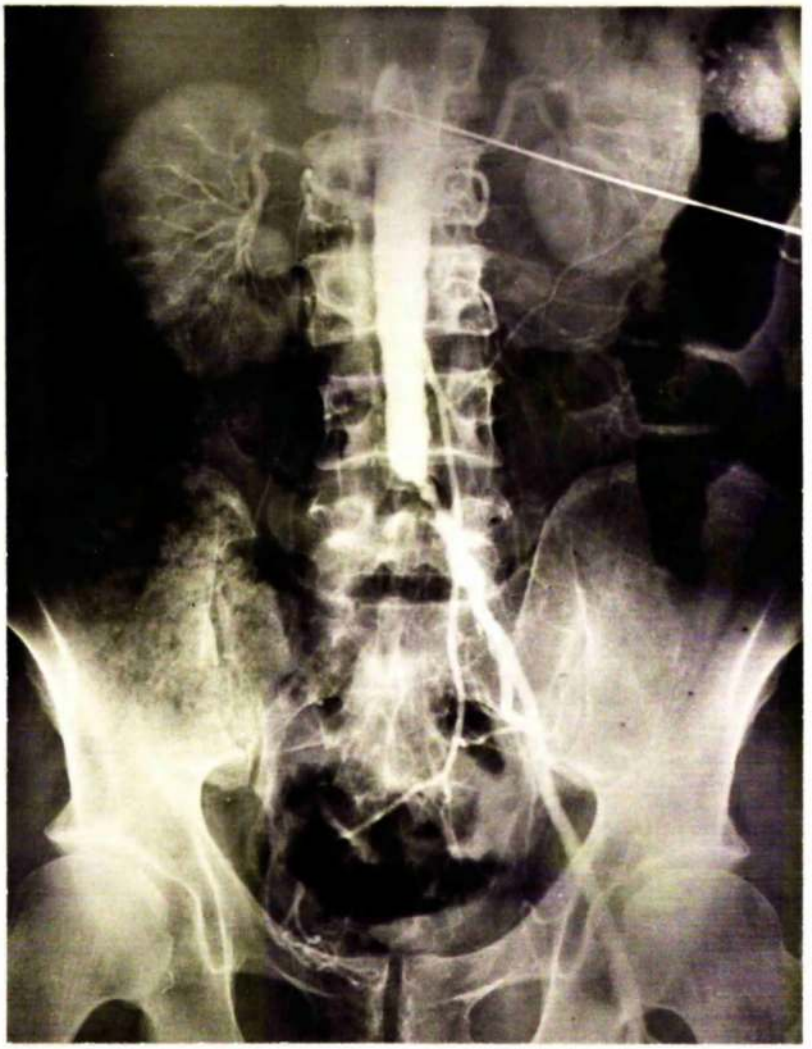


Fig. 84 : Occlusion of the Left Common Iliac Artery.

H.G., aged 60 years, developed right sided claudication at 50 yards in 1957. Aortography shows occlusion of the right common iliac artery, the external iliac artery being visualised on the second film.

The occlusion is believed to have originated from a lesion at the aortic bifurcation as the left iliac artery is grossly diseased at its origin.

In 1961, he developed ischaemic rest pain in the left foot which was relieved by phenol injection and aortography showed that occlusion of the aorta to the level of the renal arteries had occurred. Operative treatment was not undertaken because of age and poor physique.

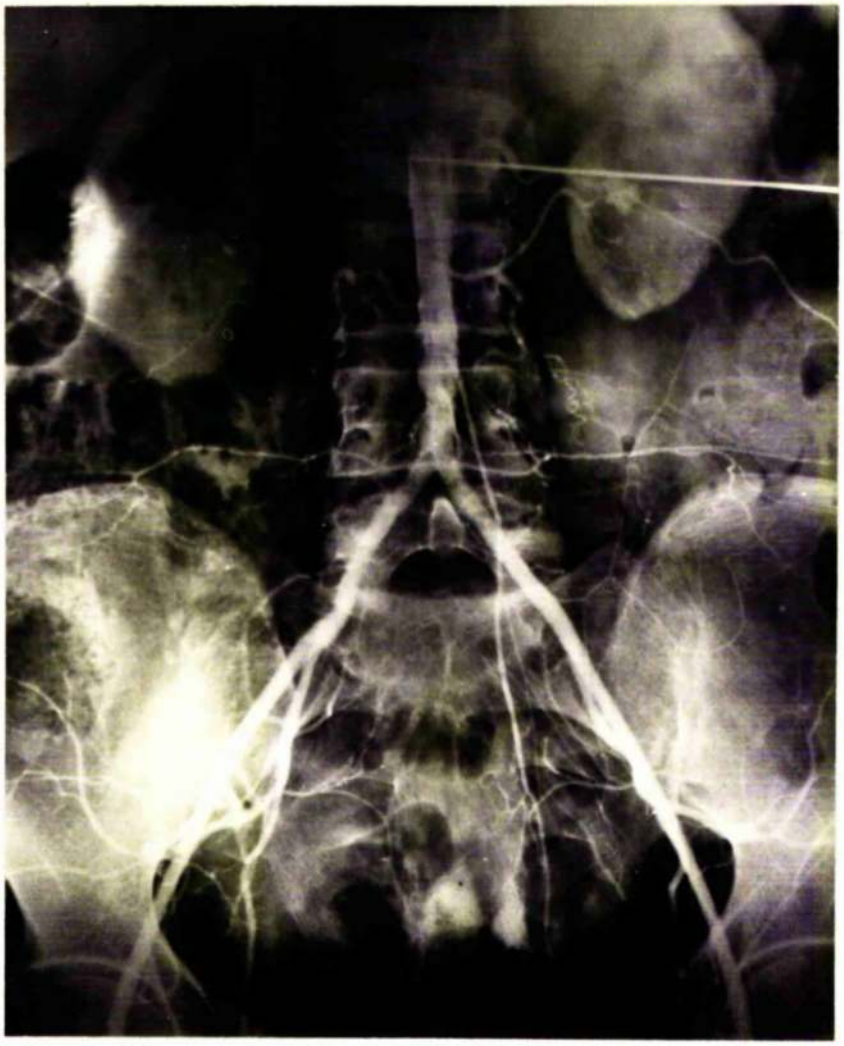


Fig. 85 : Incipient Aortic Occlusion.

L.H., a woman aged 60 years, complained of bilateral claudication at 100 yards distance in 1960.

Both femoral pulses were reduced in volume and aortography shows an atherosclerotic aorta which is narrowed above the bifurcation. Thrombo-endarterectomy was performed on 16.5.60 and severe stenosis of the aorta was found, more marked than the aortogram reveals. Since operation, her claudication has been abolished and she has remained well to date.

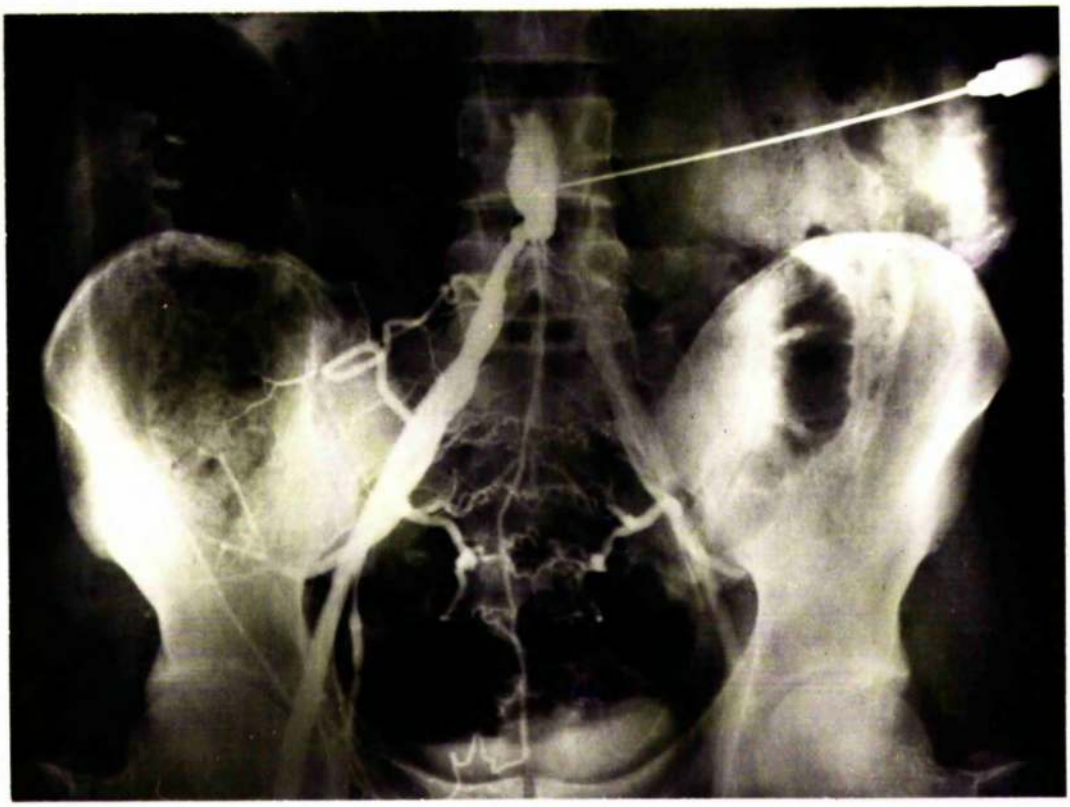


Fig. 86 : Occlusion of the Left Common Iliac Artery.

G.R., aged 39 years, complained of left sided claudication at 100 yards in 1961. The aortogram shows occlusion of the left common iliac artery due to a lesion arising at the aortic bifurcation.

A left aorto-iliac bypass teflon graft was successfully inserted but the right iliac artery thrombosed. Six months later, a second operation was abandoned owing to intense peri-aortic fibrosis which prevented mobilisation and the graft thrombosed 10 days later. He now has an aortic occlusion but is able to work as a clerk and his present claudication distance is 150 yards.

In this aortogram, the cross-midline anastomosis between the paired branches of the internal iliac arteries is beautifully demonstrated.

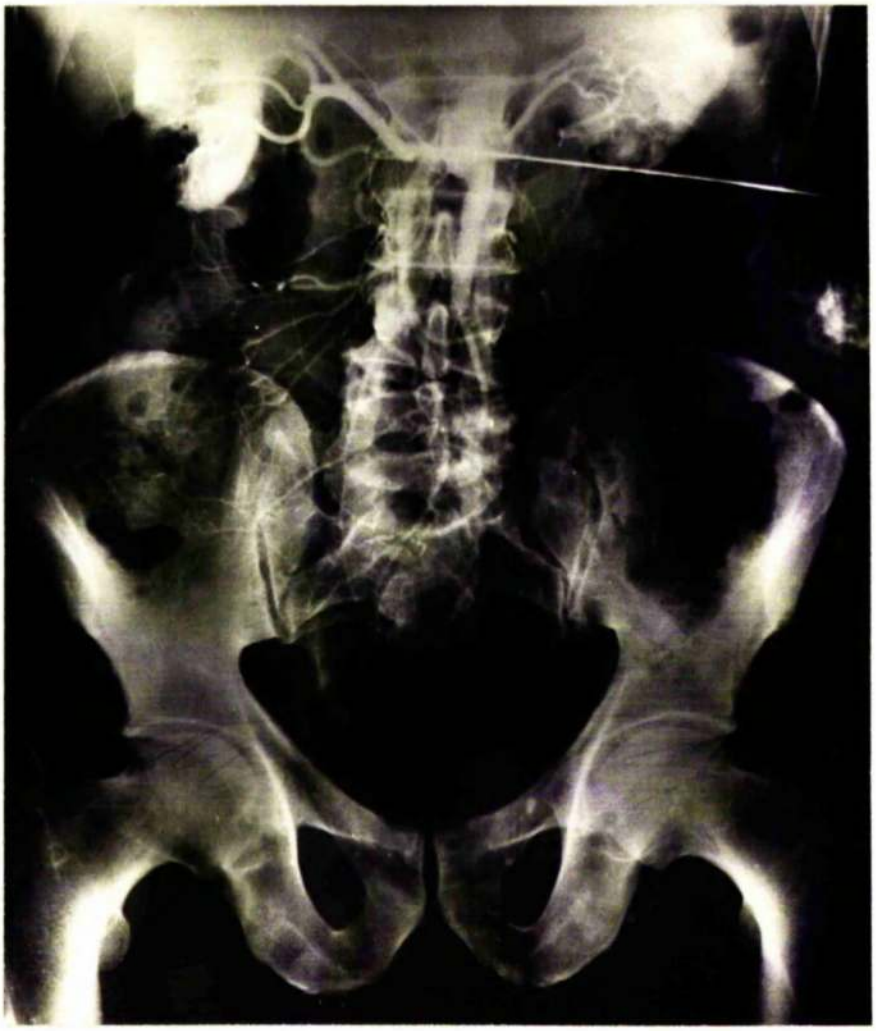


Fig. 87 : Aortic Occlusion to the Level of the Inferior Mesenteric Artery.

T.McC., aged 58 years, complained of left sided claudication at 100 yards in 1959. Both femoral pulses were absent.

Aortography shows that the aorta is occluded up to the level of the inferior mesenteric artery and is reduced in diameter above this level. No treatment was given and he is still working as a machineman.

A younger patient with a similar lesion in which the aorta was also grossly narrowed above the occlusion was successfully operated on in 1963.

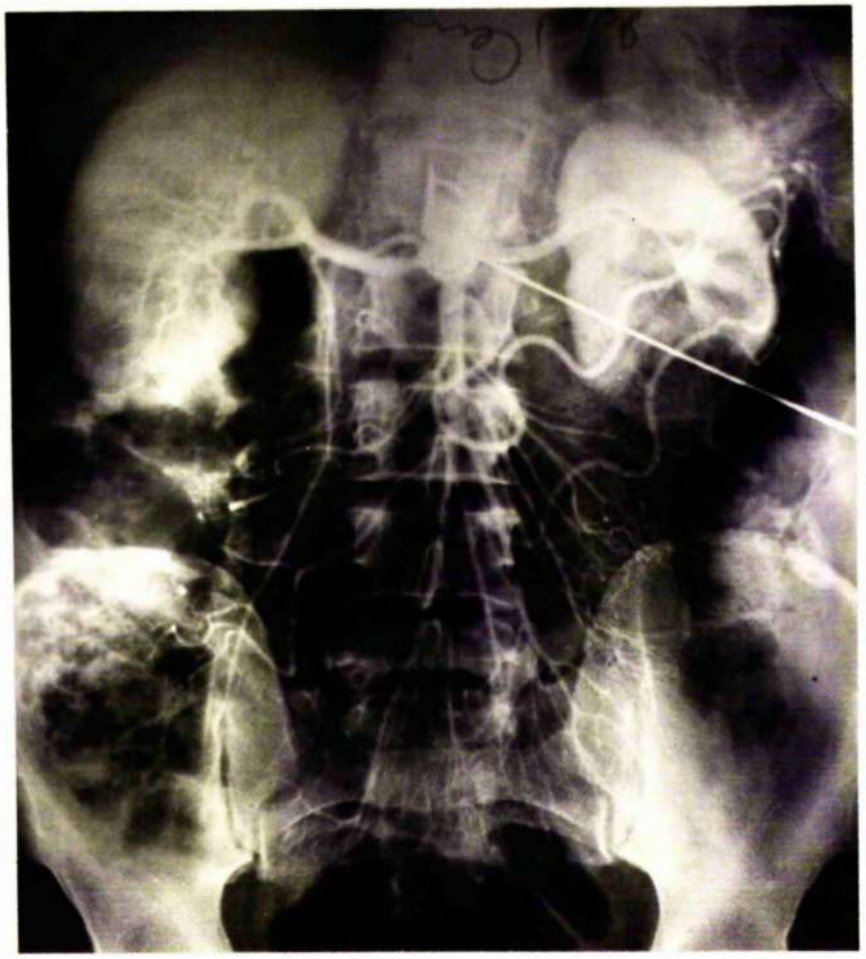


Fig. 88 : Aortic Occlusion to the Level of the Renal Arteries.

In this aortogram, the abdominal aorta is occluded up to the level of the renal arteries. The superior mesenteric artery is large and the middle colic artery is prominent as it courses to the left as the source of supply to a hypertrophied marginal artery supplying the colon.

No reconstitution of the iliac arteries was seen on the late film but, in spite of this extensive occlusion, the patient, J.C. aged 59 years, suffered from claudication only and both feet were healthy.

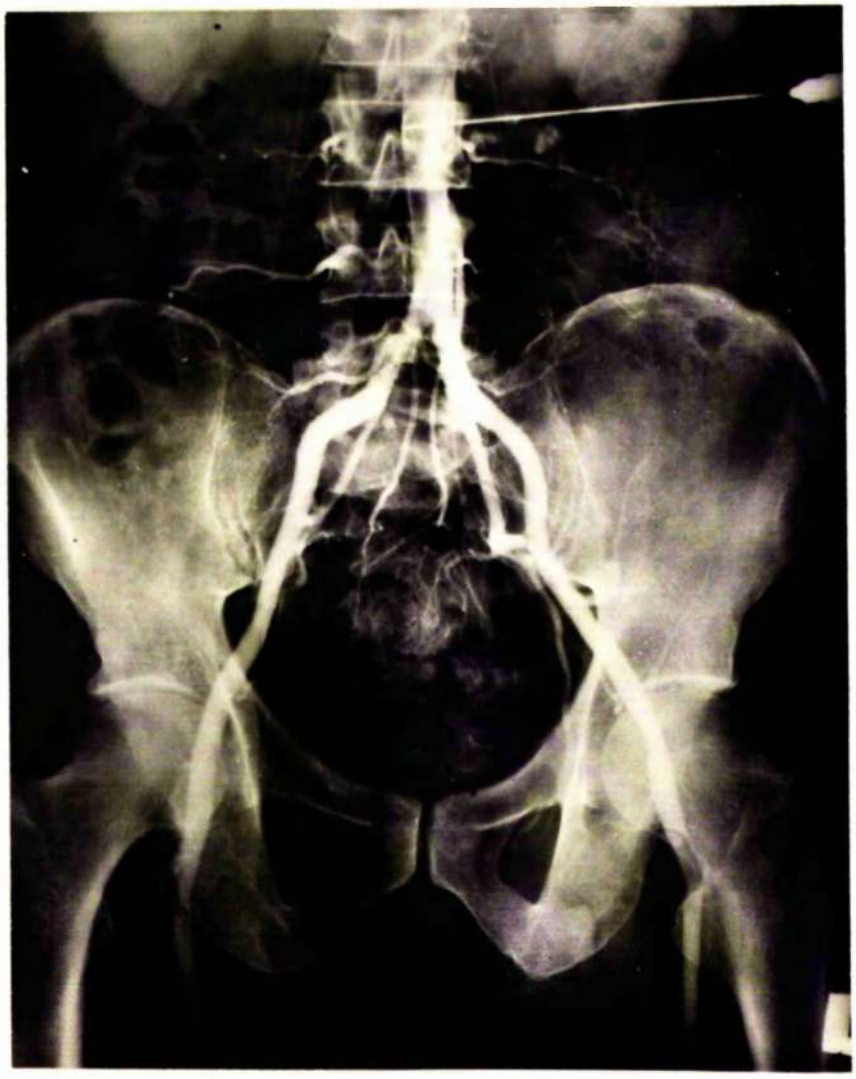


Fig. 89 : Stenosis of the Right Common Iliac Artery.

J.A., aged 51 years, complained of right sided claudication at 200 yards in 1962 and the right femoral pulse was weak.

In the aortogram, the density of the dye column in the right common iliac artery is poor. At operation (Fig.122), the lesion illustrated in Fig.33 was removed by thrombo-endarterectomy. The femoral and popliteal arteries were patent (Fig.4) and he has remained free of symptoms to date.

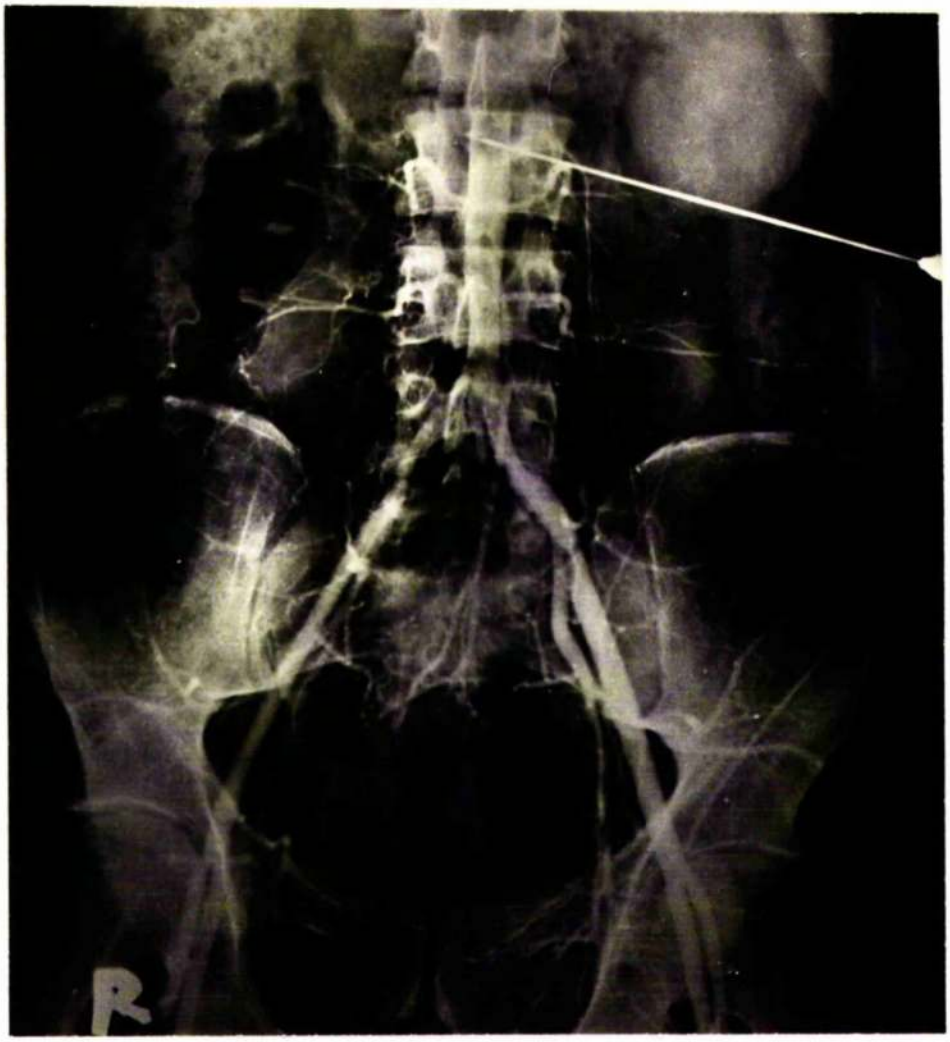
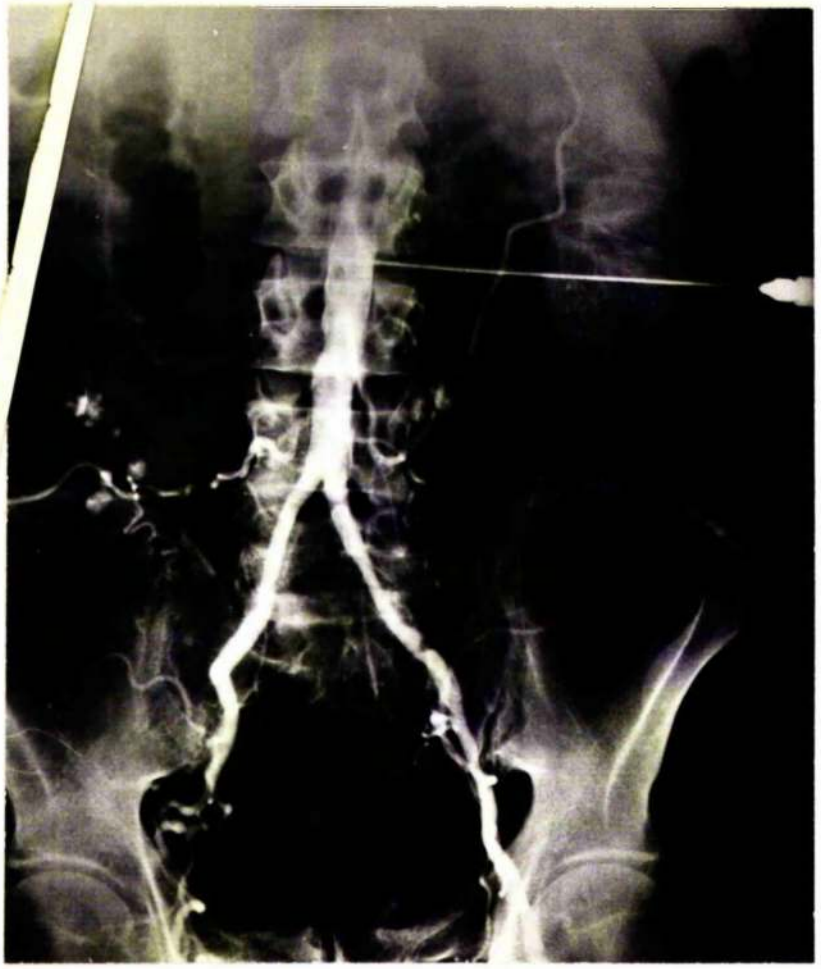


Fig. 90: Incipient Occlusion of the Right Common Iliac Artery.

J.McB., a young diabetic aged 30 years, developed right sided claudication at 200 yards in 1957.

Aortography shows an incomplete lesion of the right common iliac artery and there is a small niche in the medial wall of the left common iliac artery. Narrowing at the left common iliac bifurcation is also present.

Operation was not undertaken in this patient as he was able to walk 2 miles at a fast pace after phenol injection of the right lumbar sympathetic chain and he has remained well to date.



F . 91 : Occlusion of the Right External Iliac Artery : Left Common Iliac Bifurcation Stenosis.

This aortogram shows occlusion of the right external iliac artery and a collateral circulation has been developed through the obturator artery.

A lesion producing stenosis can be seen on the left side and careful scrutiny of the superimposed external and internal iliac arteries suggests that the lesion is arising at the common iliac bifurcation.

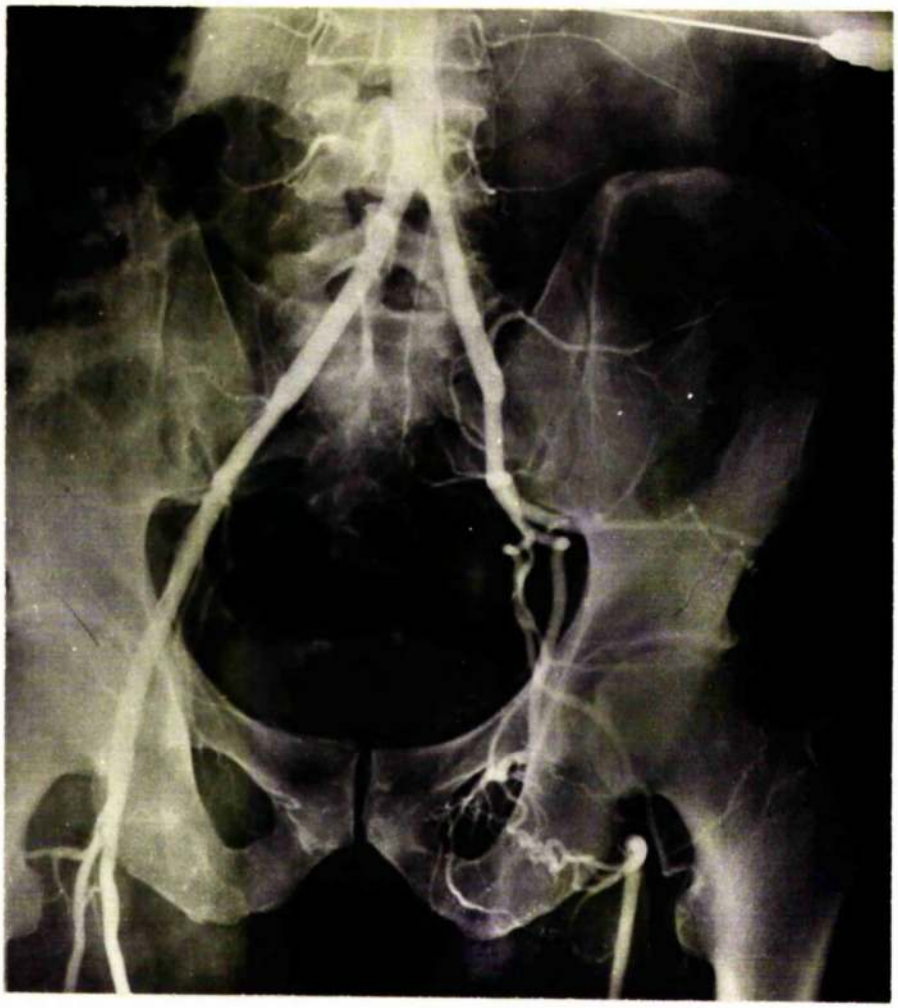


Fig. 92 : Occlusion of the Left External Iliac Artery.

This aortogram illustrates an occlusion of the left external iliac artery which also involves the left common femoral artery. The left internal iliac is hypertrophied and anastomoses through its obturator branch with the medial femoral circumflex artery which reconstitutes the superficial femoral artery. The left profunda femoris is not well visualised.

The right internal iliac artery is small and a notch on the lateral wall of the common iliac artery can be seen opposite its origin.

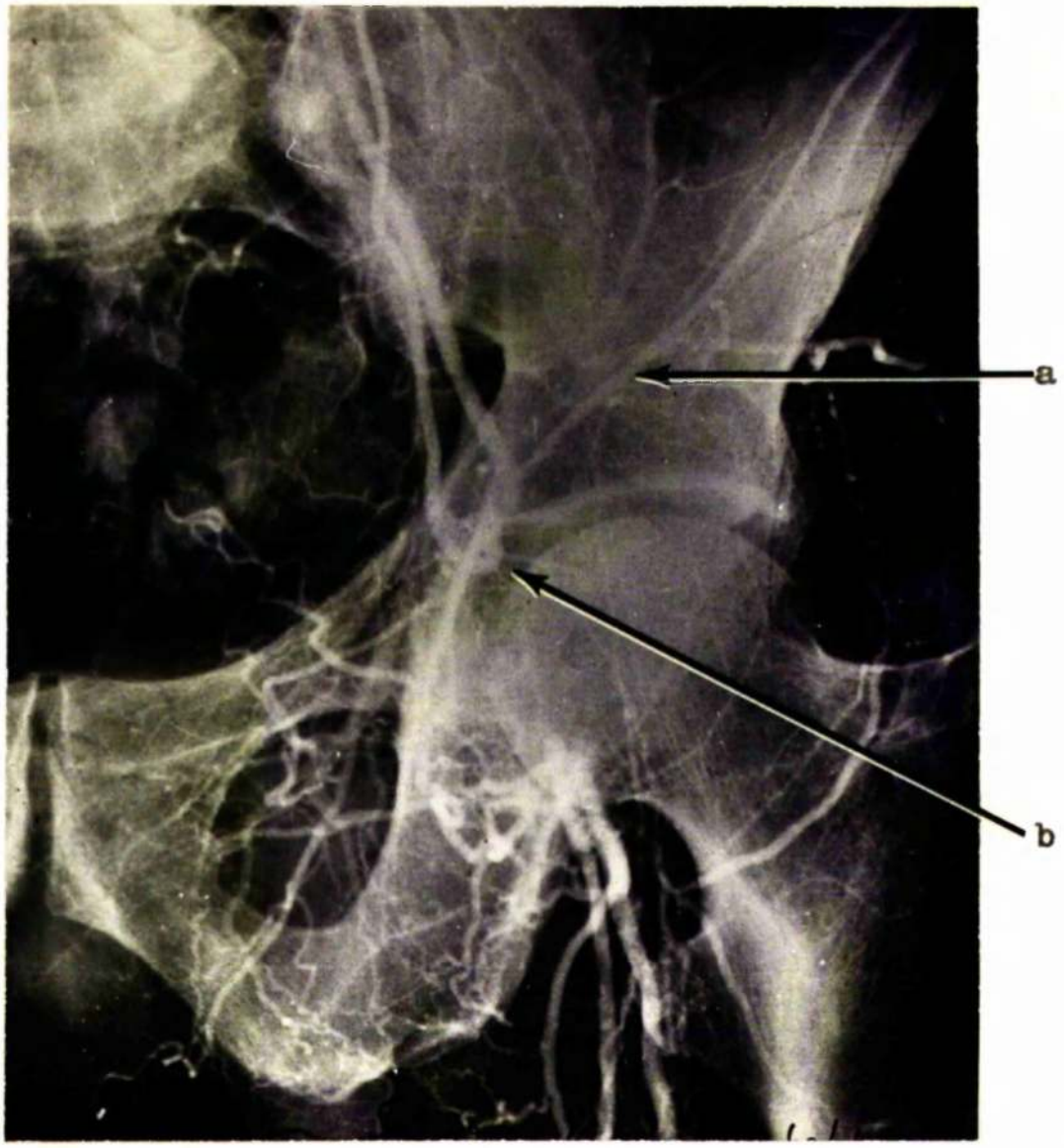


Fig. 93 : Occlusion of the Left Common Femoral Artery.

In this aortogram, the left common femoral artery is occluded and the superficial and deep femoral arteries are separate. Occlusion extends up to the origin from the external iliac artery of the deep circumflex iliac artery (a) and the inferior epigastric artery (b) which forms a U before passing upwards and medially.

Numerous collateral vessels surround the obturator foramen but most of the dye has left the obturator artery which is not easily distinguishable.

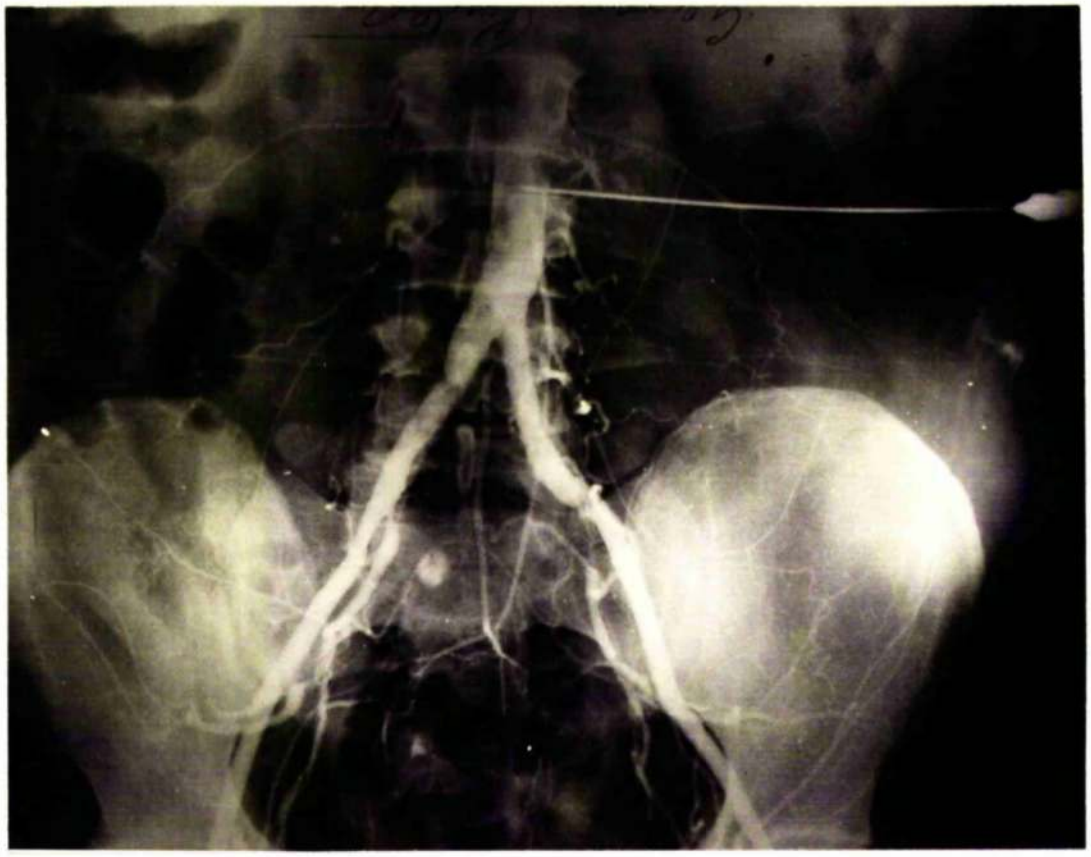


Fig. 94 : Occlusion of the Internal Iliac Artery.

On the left side, the proximal 2 cms. of the internal iliac artery are occluded and the reduced density of the dye shadow at the common iliac bifurcation indicates a plaque which is the probable source of the occlusion.

On the right side, the internal iliac artery is narrowed at its origin and eventual occlusion appears probable.

A notch in the lateral wall of the right common iliac artery and a less intense dye shadow at this level indicates the presence of an atherosclerotic plaque.

This patient complained of bilateral buttock claudication only and all the pulses were palpable in both legs.

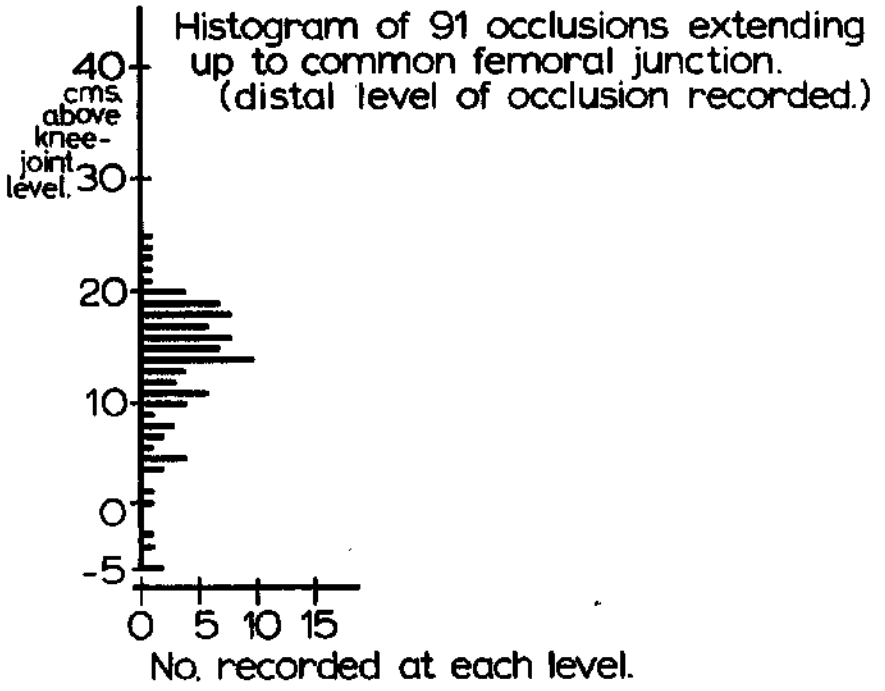


Fig. 95 : Distribution of the Lower Level of Occlusion in 91 Occlusions extending to the Common Femoral Junction.

In this histogram, the lower level of the 91 femoro-popliteal occlusions extending upwards to the level of the common femoral junction is recorded.

The majority extend from the adductor region (14-20 cms.) but many others involve the upper part of the popliteal artery (4-13 cms.) and two reach the popliteal bifurcation (-5cms.).

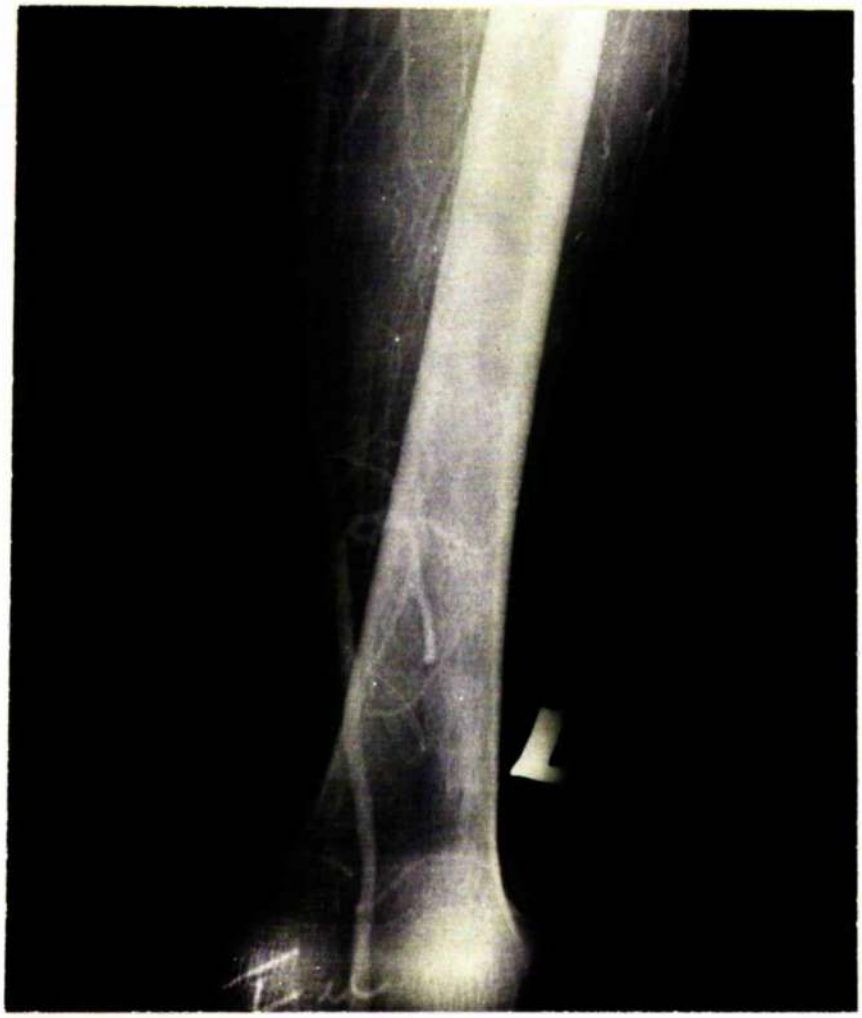


Fig. 96 : Incipient Occlusion at the Adductor Opening.

In this arteriogram, severe stenosis at the level of the adductor opening is seen and the descending genicular branch arises just below the lesion.

At operation, recent thrombosis was found to have occurred proximal to this level (Fig.97) and thrombo-endarterectomy was performed through a long arteriotomy (Fig.126). Unfortunately, the artery thrombosed a few weeks later. Further arteriography showed a long femoro-popliteal occlusion but his present claudication distance (200 yards) is no worse than before operation.

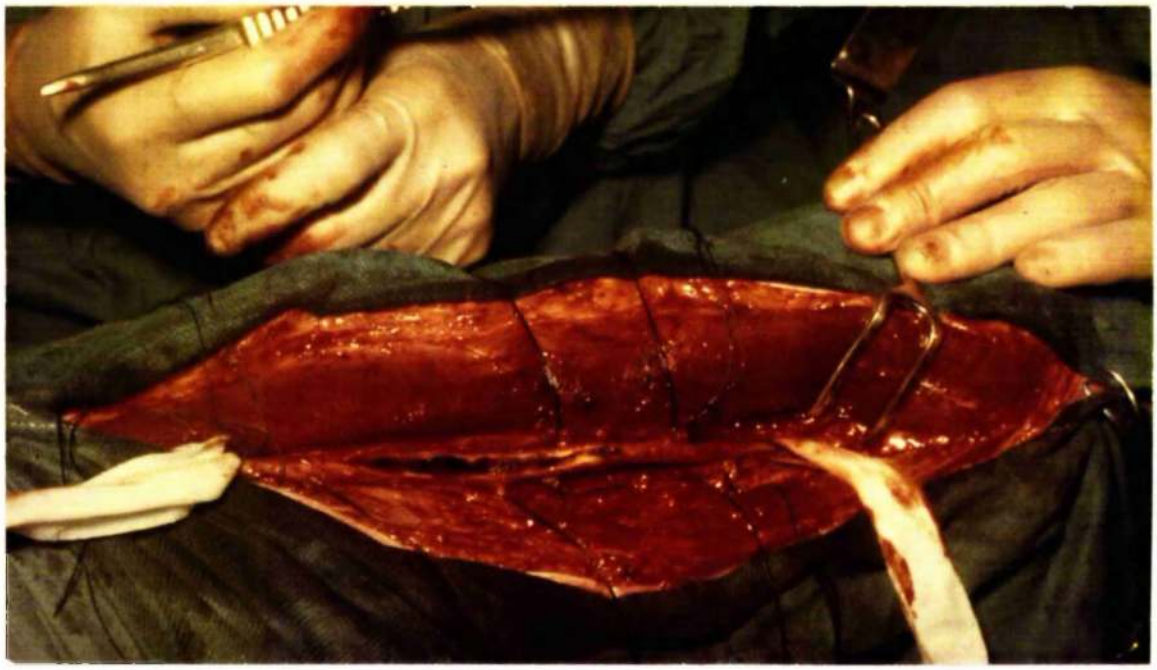


Fig. 97 : Recent Thrombosis in the Superficial Femoral Artery.

The left superficial femoral artery has been exposed by a long oblique incision in the thigh. The collateral vessels have been dissected free and are indicated by the black silk loops passed round them. The lesion illustrated in Fig.96 is seen as a firm whitish plaque near the distal end of the arterial incision and recent thrombus is seen to lie proximal to the lesion. This thrombus was not adherent to the vessel wall and was easily extracted from the upper part of the artery.

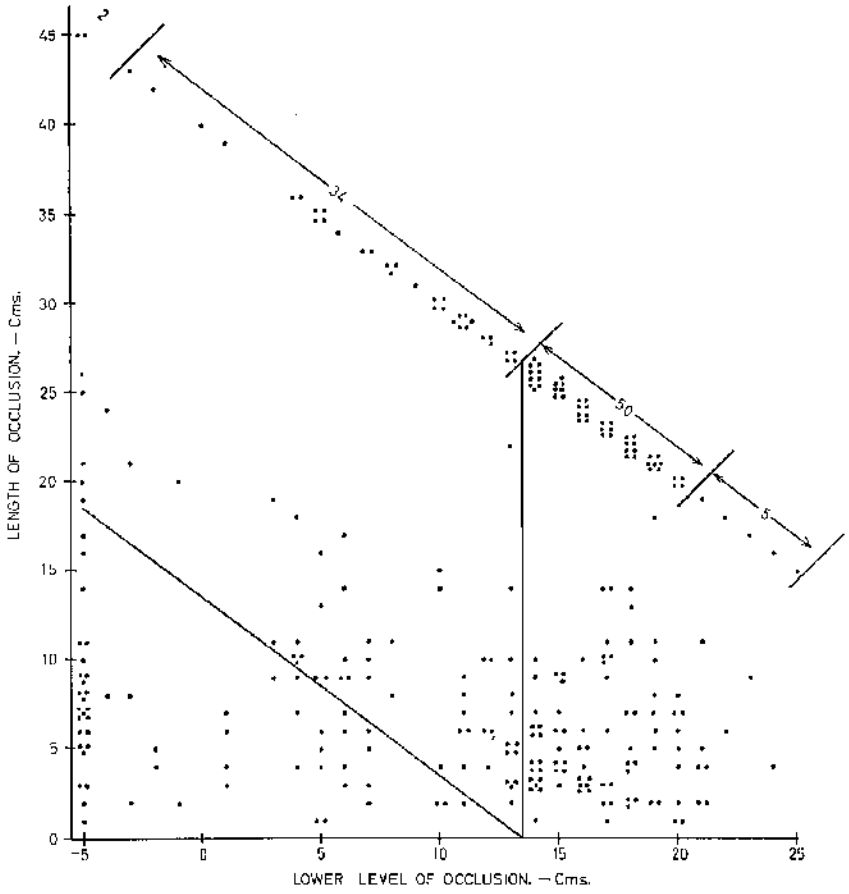


Fig. 98 : Diagrammatic Representation of 295 Femoro-Popliteal Occlusions.

Each occlusion is represented in this diagram by a dot placed according to its length and the lower level of occlusion. If the division between the femoral and popliteal arteries is taken as 13-14 cms., occlusions to the right of the vertical line affect only the superficial femoral artery.

The oblique line is drawn from a point between 18 and 19 cms. on the ordinate (i.e. 13-14 cms. above knee joint level) to the base of the vertical line, and the occlusions below the oblique line involve only the popliteal artery.

Occlusions above the oblique line and to the left of the vertical line affect portions of both arteries i.e. they are femoro-popliteal occlusions.

The tendency for the observations to aggregate in groups permits the identification and classification of certain common types of occlusion.

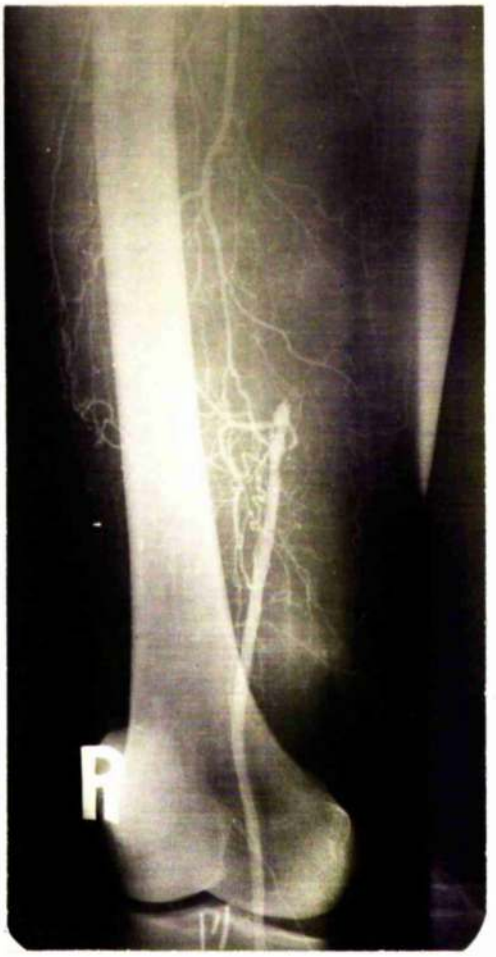


Fig. 99 : Long Superficial Femoral Occlusion.

In this arteriogram, most of the superficial femoral artery is occluded and reconstitution occurs through collateral vessels anastomosing with the profunda femoris. The popliteal artery is relatively healthy.

The lower level of occlusion is at 18 cms. above the knee joint level and the length of the occlusion (to the common femoral junction) is approximately 22 cms. i.e. it is classified as a long superficial femoral occlusion.

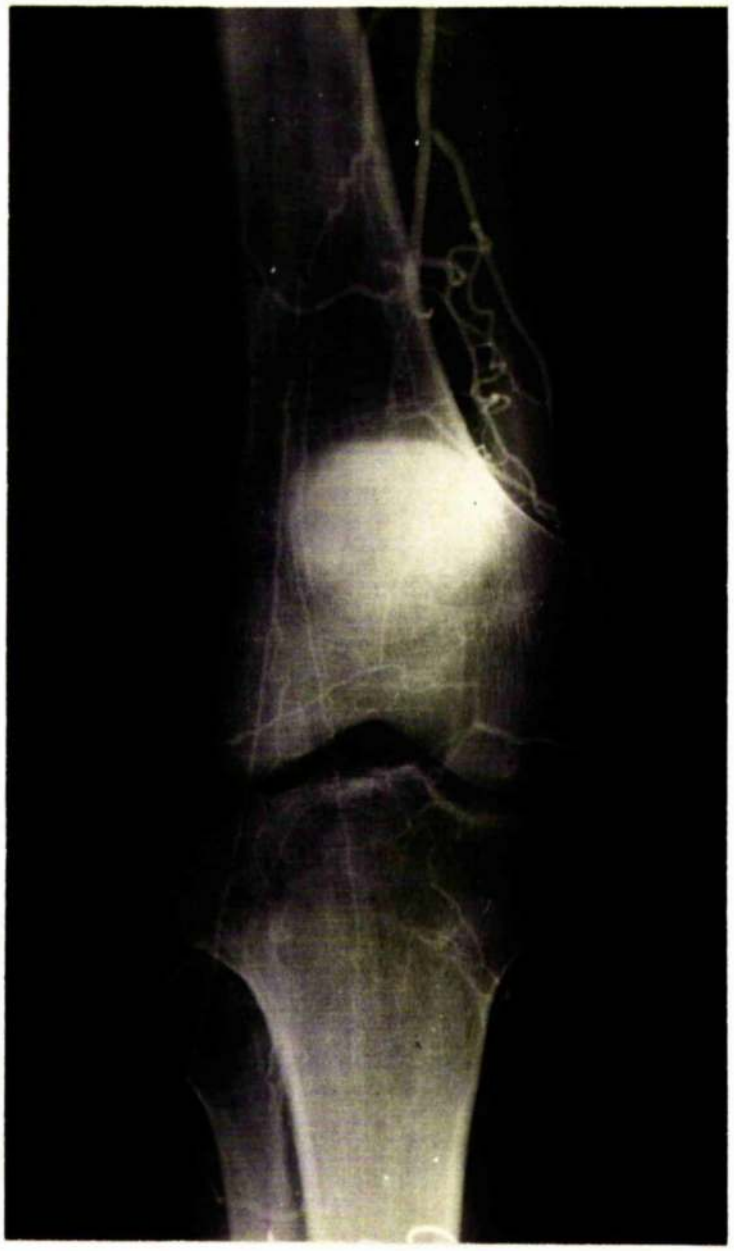


Fig. 100 : Total Popliteal Occlusion.

G.K., aged 52 years, complained of bilateral claudication in 1960. The left femoral pulse was absent and aortography showed occlusion of the left common femoral artery (Fig.93).

The film of the right leg illustrated here shows an occlusion of most of the popliteal artery. The saphenous artery and other collateral vessels course downwards but no reconstitution of the popliteal artery is visible.

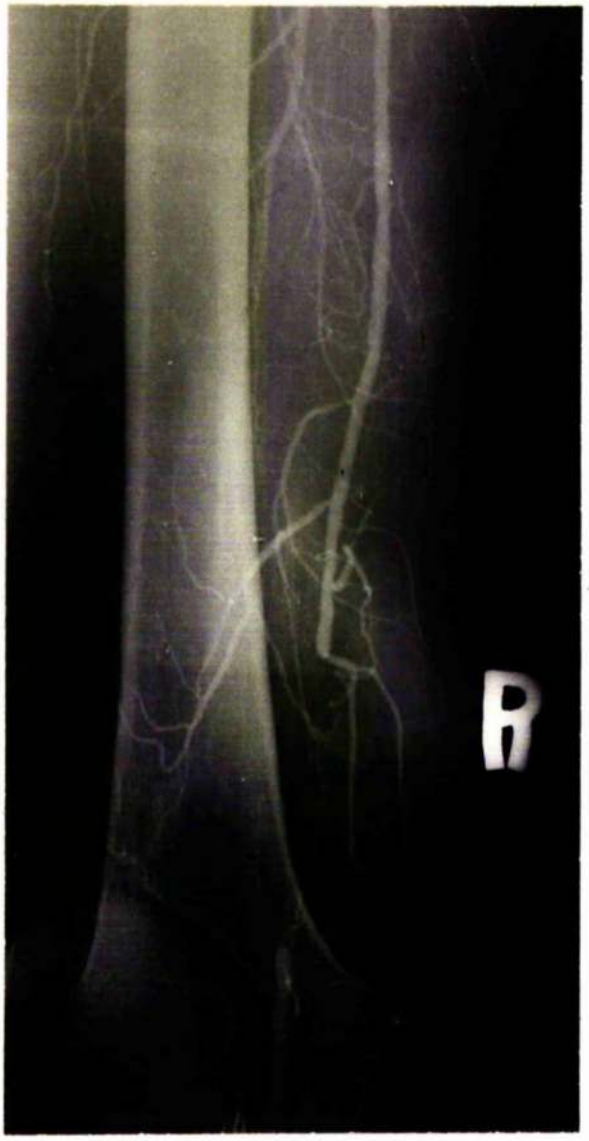


Fig. 101 : Short Popliteal Occlusion.

This arteriogram illustrates a typical occlusion of the upper part of the right popliteal artery. It is limited above by the descending genicular and saphenous arteries and below by the lateral superior genicular artery and the sural arteries.

This patient, R.Y., aged 62 years, complained of right sided claudication in 1959. Late in 1959 he developed angina of effort and in 1961 an occlusion of the opposite leg. This tendency to develop multiple lesions is typical of many atherosclerotic patients.

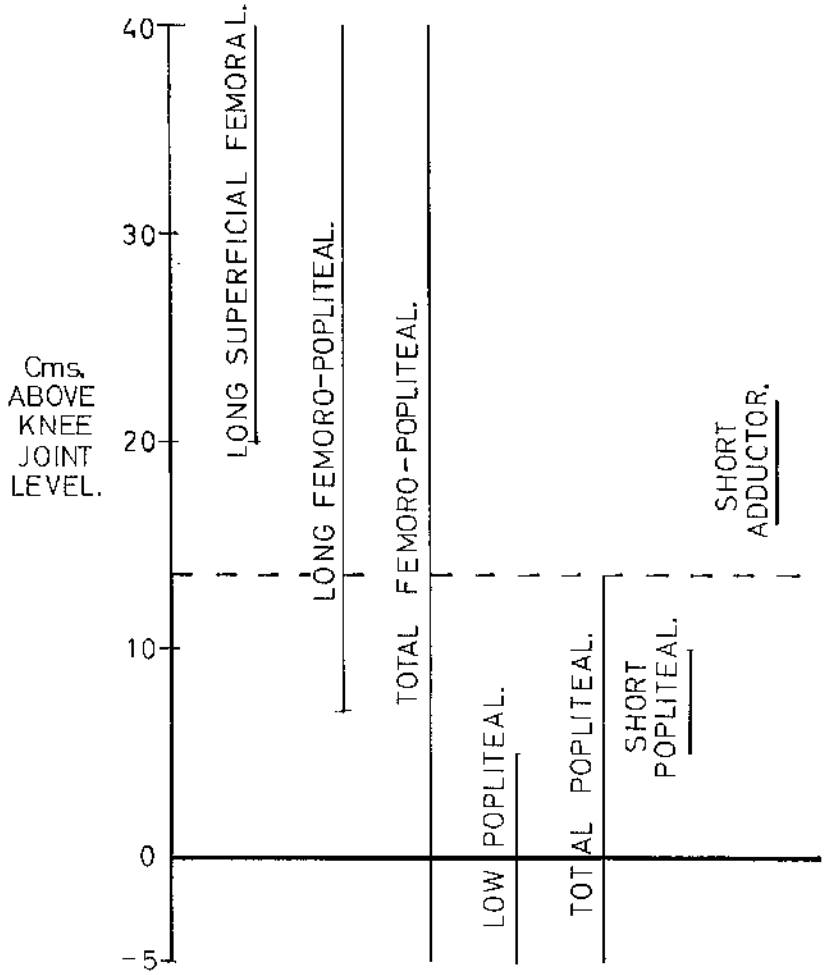


Fig. 102 : Classification of Femoro-Popliteal Occlusions.

In this diagram, the various types of femoro-popliteal occlusion described in the text are drawn against the 45 cm. scale to indicate their situation.

The short adductor occlusion shown (16-22 cms.) could equally well be represented by a variety of lines proximal or distal to the one illustrated as there is a large variation in the site of these occlusions (Fig.98).

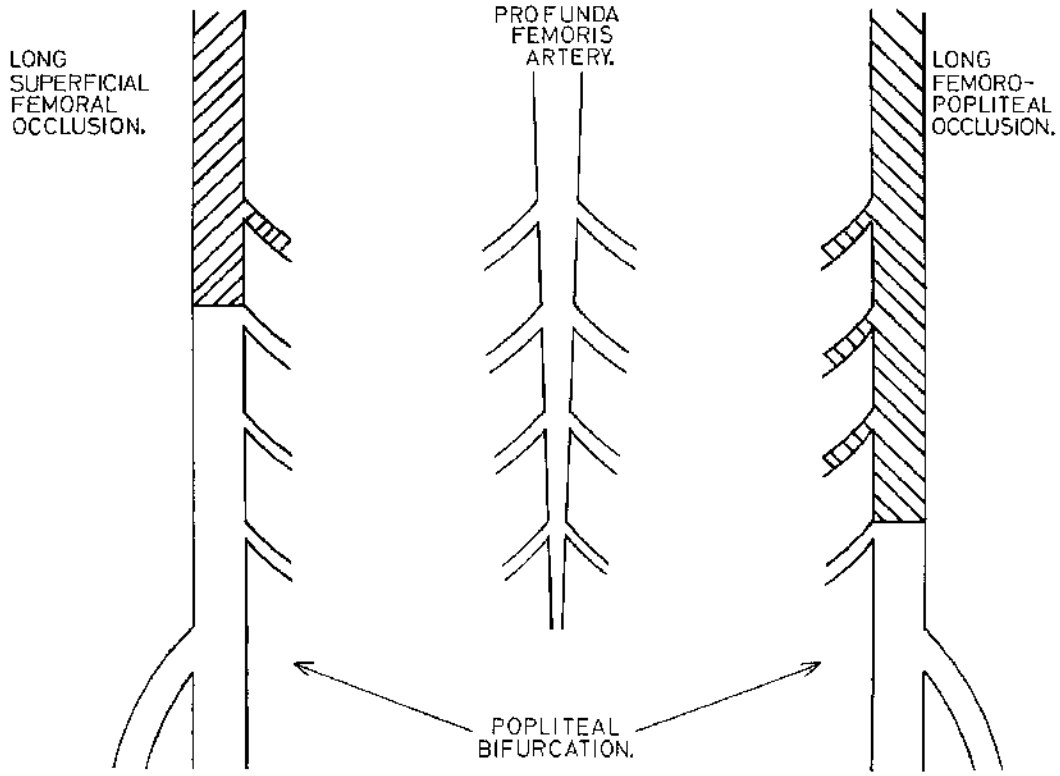


Fig. 103 : The Relationship between the Profunda Femoris Artery and Long Occlusions.

In this diagram, the profunda femoris is drawn in relation to a long superficial femoral occlusion on the left and a long femoro-popliteal occlusion on the right. The available collaterals in the former are theoretically more numerous than those available in the latter.

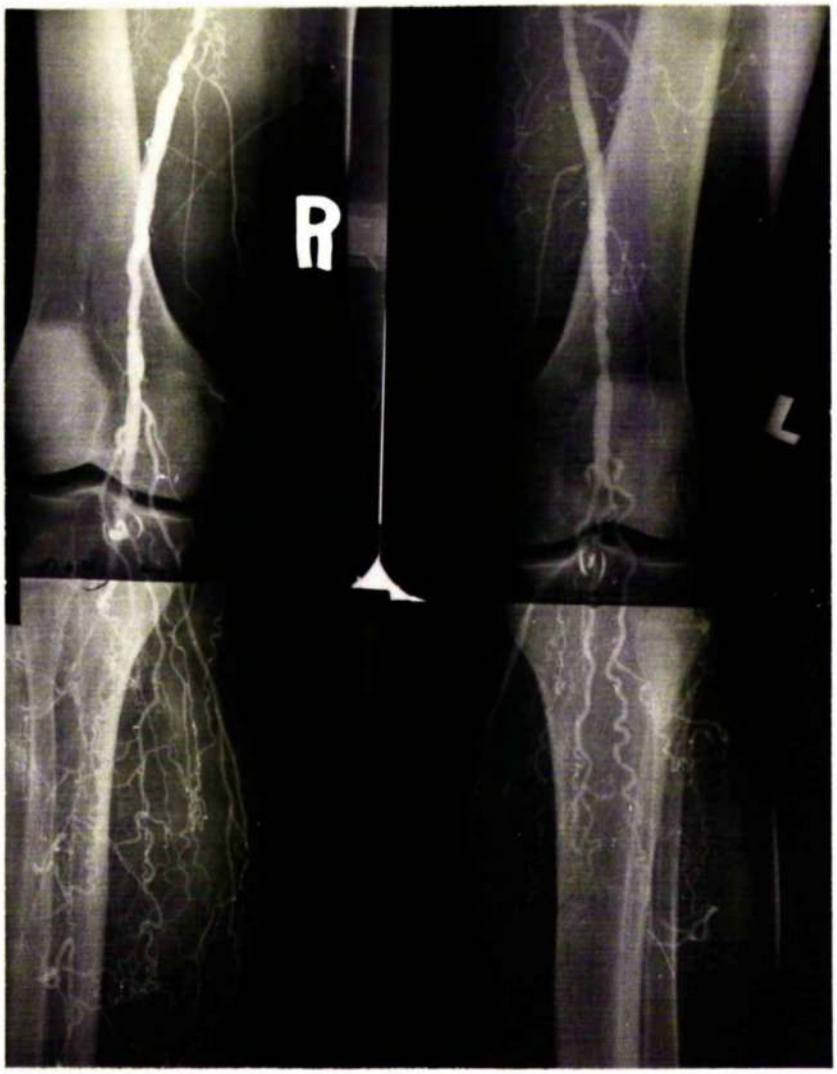


Fig. 104 : Low Popliteal Occlusion.

G.G., aged 68 years, had experienced bilateral claudication at 200 yards for seven years when first seen.

Arteriography shows symmetrical low popliteal occlusions with extensive development of collateral vessels derived from the sural arteries. There is a remarkable degree of symmetry in the two lesions and in the pattern of the collateral circulation.

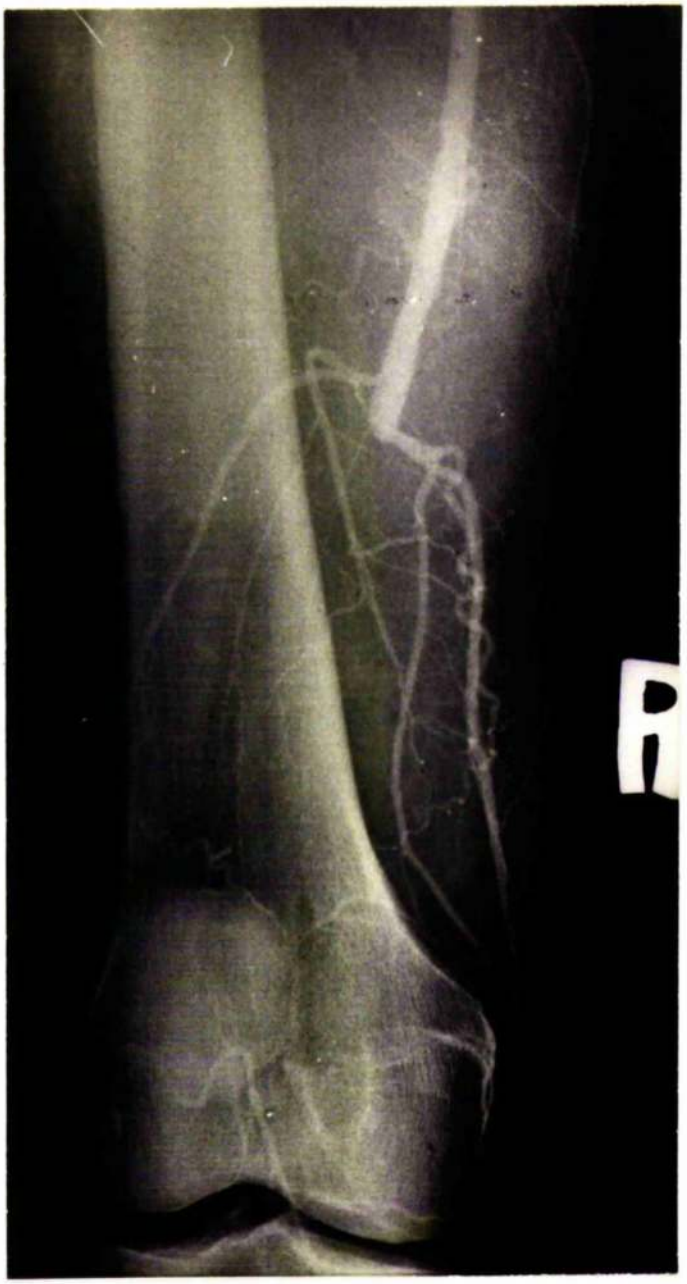


Fig. 105 : Total Popliteal Occlusion.

This arteriogram shows an occlusion of the popliteal artery from the adductor opening downwards. Occlusion of the popliteal artery was complete and the proximal half of the posterior tibial artery was also occluded.

A large saphenous artery is visible and this was felt to pulsate during clinical examination of the patient.

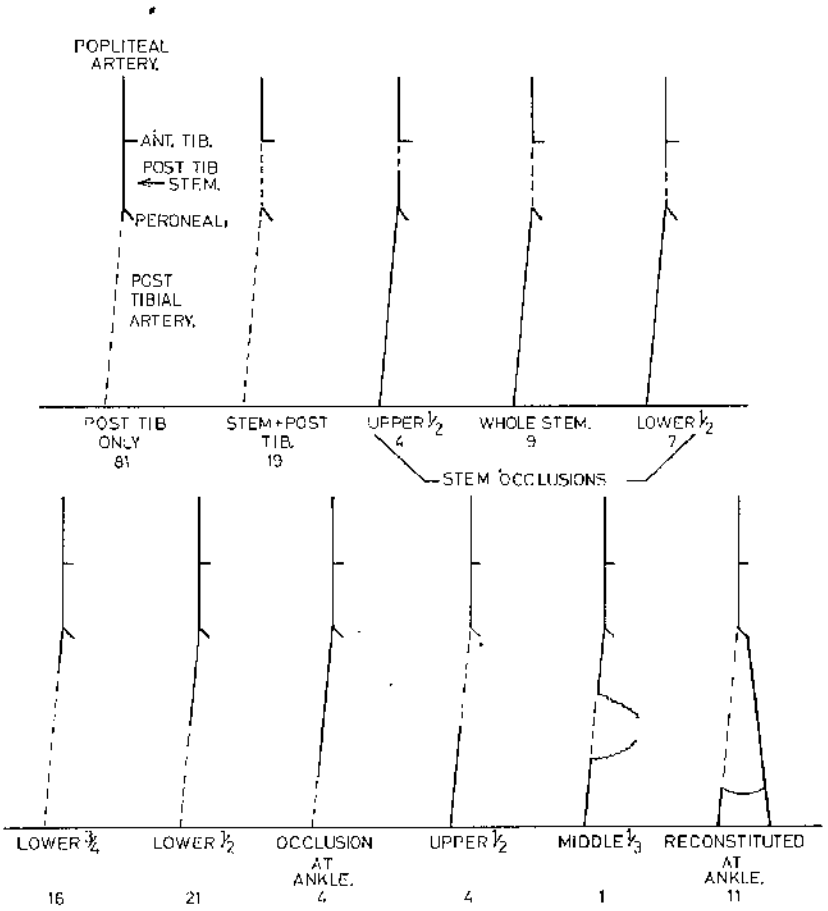


Fig. 106 : Occlusions of the Posterior Tibial Artery.

177 Occlusions of the posterior tibial artery have been classified in 11 patterns and the frequency of each type is shown by the number below each diagram. In the interests of clarity, associated occlusions of the anterior tibial and peroneal arteries are not shown.

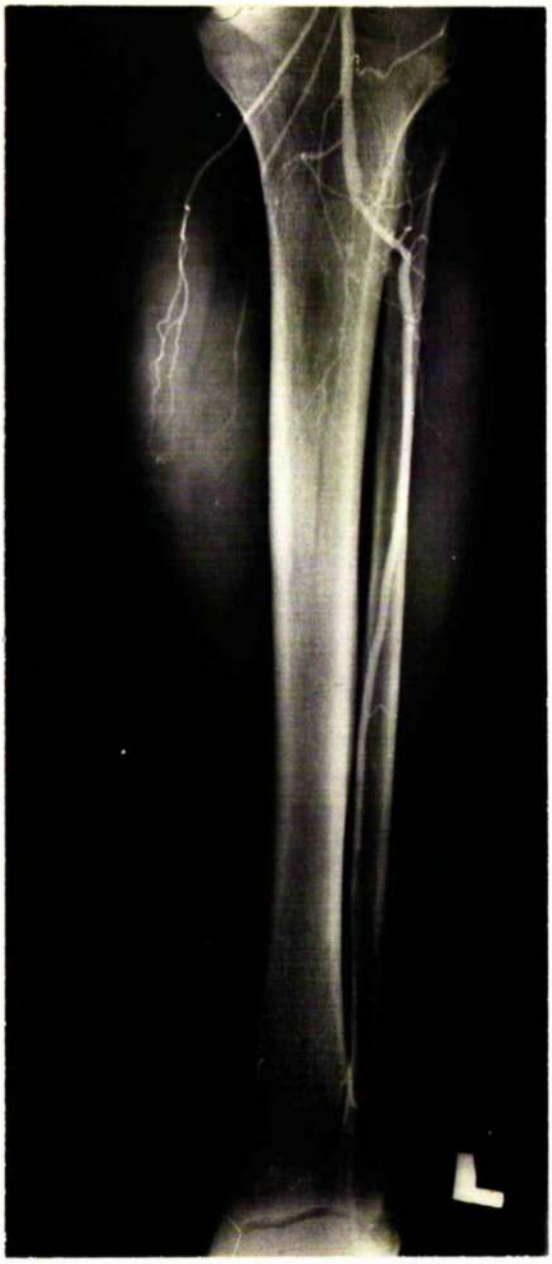


Fig. 107 : Occlusion of the Posterior Tibial and Peroneal Arteries.

In this arteriogram, the anterior tibial artery is the only patent lower leg artery and shows minor atherosclerotic changes. The posterior tibial and peroneal arteries are occluded and there is only a trace of dye in the posterior tibial stem which is virtually occluded.

Fig. 108 : Occlusion of the Posterior Tibial Artery.

In this arteriogram, there is a faint dye shadow in the proximal part of the posterior tibial artery but this peters out and the course of the rest of the vessel is shown by a line of irregular calcification. Reconstitution occurs above the level of the ankle joint from collateral vessels derived from the peroneal artery.

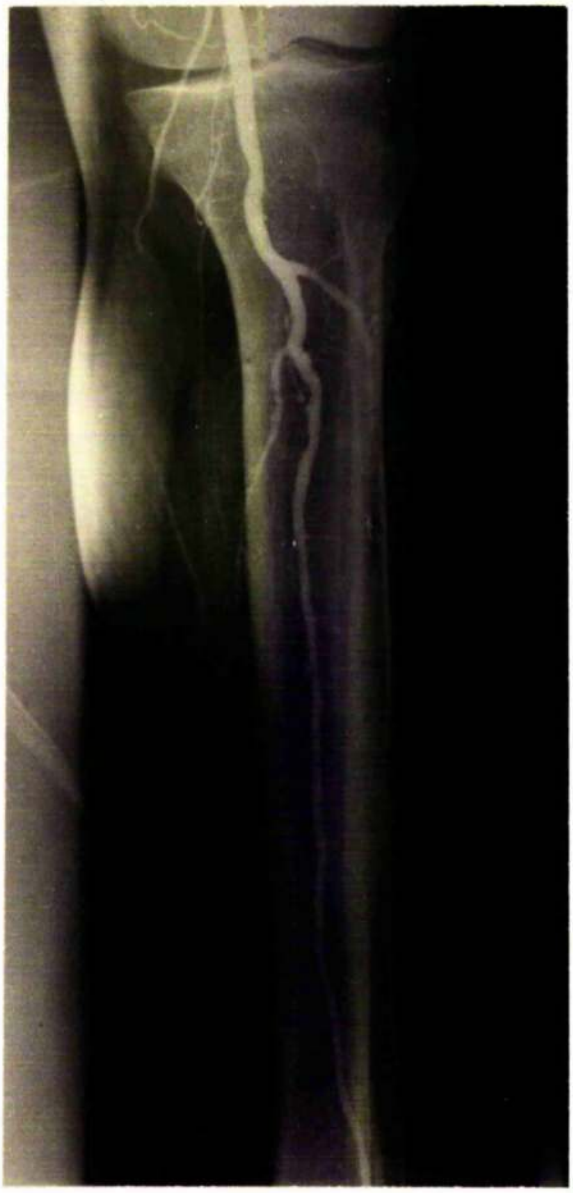


Fig. 109 : Occlusion of the Posterior Tibial Artery.

There is diffuse atherosclerosis of the lower leg vessels and the peroneal artery is the main source of supply to the foot. The posterior tibial artery is patent at its origin but gradually narrows and is occluded after a course of about 10 cms. The anterior tibial artery is patent but its course is obscured by superimposition on the cortex of the tibia.

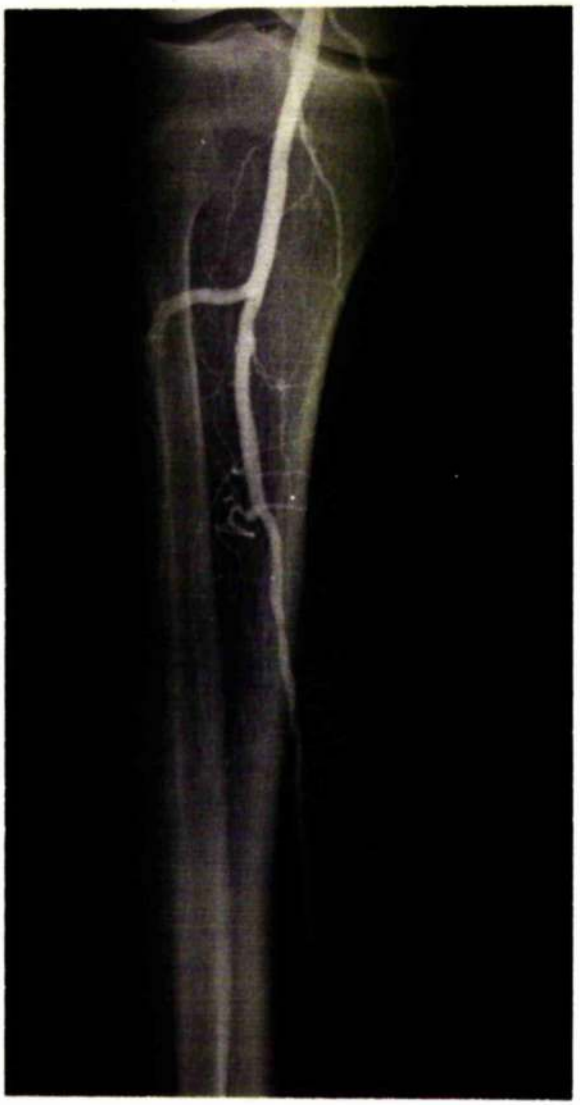


Fig. 110 : Occlusion of the Anterior Tibial and Peroneal Arteries.

In this arteriogram, the anterior tibial artery is occluded after a short course and the peroneal artery is not visible. The posterior tibial artery is the only patent vessel and this combination of anterior tibial/peroneal occlusion was found in only three cases.

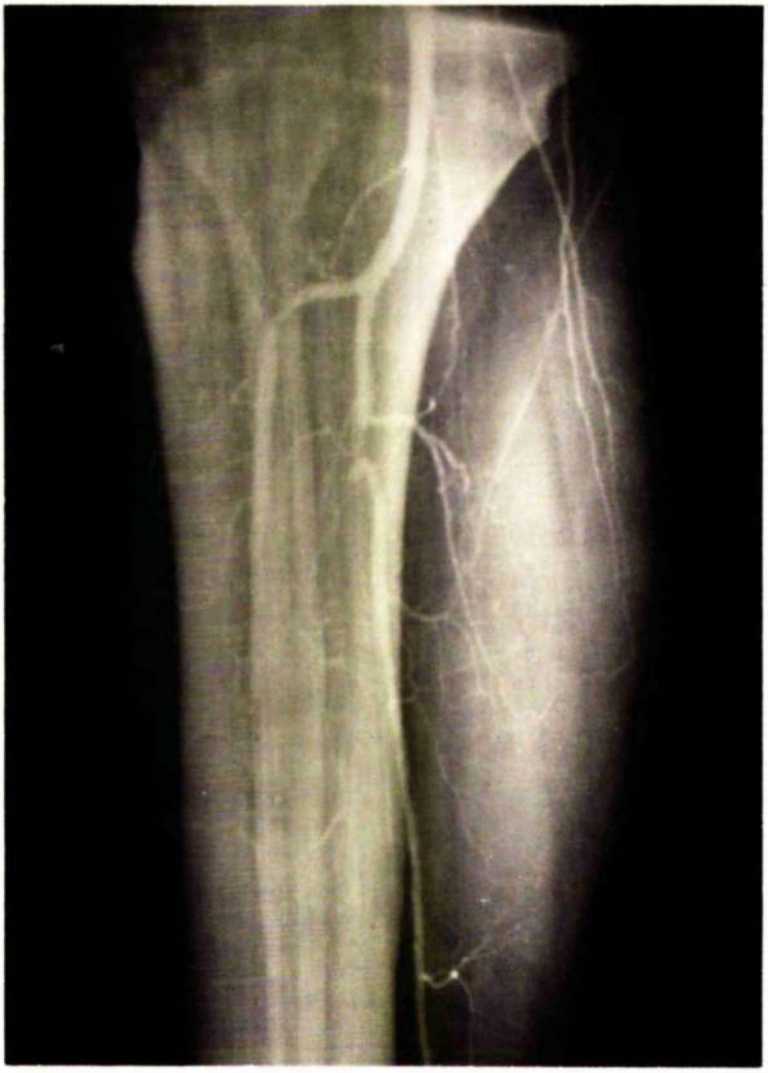


Fig. 111 : Incipient Occlusion of the Anterior Tibial Artery : Occlusion of the Peroneal Artery.

The anterior tibial artery is patent but is narrowed where its shadow overlies the medulla of the fibula. Progression of this lesion is likely to produce the typical occlusion (Fig.110) where the vessel is occluded after a short course.

Occlusion of the peroneal artery is present and the posterior tibial artery is patent.

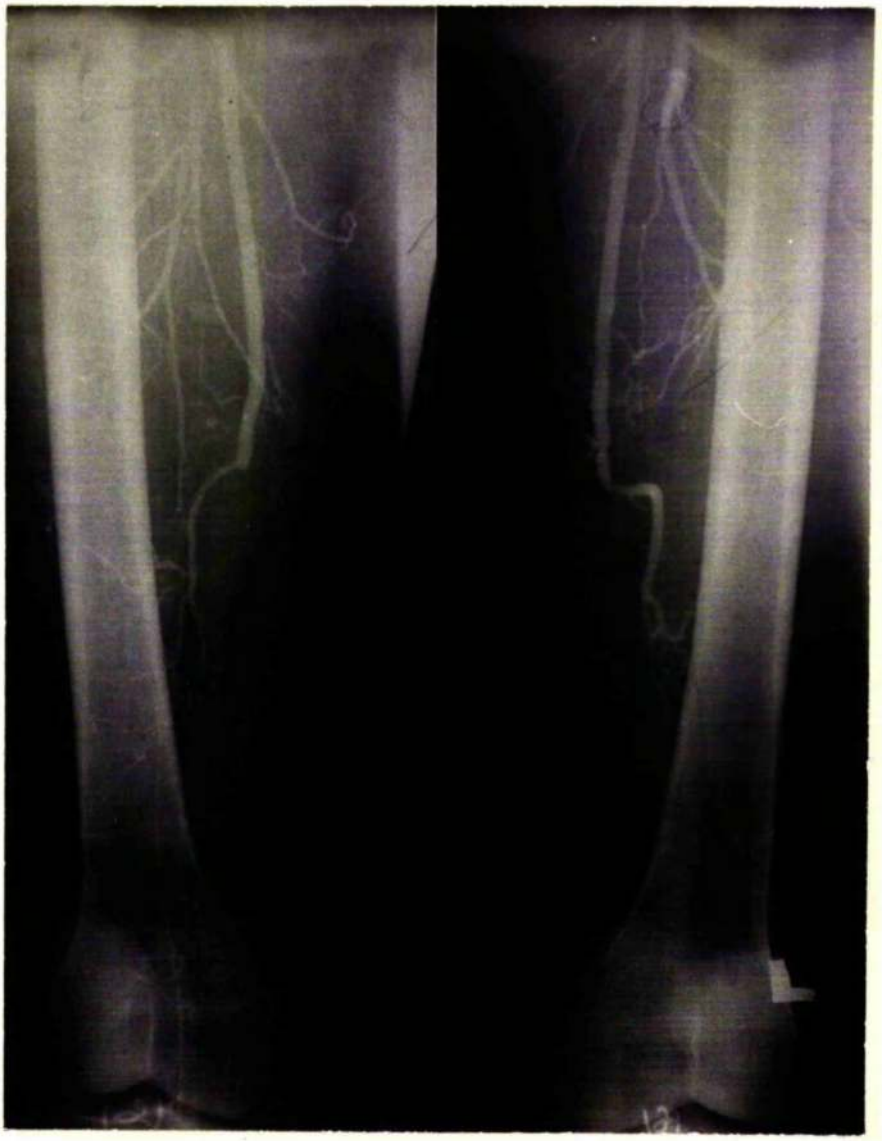


Fig. 112 : Symmetrical Femoro-Popliteal Occlusions.

Bilateral femoral arteriography in this patient (J.McG., aged 56 years) shows occlusion of the lowest part of the superficial femoral artery and the whole of the popliteal artery in both legs. Reconstitution of the tibial vessels occurred and he experienced bilateral claudication at 100 yards.

There is evidence of symmetry in regard to the length of artery occluded and the appearances of the limiting collaterals issuing from the lower end of the patent segments.

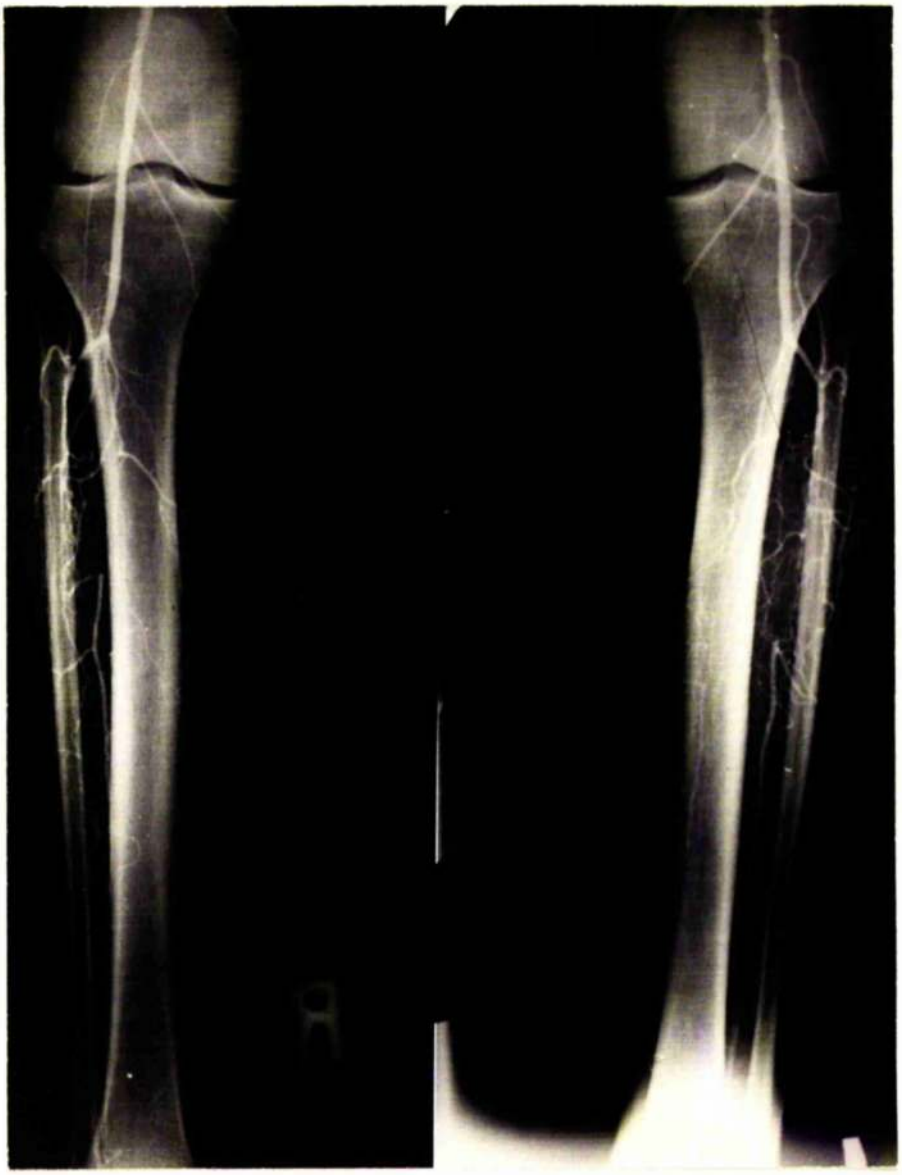


Fig. 113 : Symmetrical Multiple Occlusion in the Lower Leg.

In this female patient, both femoral and popliteal arteries were patent but the lesions in both lower legs were multiple. There is bilateral occlusion of all three lower leg vessels with reconstitution of the peroneal artery at corresponding levels, and the pattern of the collateral circulation is virtually identical.

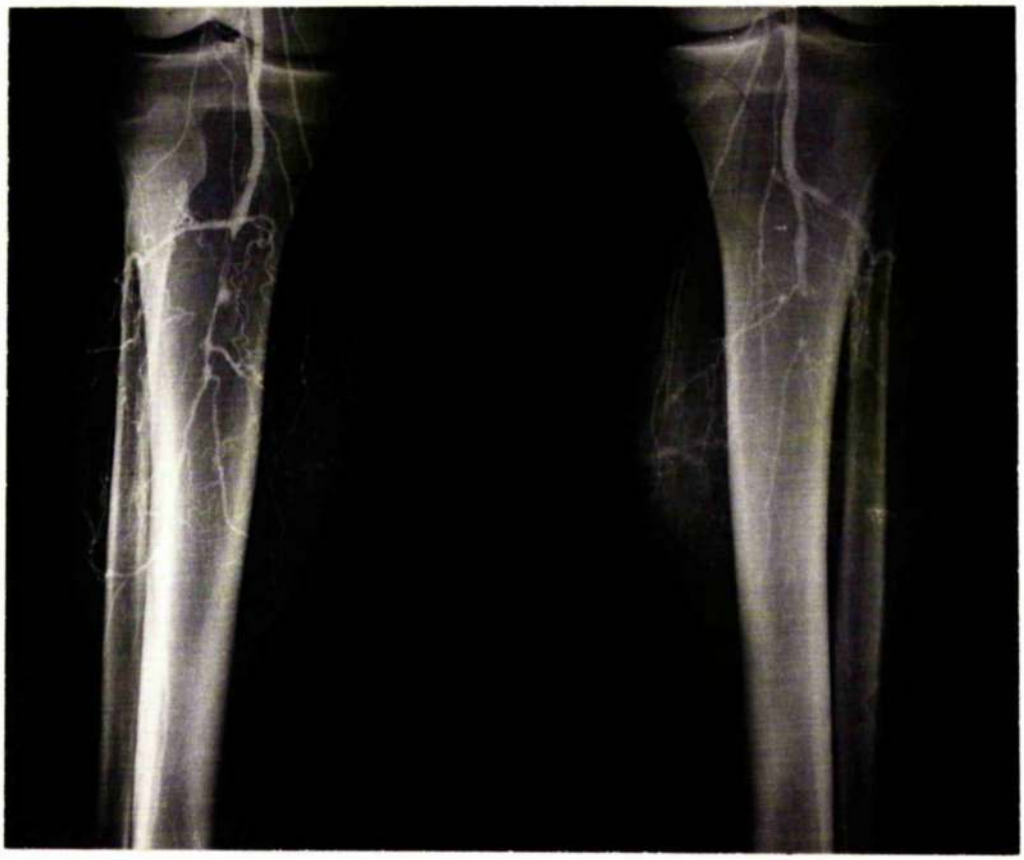
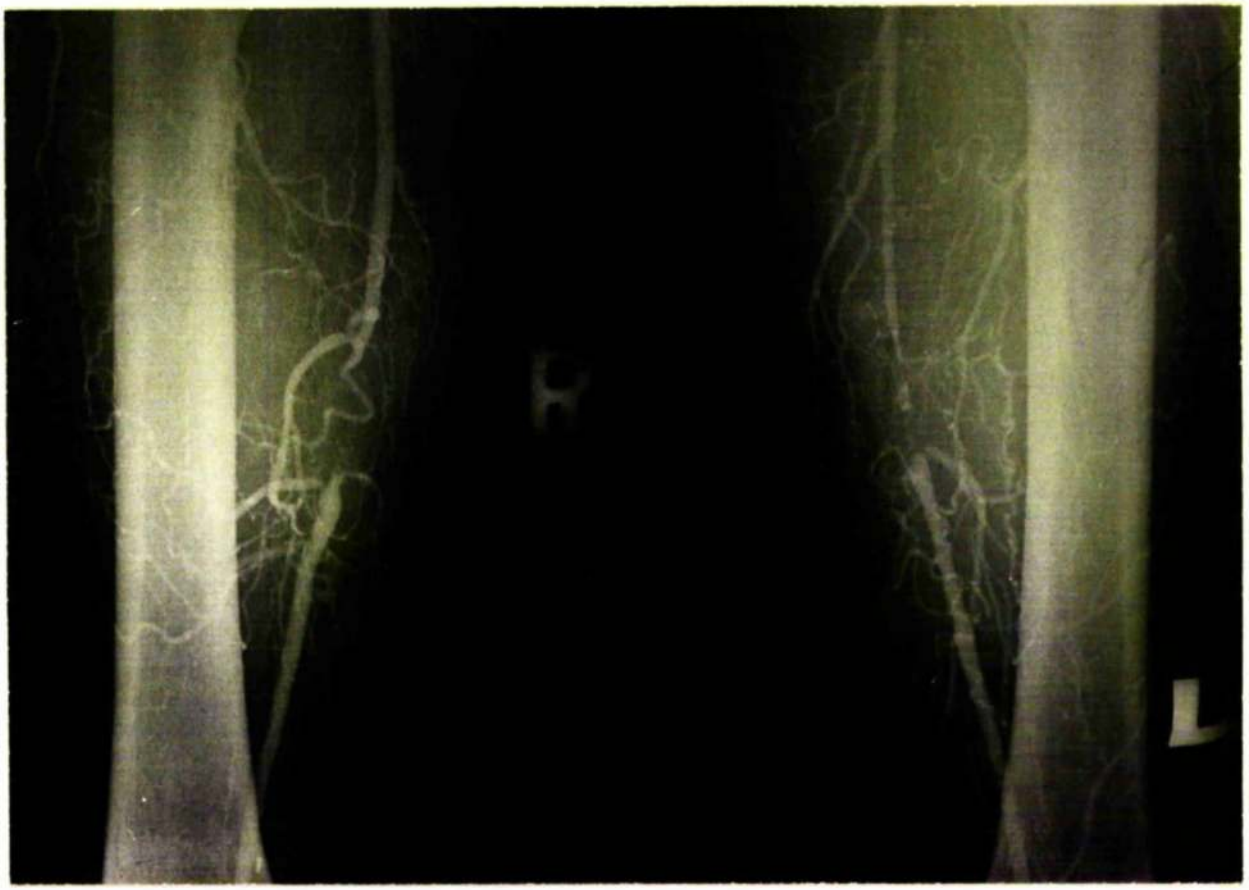


Fig. 114 : Symmetrical Multiple Occlusion in the Lower Leg.

In this elderly man, both femoral and popliteal arteries were patent. The pattern of arterial occlusion in both lower legs is similar and lesions of all three arteries are present. The appearances of the two legs resemble each other sufficiently as to be classified as symmetrical lesions.



1960. 1959.

Fig. 115 : Symmetrical Short Adductor Occlusions.

J.H., aged 47 years, complained of left sided claudication in 1959 and arteriography revealed a short adductor occlusion (illustrated on the right of the photograph).

In 1960, he developed bilateral claudication and was found to have an occlusion in the right leg similar to that in the left leg.

These two occlusions occurred at intervals of one year and it appears that his lesions were originally symmetrical although, at the time of the first occlusion in 1959, asymmetry was present.

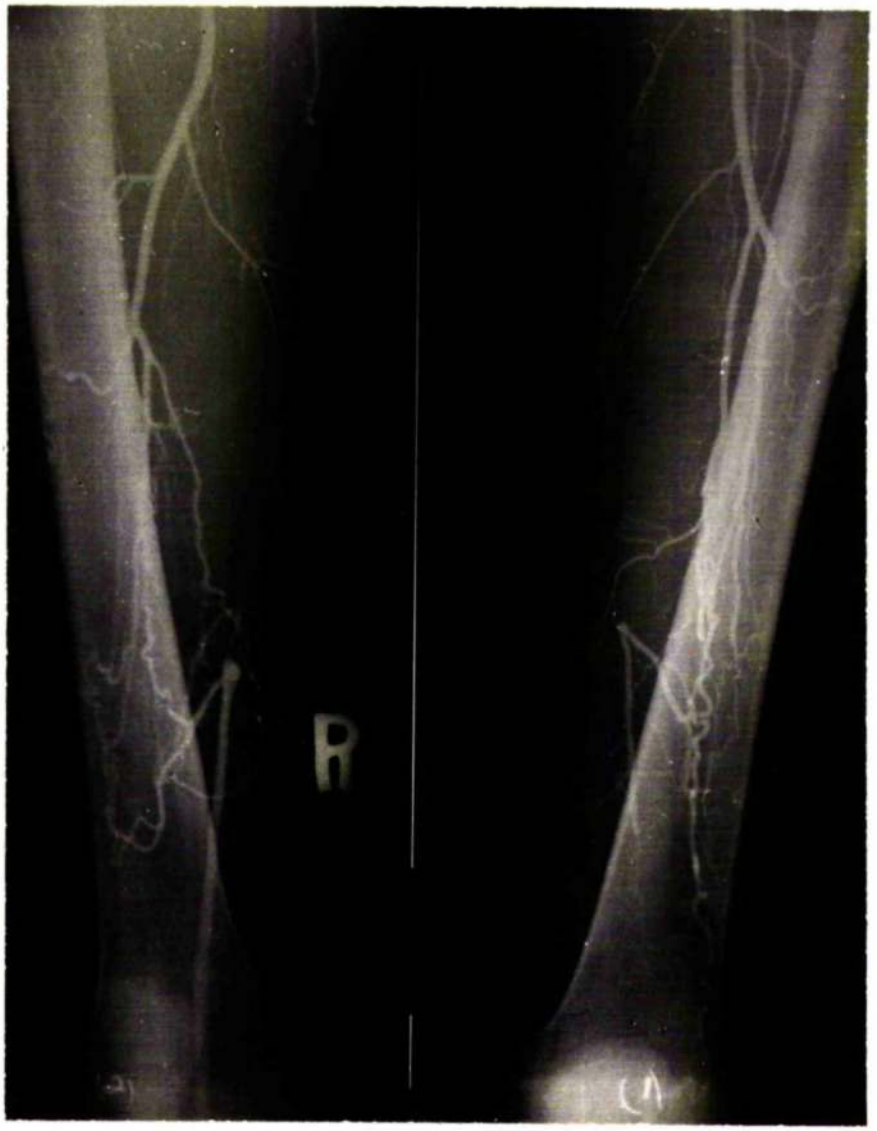


Fig. 116 : Symmetrical Superficial Femoral Occlusions.

A.B., aged 56 years, complained of bilateral claudication at 100 yards in 1958. Before investigation was commenced, he developed ischaemic rest pain in the left foot which was not relieved by sympathectomy and below knee amputation was necessary.

Bilateral femoral arteriography after the onset of rest pain shows symmetrical long superficial femoral occlusions and an additional popliteal occlusion on the left. The latter occlusion is believed to have occurred after his first attendance i.e. his lesions were initially symmetrical.

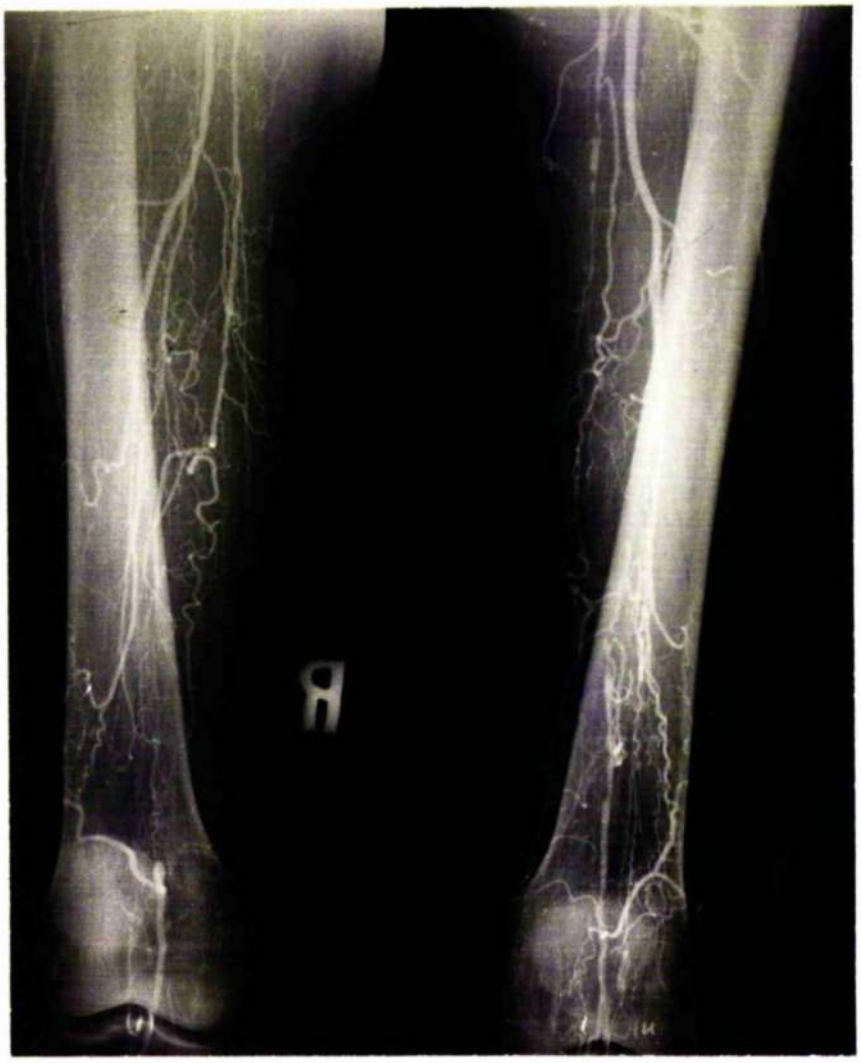


Fig. 117 : Bilateral Femoro-Popliteal Occlusion.

Bilateral femoral arteriograms of this patient (P.C., aged 69 years) show a symmetrical pattern in the thighs. Both superficial femoral arteries are grossly diseased proximally and there is a femoro-popliteal occlusion in each leg. Although the main channel is reconstituted at a higher level on the left side, the pattern of the collateral circulation in each leg is very similar.

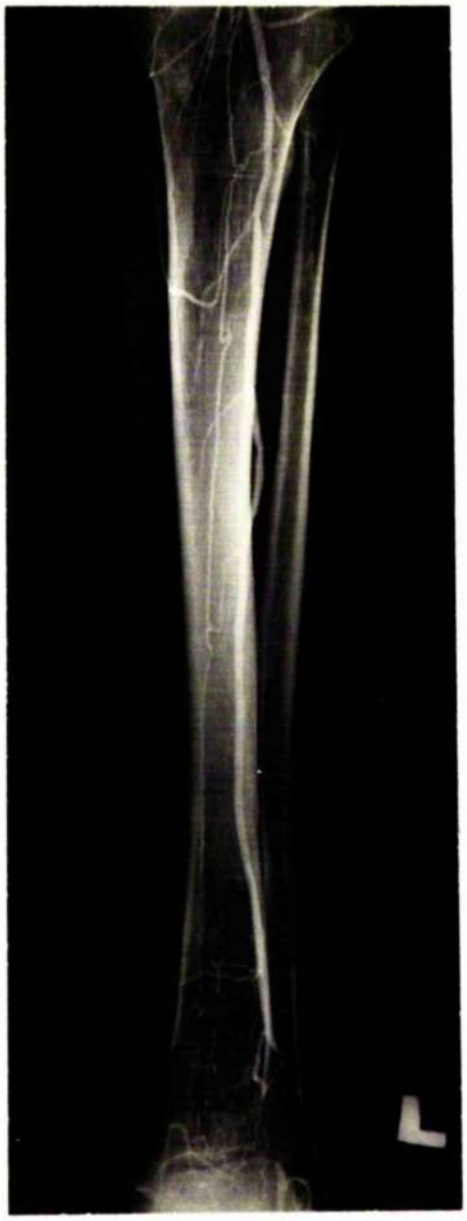


Fig. 118 : Occlusion of the Anterior and Posterior Tibial Arteries :"Peroneal Leg".

This arteriogram shows the vessels in the left lower leg of the patient illustrated in Fig.117, the arteries of the right lower leg being patent.

The anterior and posterior tibial arteries are occluded and the blood supply to the foot is carried by a large peroneal artery. This terminates above the ankle joint in a number of collateral vessels from which the dorsalis pedis artery is reconstituted.

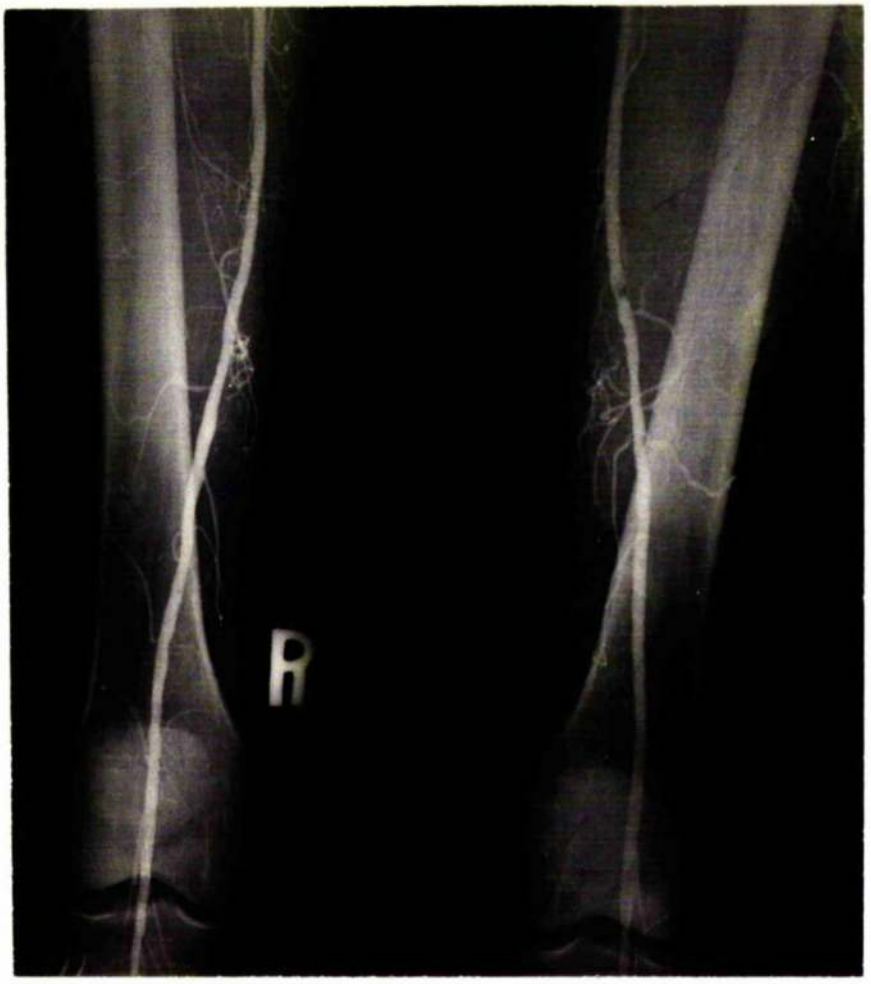


Fig. 119 : Incipient Left Superficial Femoral Occlusion.

The left arteriogram in this patient (J.P., aged 47 years) shows narrowing of the superficial femoral artery due to a large plaque. There are atherosclerotic changes only on the right side and the lower leg films are reproduced in Fig.120.

This patient experienced bilateral claudication at 200 yards which was more severe in the left leg. A left selective nerve crush was performed in 1959 and he was subsequently able to walk 2 miles before the onset of pain and he has remained well to date.

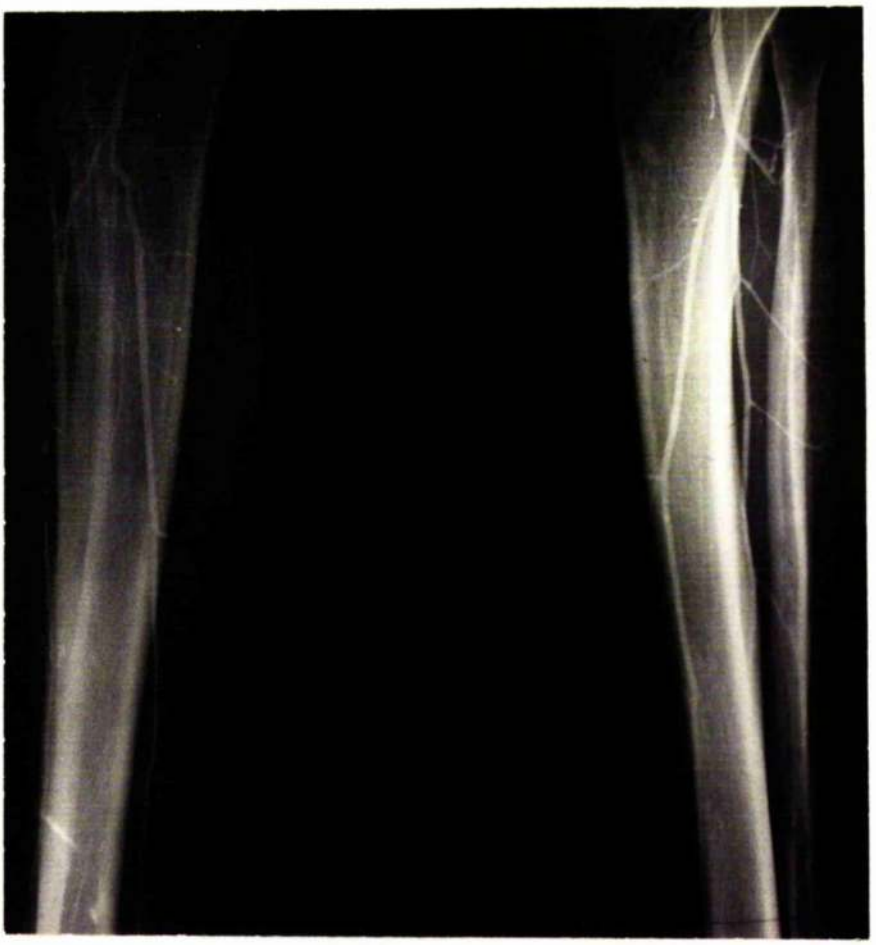


Fig. 120 : Occlusion of the Right Anterior Tibial and Peroneal Arteries : Occlusion of the Left Anterior Tibial Artery.

The upper films of this patient are illustrated in Fig.119.

The right lower leg films show some irregularity of the posterior tibial artery near its origin, occlusion of the peroneal artery and occlusion of the anterior tibial artery in its upper half.

The left anterior tibial artery is occluded after a short course and the other two arteries are patent.

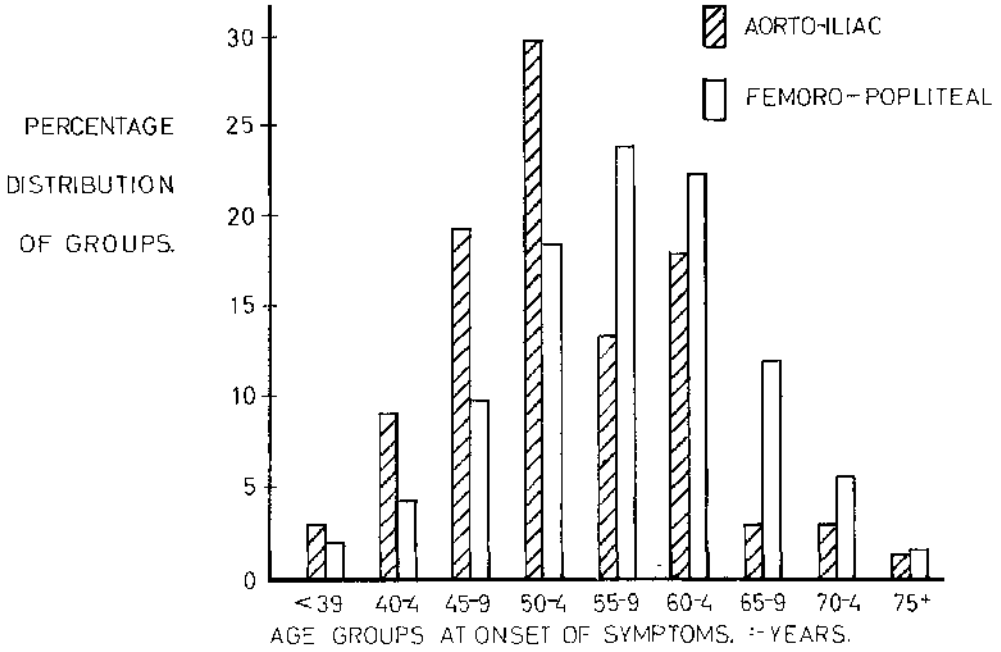


Fig. 121 : Age Distribution of Patients with Intermittent Claudication.

In this figure, the percentage distribution by age groups has been charted for 67 patients with aorto-iliac occlusion and 305 patients with femoro-popliteal and tibial occlusion.

There appear to be two peaks in the aorto-iliac curve and the distribution of observations in this group suggests that a large proportion experience symptoms relatively early in life when compared with femoro-popliteal occlusions.

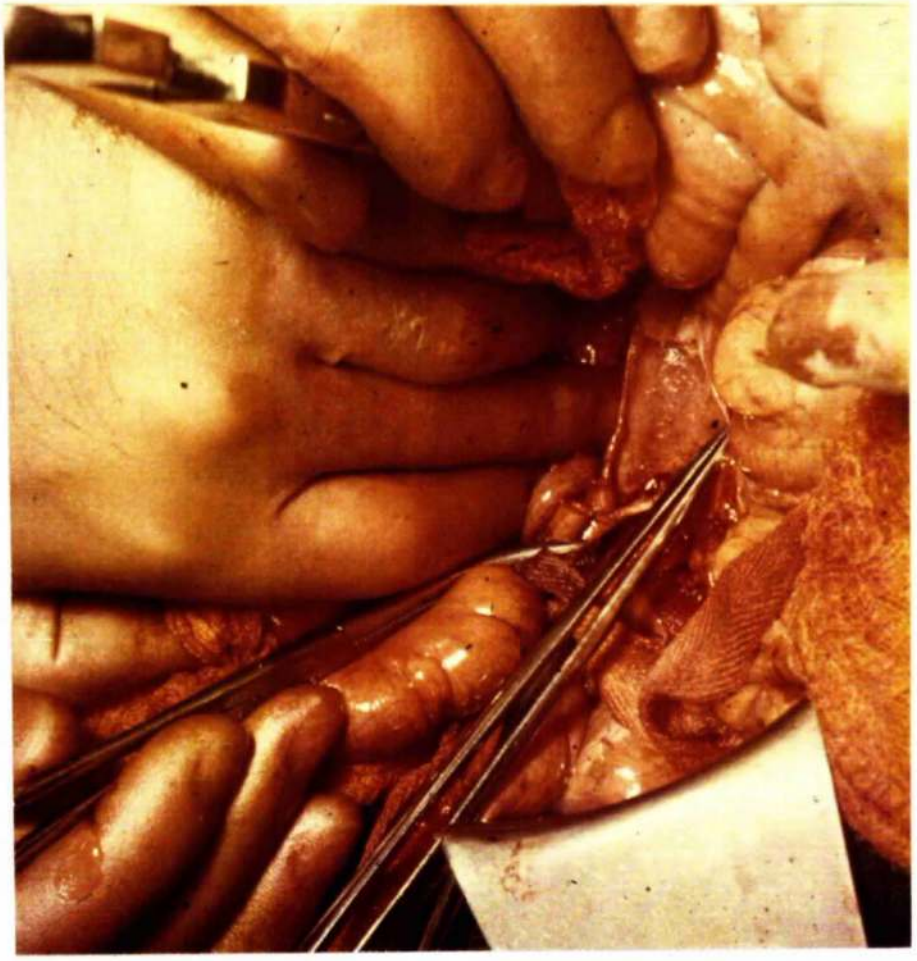


Fig. 122 : Thrombo-Endarterectomy of the Right Common Iliac Artery.

In this operation photograph, the lesion illustrated in Fig.89 has been exposed by incision of the right common iliac artery (Fig.33) and thrombo-endarterectomy has been performed.

The dissecting forceps is holding the medial wall of the artery and a dark red patch 1 cm. from the tip of the forceps is the orifice of the internal iliac artery from which a calcified plug has been removed.

Dissection was commenced at the origin of the external iliac artery and the lesion was stripped up and excised leaving white intima at the upper end and a glistening attenuated medial coat where the lesion was removed.

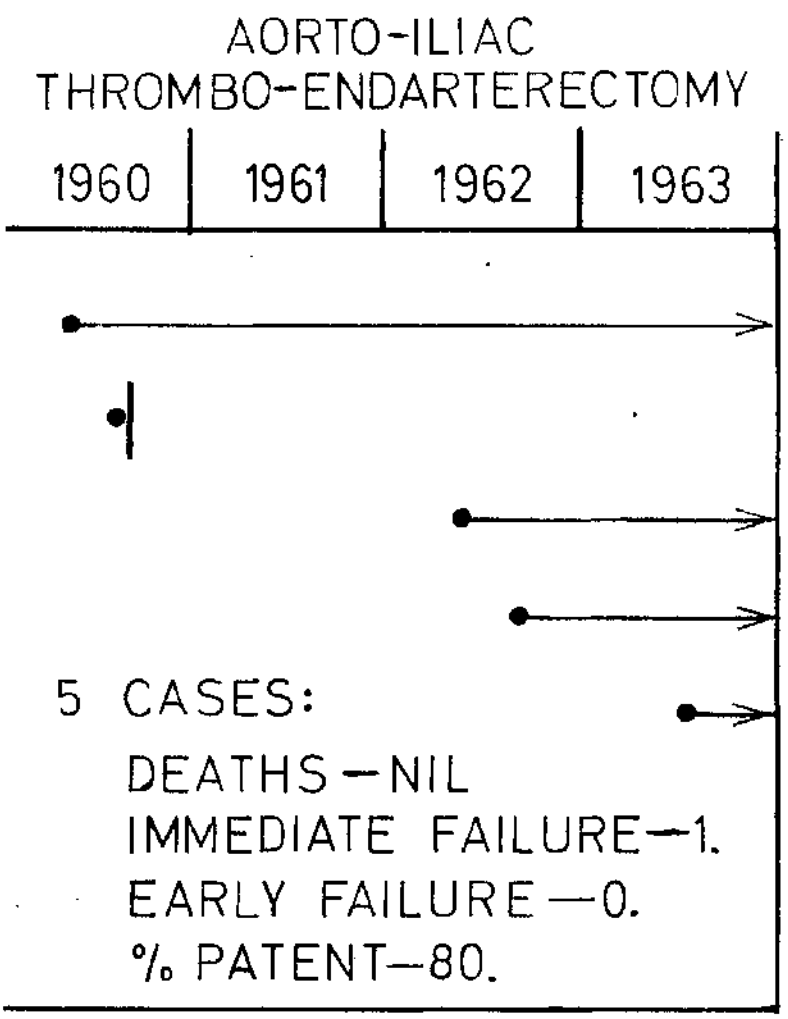


Fig. 123 : Results of Thrombo-Endarterectomy in Five Patients with Intermittent Claudication.

These five patients had relatively early lesions and the results of operation have been good as judged by the patency rate and the freedom from symptoms in four patients. The lesions of the 1st and 3rd patients are illustrated in Figs. 85 and 89 respectively.

The lesion in the second patient is illustrated in Fig.8 and, after failure of the thrombo- endarterectomy, she developed aortic occlusion up to the level of the renal arteries and appears in Fig.125 as the third patient. A bilateral aortic graft was also unsuccessful and she died from haemorrhage.

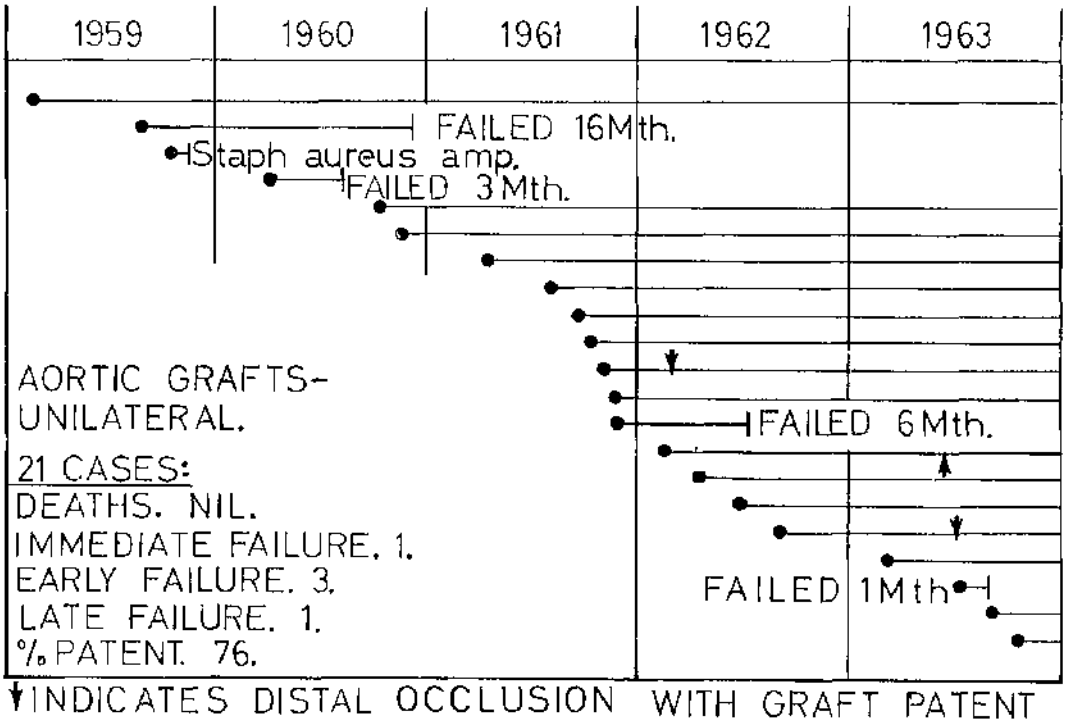


Fig. 124 : The Results of Unilateral Bypass Teflon Grafts in 21 Patients with Intermittent Claudication due to Common Iliac Occlusion.

The results are good and there was no operative mortality.

16 (76%) are still patent although the follow-up in three patients is less than one year.

However, seven of these sixteen still have symptoms although the graft is patent. Two had known distal occlusion at the time of operation, three have subsequently developed femoro-popliteal occlusion, and two have mild residual symptoms although all vessels are known to be patent.

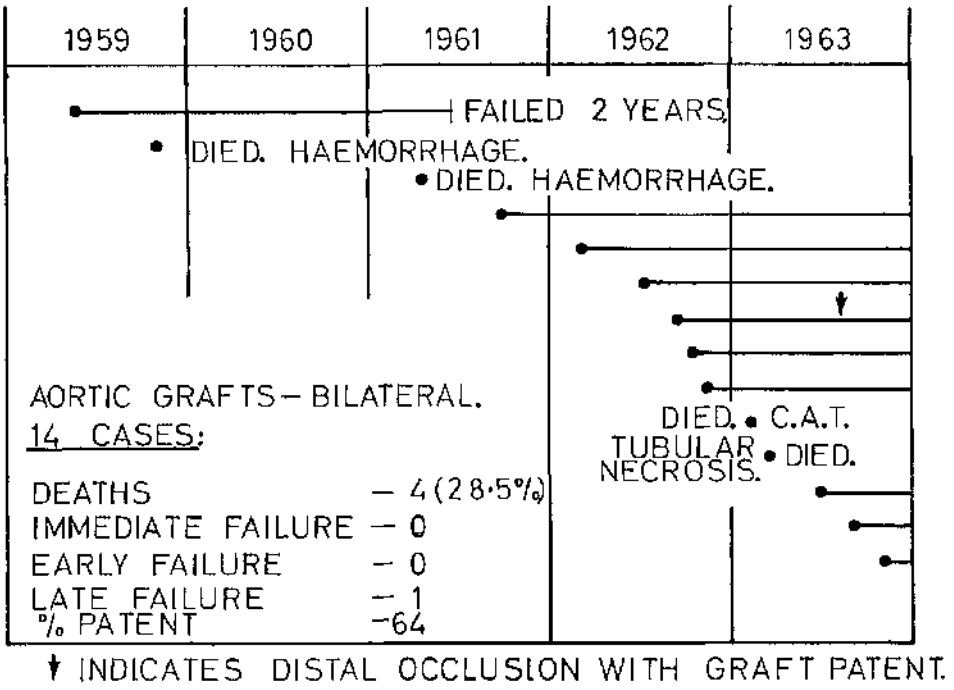


Fig. 125 : Results in 14 cases of Bilateral Bypass Teflon Grafts performed for Intermittent Claudication.

These patients had aortic occlusion, aortic bifurcation lesions or bilateral common iliac lesions. The operative mortality is high and the causes of death are discussed in Chapter 12.

All the grafts in the 10 survivors were patent at the time of discharge from hospital but one thrombosed after 2 years and another patient (No.7) has developed a femoro-popliteal occlusion. The last patient had a known distal occlusion at the time of operation and the remaining seven are symptom free.

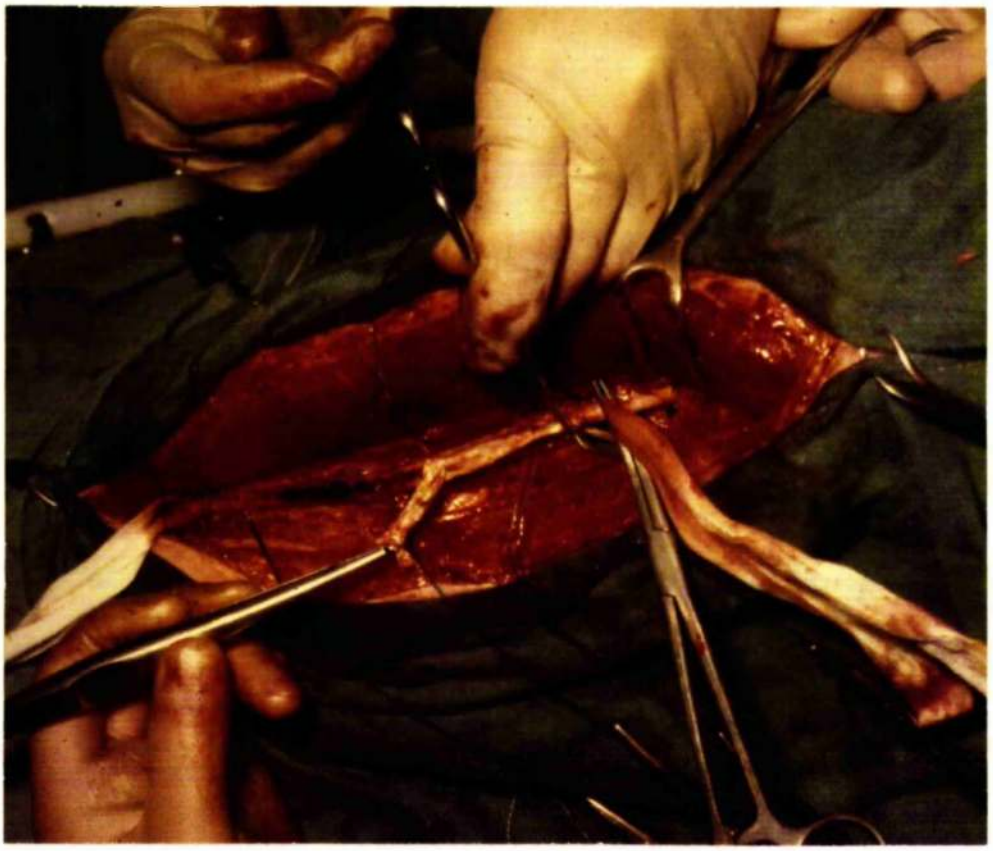


Fig. 126 : Thrombo-Endarterectomy of the Superficial Femoral Artery.

The lesion illustrated in Figs. 96 and 97 has been exposed by a long arteriotomy and dissection has been commenced below the stenotic area. About 3 ins. of thickened intima were removed together with recent thrombus and the artery was reconstituted by a long continuous suture.

Re-thrombosis occurred a few months later and he has an occlusion of the whole length of the superficial femoral artery. In spite of this, his claudication distance has not decreased and he is able to continue working.