Fig. 2.—Juvenile obliterative arteritis—Later stage.

(By courtesy of the Editors of the 'Journal of Bone and Joint Surgery')
INTERMITTENT CLAUDICATION
A REVIEW

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PART I
HISTORICAL

Introduction

Intermittent claudication, now recognized as one of the most constant symptoms of obliterative arterial disease, may be defined as a discomfort of varying severity, produced by exercising a muscle under ischaemic conditions and being rapidly relieved by rest. The original report of the phenomenon appears to be that of the veterinary surgeon Boullay (1831), who observed it in a six-year-old mare. According to Boullay the cause was obliteration of the femoral arteries; when the animal was at rest the collateral blood supply was adequate, but when it trotted the blood supply was insufficient. 'The result was 'une douleur profonde.'

The clinical picture was described by Charcot (1858) in a 54-year-old man having an aneurysm of the right common iliac artery with obliteration of the distal part of the vessel. Strangely enough this is the least common site of obstruction of a vessel supplying the leg and the occurrence of such a case is still considered worthy of comment (Boyd and Jepson, 1950). Further examples of intermittent claudication were given by Marinesco (1896) and Erb (1898).

Classification of Occlusive Arterial Disease

The medical literature of the 19th century abounds with clinical reports of occlusive arterial disease leading to gangrene and resultant amputation. Each individual author appears to have noticed some distinctive feature with consequent justification for the coining of a new term to describe the general clinical condition. Brown, Allen and Mahorner (1928) give a list of no fewer than 34 names which have been coined in this way.

The first attempt at investigation was that of von Winiwarter in 1879. He studied the vessels in a leg amputated for gangrene, and noted the proliferation of the intima, the presence of a cellular mass gradually producing occlusion of the vessel, and the presence of thrombi in the vessels. The thrombi appear to have been regarded as adventitious, as von Winiwarter concluded that the final obliteration of the vessel was the direct result of the cellular proliferation. The term applied to this condition was 'endarteritis obliterans.' This classic article has been used as a stepping stone by many writers since its publication, and, in consequence of the deductions made from the report, it should be emphasized at this point that von Winiwarter's patient was 57 years of age. The significance of this will be shown later.

The next important advance in the appreciation of occlusive vascular disease is associated with the name of Leo Buerger. In 1908 he made a report based on the study of the arteries and veins in 11 amputated limbs. A series of papers by the same author culminated in a monograph which appeared in 1924. Buerger distinguished two main conditions and called them 'thromboangiitis obliterans' and 'atherosclerosis obliterans.' The former condition has acquired the name Buerger's disease.

The distinction between the two diseases rested chiefly on the age of the patient. Thromboangiitis obliterans was diagnosed in individuals below the age of 45 and arteriosclerosis obliterans over the age of 55. Between those two ages there existed a mixed group the nature of whose condition was difficult to ascertain. An additional distinction was the presence of superficial phlebitis in 30 per cent. of cases of thromboangiitis obliterans; this adjunct was never observed in arteriosclerosis obliterans.

The tacit acceptance of these definitions has led to the widespread idea that two very distinct forms of occlusive vascular disease exist, one occurring in young men in whom arteriosclerotic changes are completely absent; the other occurring in later life, arteriosclerotic changes being diagnostic. Here a reference to Buerger (1908) himself is worthy of note. The original pathological study led Buerger to the conclusion that thromboangiitis obliterans was an acute inflammatory condition with a sequel of thrombosis. The intimal proliferation reported by von Winiwarter (1879) was not considered important, the
FIG. 1.—Juvenile obliterative arteritis—Early stage.

changes observed being in Buerger's opinion due to arteriosclerosis.

De Takats (1934) added two further groups to Buerger's nomenclature. Under the heading of 'acute vascular occlusion' he listed patients with thrombotic or embolic block of a main vessel of the limb, irrespective of cause. He revived the term 'endarteritis obliterans' and applied it to 'the obliterative healed stage of many different chemical and bacterial injuries that affect the intima.' This group included lesions due to frost bite, syphilis, tuberculous arteritis and poisoning by metals such as lead and arsenic.

Whatever the views as to the nature of the underlying arterial lesion, all writers are agreed that the principal symptom causing the patient to seek medical advice is the onset of intermittent claudication. It was present in approximately 80 per cent. of the cases reported.

Treatment

If the 19th century may be said to be noted for the variety of the terminology applied to occlusive vascular disease, then the first part of the 20th century can with equal justice be said to be noted for the multiplicity of treatments.

Repeated intravenous injections of various solutions, which it was considered might halt the progress of arterial disease, have been tried. Steel (1921) considered that there was a reduction in clotting time of the blood which resulted in thrombosis. He therefore advocated the use of sodium citrate. Koga (1913) felt that the viscosity of the blood was increased in thrombangiitis obliterans and advised carrying out the appropriate dilution with Ringer's solution. Silbert (1935) reported the results of treatment of 524 cases of occlusive vascular disease with hypertonic (5 per cent.) salt solution and recorded improvement in 434 cases.

Ambard, Boyer and Schmid (1926) found that pain was relieved and the colour of the skin improved following the subcutaneous injection of 10 units of insulin daily. Barker, Brown and Roth (1935) obtained results with various tissue extracts in alleviating the pain of intermittent claudication.

Various substances have been used to produce
vasodilatation. Goodman and Gottesman (1923) suggested the use of non-specific protein for this purpose. Good results have been reported in a limited number of cases by Brown, Allen and Mahorner (1928) from the Mayo Clinic, and A. W. Allen (1930) from the Massachussets General Hospital. Papaverine hydrochloride has been used by Allen and MacLean (1935) in cases of sudden arterial occlusion. The tetraethylammonium compounds have been suggested as vasodilators in peripheral vascular disease (Lyons et al., 1947) although their action has been shown to be capricious (Boyd et al., 1948).

Buerger (1924) devised a series of postural exercises consisting primarily of elevating the legs at an angle of 45° for two minutes followed by the patient sitting on the edge of the bed with his feet hanging down for a further two minutes. During this period the patient was supposed to carry out a routine of dorsiflexion, extension, inversion and eversion of the feet and flexion and extension of the toes. After this he lay flat in bed, with radiant heat treatment, for five minutes.

Physiotherapy has been advised by several writers, the most popular form being short-wave diathermy (Wright, 1938). A thermo-regulated foot cradle was produced by Starr (1931). Contrast baths were recommended by Brown et al. (1928). The legs were immersed for one-minute periods alternately in hot (100° to 110°F.) and cold (40° to 50°F.) water for a total of 15 minutes three times a day.

Another form of mechanical treatment was 'passive vascular exercise' or 'Pavaex' therapy. Here the leg under treatment was inserted through an airtight seal into a glass boot and subjected firstly to a negative pressure to fill the capillaries and secondly to a positive pressure to empty them. This system appears to have been developed more or less coincidentally by Landis and Gibbon (1933a and b) and Herrman and Reid (1934). The former used a negative pressure of from 80 to 120 mm. of mercury for 25 seconds followed by a positive pressure of 60 to 80 mm. for five seconds; the latter used 80 mm. negative and 20 mm. positive pressure at a rate variable from two to four cycles a minute. Reports on the success of 'Pavaex' vary from the enthusiastic to...
the very critical. Investigators include de Takats (1934), Collens and Wilensky (1936) and Conway (1936) in addition to the originators of the technique.

Surgical measures for the treatment of occlusive vascular disease have also been many and varied. Perhaps the most revolutionary was the suggestion put forward by Lewis and Reichert (1926) that the *femoral artery should be ligated* distal to the origin of the profunda before the popliteal artery became thrombosed; by this means it was considered that the collateral circulation would be increased. *Femoral vein ligation* was tried, but Ginsburg (1917), reviewing the subject, considered the results far from satisfactory. Lilienthal (1907) carried out an end-to-end *anastomosis of the femoral artery and vein*, but the patient died three days after the operation. The procedure was performed successfully by Meyer (1925) with reported good result. The rationale of arteriovenous anastomosis was considered to be invalid by Buerger (1924) on the grounds that a venous thrombosis is not uncommonly present.

The hypothesis that occlusive vascular disease might be due to an excess of circulating adrenalin (Josuè, 1903) was advanced as a reason for *unilateral adrenalectomy*. Reviewing 110 cases, Herzberg (1926) condemned the procedure.

A local vasodilatation and relief of pain as a result of *periarterial sympathectomy* was described by Leriche and Heitz (1917). Although the operation has been performed by many surgeons, the clinical results do not appear to have been successful. Brown and Rowntree (1925), in a series of 17 cases, were unable to demonstrate any vasodilatation. Allen (1930) considered that the procedure was not worthwhile on clinical grounds.

*Lumbar ganglionectomy* was advocated by Adson and Brown (1932) in cases where a preliminary nerve block resulted in a rise of skin temperature of 3°C. or greater. Good results from the operation have been recorded by Telford and Stopford (1933), Flotow and Swift (1933), Diez (1934), Atlas (1941), van Ouwerkerk (1946) and many others. Atlas (1942), however, sounded a warning note on the subject. He pointed out that in certain cases the main effect of sympathec-
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RATCLIFFE: Intermittent Claudication

Fig. 5.—Senile obliterative arteritis—Secondary popliteal thrombosis.
(By courtesy of the Editors of the 'Journal of Bone and Joint Surgery')

Itomy is to open up arteriovenous shunts in the skin with disastrous results.

Pain-relieving procedures consisting of destruction of peripheral nerves by section (Laskey and Silbert, 1933), crushing or alcohol injection (Smithwick and White, 1930, 1935) have also been described.

PART II

THE MECHANISM OF PAIN IN INTERMITTENT CLAUDICATION

Although from the time of Boullay (1831) it was realized that the underlying cause of the syndrome of intermittent claudication was arterial deficiency, the reason why this should cause pain was not clear. Various theories have been advanced from time to time:

Muscular Spasm

It was observed by the earlier clinicians that after a patient with intermittent claudication had exercised there was sometimes a palpable change in the consistency of the calf muscles. This suggested that the ultimate cause of the pain was muscle spasm. Charcot (1858) and Marinesco (1896) went so far as to compare the state of the muscle with cadaveric rigidity. Support was given to the muscular spasm hypothesis by Erb (1898). Two facts make the theory untenable; the pain does not vary with each individual muscle contraction, and the change in muscular consistency is far from constant.

Arterial Spasm

Oppenheim (1900) described cases where intermittent claudication was present without obvious arterial disease. He considered this to be due to arterial spasm. From this it was but a short step to the assumption that the pain in all intermittent claudication is angiospastic in origin, despite the physiological concept that the response to muscle exercise is vasodilatation. Veal and McFetridge (1936), by arteriographic studies, showed that exercise pain did in fact occur with the vessels dilated.

Certain anomalous cases of intermittent claudication, with disappearance of the limb pulses
after exercise, although no arterial disease was demonstrable (Pearl, 1937), are most probably due to a thrombotic lesion proximal to the femoral artery (Boyd and Jepson, 1950).

Muscular Ischaemia

Zak (1921) showed that repeated opening and closing of the hand in a normal upper extremity produced symptoms akin to intermittent claudication if the brachial artery was artificially compressed. Detailed investigation of this phenomenon was carried out by Lewis and his co-workers (1929); a review of the work was made by Lewis (1942).

The concept advanced by Lewis was that the immediate cause of the claudication pain was not oxygen lack but the accumulation of metabolites acting on sensory nerves. The particular substance responsible is designated as 'factor P.' The observations which support this idea may be summarized briefly as follows:

(a) The intensity of the claudication pain does not increase and diminish as the muscle contracts and relaxes but is a steady ache.

(b) With a test involving a standard rate of work done by normal subjects with the circulation occluded, it was found that pain began some 35 seconds after exercise started, becoming intolerable after a further minute. If the circulation is restored the pain disappears within 3 seconds; if the circulation is not restored the pain persists.

(c) If the circulation is completely occluded the onset of the pain depends on the total work done; if the rate at which the work is done is increased the time of onset of the pain is reduced in proportion. If the circulation is incompletely occluded the total amount of work required to produce pain increases as the rate of work is reduced (Katz, Lindner and Landt, 1934). The explanation is that, with the circulation only partly occluded, reducing the rate of exercise allows a greater time between muscle contractions for the metabolites to be removed.

(d) After the claudication pain has become established, if the circulation is restored for an interval just sufficient to remove the pain, and the experiment repeated, it is found that the amount of work which causes pain has been reduced.
This suggests the carry-over of metabolites from the previous experiment.

The composition of 'factor P' has not yet been determined. Katz, Lindner and Landt (1935) believe it to be a non-volatile acid. In parenthesis it may be noted that Buerger (1924) suggested that the failure to remove lactic acid from the muscle might be the cause of claudication pain.

PART III
THE WORK OF THE MANCHESTER NEUROVASCULAR TEAM

A study of the underlying causes, methods of investigation and treatment of intermittent claudication was begun in 1947 in the neurovascular clinic at the Manchester Royal Infirmary. The conclusions reached at the end of the first two years have been reported by Boyd, Hall Ratcliffe, Jépson and James (1949), together with an analysis of 276 cases followed up for a minimum period of six months. A review of this and subsequent publications is given below.

A Classification of Occlusive Arterial Disease

The arteriographic findings and clinical features in over a thousand patients with deficiency of circulation in the lower limbs investigated during the last three years have been reviewed by Boyd (1950). The arterial lesions may be classified under four main headings:

1. Traumatic thrombosis.
2. Juvenile obliterative arteritis.
3. Primary popliteal thrombosis.
4. Senile obliterative arteritis.

(1) Traumatic Thrombosis

The designation of this group is self-explanatory, the arterial thrombosis being directly due to gross trauma.

(2) Juvenile Obliterative Arteritis

Here the degenerative process begins in the small arteries of the feet (Figs. 1 and 2). Although there are quiescent periods the disease inevitably ascends the limb, eventually resulting in gangrene. The condition is bilateral, although one limb may be affected a considerable time before the other. A patchy superficial phlebitis usually precedes the arterial changes. A fungus infection of the feet is an almost constant finding. That certain fungi give rise to toxins which affect the endothelium of blood vessels has been shown by Thompson (1944) and it may well be that this is the causative factor. An unusually high occurrence of patchy superficial phlebitis and juvenile obliterative arteritis was seen among troops in Egypt under conditions where fungus infection was rampant.

A careful distinction must be made between this condition and the distal form of senile obliterative arteritis, which is clinically similar but is not accompanied by superficial phlebitis or fungus infection.

(3) Primary Popliteal Thrombosis

Arteriographic studies show that the lesion is confined to the popliteal artery (Fig. 3). The thrombus may extend from the level of the knee joint upwards to the adductor opening or downwards to the bifurcation of the artery, or occasionally in both directions. The constancy with which the thrombosis starts at the level of the knee joint suggests that repeated minor traumata may be the causative factor. Arteriograms of patients known to have sustained traumatic thrombosis of the popliteal artery from posterior dislocation of the knee joint are indistinguishable from those of primary popliteal thrombosis.

Histologically there is no evidence of inflammatory changes in the arterial wall (Boyd, 1938). The lumen is found to be occluded by healthy clot in various stages of organization.

Primary popliteal thrombosis follows a benign course and there has been no further evidence of arterial lesions in patients who have been seen by Boyd ten years after the onset of the condition. This course differs greatly from the after-histories of patients with juvenile obliterative arteritis.

In this series patients under the age of 35 fall into the categories of primary popliteal thrombosis or juvenile obliterative arteritis. It is considered that, in spite of the marked clinical difference, these two conditions have in the past been grouped together under the general term 'thromboangiitis obliterans,' although it is felt that some reported series contain a generous admixture of the distal type of senile obliterative arteritis described below.

(4) Senile Obliterative Arteritis

Over 90 per cent. of the patients in this series are included in this group. It must be emphasized that senile obliterative arteritis is not solely confined to the elderly. Atheroma has been recorded in young children, and it is far from rare in the third and fourth decades; it accounts for practically all occlusive arterial disease over the age of 40 years. Senile obliterative arteritis may be of the diffuse form; there may be secondary thrombosis of a major vessel; or the lesion may affect only the feet.

(a) Diffuse Obliterative Arteritis. Arteriography shows partial occlusion and irregularity of the main vessels and larger branches (Fig. 4). In the most common group the small branches are abundant and the collateral circulation is markedly
developed. Less frequently there seems to be a paucity of muscular branches which appear to end abruptly; the collateral circulation is poorly developed and there is pronounced muscle wasting.

(b) Secondary Popliteal Thrombosis. Before the severity of the intermittent claudication compels patients with senile obliterative arteritis to seek advice, nearly half of them have experienced a secondary popliteal thrombosis, usually associated with some definite incident of over-exertion. As in the primary type the thrombosis begins behind the knee joint and usually extends upwards to the adductor opening (Fig. 5), although sometimes it may spread downwards or in both directions.

The effect of the secondary popliteal thrombosis depends on the degree of development of the collateral circulation. If the arterial disease is of long standing the Collins are probably well developed, whereas a thrombosis occurring early in the disease is bound to lead to severe ischaemia.

(c) Secondary Femoral Thrombosis. Thrombosis of the superficial femoral artery occurred in 13 per cent of the patients with senile obliterative arteritis. The thrombosis begins in the region of the adductor opening (Fig. 6); the proximal extension is limited by the brisk flow of blood through the profunda femoris.

Clinically a secondary femoral thrombosis is to be suspected in a patient complaining of intermittent claudication in the calf and yet having an apparently healthy limb. In general the higher the arterial block the less marked are the effects on the peripheral circulation.

(d) Distal Type. The distal type of senile obliterative arteritis affects the feet and often the hands. It occurred in approximately 10 per cent of the cases in this series. Proximal spread of the lesion is uncommon and the condition rarely leads to more than superficial necrosis or the loss of the terminal phalanges of the toes.

In young people the condition can be distinguished from juvenile obliterative arteritis by the lack of patchy superficial phlebitis and fungus infection. Juvenile obliterative arteritis does not affect the hands.

In the elderly patient clinical recognition is important. The sudden appearance of ischaemic changes in the feet is usually regarded as hopeless, whereas in fact this type generally responds readily to treatment.

Investigation and Clinical Typing

Patients complaining of intermittent claudication are first seen in the Neurovascular Clinic. Investigation, therefore, was designed to enable the surgeon to decide whether treatment can be given as an out-patient or whether admission for further study is desirable. The information required consists of the clinical history and general data, the diagnosis of the nature of the causative lesion, the muscle group affected and the degree of severity of the condition.

Clinical History and General Data

Since peripheral vascular disease is part of a general degenerative process the patient must be assessed as a whole. A detailed examination of the cardiovascular system is not possible in the out-patient clinic, but where operative procedure is contemplated, is carried out when the patient is admitted.

During the out-patient investigation the data are entered on a standard pro-forma. A record is made of the location of pain, the date when it was first noticed, whether the onset was gradual or sudden; the distance walked originally and at the time of examination, whether the change was gradual or sudden; any history of injury, frostbite or phlebitis; the amount of tobacco smoked; the parents’ age and cause of death. Clinical data include general health, state of heart and blood pressure, nutrition of the limb, muscle wasting or other degenerative changes.

The Causative Lesion

Oscillometric readings are taken above and below the knee and above the ankle. The oscillometer is not a precision instrument, but it does allow assessment of main vessel changes. Oscillations are reduced in an arteriosclerotic limb and are absent below a main vessel block.

The Muscle Group Affected

The muscle group most affected by the ischaemic changes may be found from the surface representation of the claudication pain. This was worked out by Jepson by injecting the appropriate muscle group, in himself and volunteers, with 6 per cent. saline after first infiltrating the skin and deep fascia with local anaesthetic. Three reference areas were demonstrated. Injection of the gastrocnemius produced a pain in the mid-calf extending behind the knee to the back of the thigh; from the mid-calf downwards to the instep; the anterior tibial and peroneal muscles on the anterolateral surface of the leg.

Assessment of Severity: Clinical Typing

The patients’ walking ability is tested on a ‘claudicometer.’ This consists of an endless belt driven by an electric motor at a speed ranging from one to four and a half miles per hour; a tachometer gives the speed and distance traversed. The patient steps on to the belt, which is moving
towards him at the slowest rate; the speed is then adjusted until the patient considers he is walking at his normal rate. When the claudication pain begins the distance is noted by the observer. The patient is not allowed to rest, but is urged to continue walking as far as possible, meanwhile describing the subjective phenomena. The distance at which the patient eventually halts is also noted. From the data obtained it is possible to classify the severity of the condition as belonging to one of three types.

The rationale of the classification depends on a consideration of the events taking place in a muscle during exercise. The blood supply to a muscle at rest is scanty, but it increases considerably with exercise (Krogh, 1922). If the muscle is exercised under ischaemic conditions pain results (Lewis et al., 1929). Though the exact mechanism of the vasodilatation and the production of pain has not been fully described, it cannot be doubted that the initiating event is the accumulation of metabolites in the contracting muscle (Hamilton, 1947).

With continuing exercise the level of metabolites will become steady when the rate of elimination is equal to the rate of production. With normal blood supply, stabilization takes place below the threshold of pain, but in the claudicant with reduced blood supply the pain threshold is reached before stabilization. The level at which equilibrium is attained depends on the relation between blood supply and demand, and it is this level which determines the severity of the pain and the clinical type of claudication.

Type 1. In this type the blood supply and demand are about equal. Before maximum vasodilatation is achieved the pain threshold is crossed, but equilibrium is eventually reached below the threshold. On walking, the Type 1 claudicant complains of the onset of pain, but on continuing to walk he announces, usually with some surprise, that the pain has disappeared. That the equilibrium is just below the pain threshold can be demonstrated by increasing the rate of walking above the normal, causing the pain to return.

Type 2. Here equilibrium is reached above the threshold of pain. On continuing to walk after the onset of the claudication the patient says that the intensity of the pain remains much the same. Eventually he stops walking, not because of any increase in intensity but because of the persistence of the pain. Naturally the patient with the less intense pain tends to walk further than the patient with the more severe pain.

Type 3. Equilibrium is never reached with this type; the deficiency of blood supply is such that the pain becomes intolerable before that point. The Type 3 patient, on being urged on after the onset of claudication, states that the pain is growing worse, and he halts a short distance further on, obviously in extreme pain.

Treatment

Lack of knowledge of the etiology of arterial disease limits the treatment of intermittent claudication to alleviation of the subjective manifestations which interfere with the daily life of the patient. As the pain is due to an imbalance between blood supply and demand the logical treatment would be to increase the blood supply to the required level or, if this is not possible, to reduce the demand. In practice the problem is more complex; many factors must be considered and each case assessed individually.

Factors Influencing the Choice of Treatment

(a) Clinical Type. Failure to recognize that there are different grades of claudication has made the assessment of treatment virtually impossible. The general principles of treatment indicated in each clinical type can, however, be defined clearly. In Type 1 patients blood supply and demand are very nearly equal. The slightest increase in blood supply brings about complete relief from claudication, and it appears that additional exercise will do this. In consequence the patient will respond to any treatment in which he has confidence. The gap between supply and demand is greater in the Type 2 patient. Generally the supply can be increased to meet the demand, though in the more severe cases the demand may be reduced. In the Type 3 patient the supply cannot be increased to bridge the gap and relief from pain can be secured only by reducing the demand.

(b) Age. The younger the patient the more necessary it is to secure the greatest possible increase in blood supply, not only to improve the nutrition of the limb but also to retard degenerative changes and postpone the need for amputation.

(c) Type of Arterial Disease. With diffuse obliterator arteritis and calcification of the main vessels, loss of life through coronary thrombosis or cerebral complications is more likely than loss of the limb through gangrene. It is felt that sympathectomy should only be advised in this condition when there is good reason to believe the limb to be in danger.

Patients with a main vessel thrombosis, especially the superficial femoral, are prone to further massive thrombosis and gangrene. An effort to improve the blood flow is strongly advocated in Type 3 patients in these groups, together with measures for the relief of pain, and in all patients with superficial femoral thrombosis.

(d) Associated Conditions. In patients past middle-age, arterial lesions resulting in inter-
mittent claudication are often only part of a generalized degenerative change. Apart from general conditions contra-indicative of a particular treatment, it is advisable to ascertain that claudication really is the limiting factor. It is unwise, for instance, to carry out an operation for the relief of claudication if the patient is brought to a standstill by osteoarthritis of the knee joint.

Diabetes, unless mild and well controlled, accounts for rapid progress in arteriosclerotic changes and in the development of peripheral gangrene and must be excluded before decisions are made as to treatment.

Methods of Treatment

Only those methods of treatment whose success has justified their retention are reviewed. For others which were tried and abandoned, reference should be made to the original papers.

Assessment of results has been made on the patient's ability to walk a minimum of half a mile; beyond this distance he is classed as improved, short of it as unimproved. This method has been adopted on the grounds that a patient's walking distance may be doubled—e.g. from 100 yards to 200 yards—and still not be of economic value. Assessment has been limited in this review to those patients for whom the procedure was undertaken with a view to relieving their claudication. For example, the results of lumbar ganglionectomy are reviewed on Type 2 patients, although it was also performed on Type 3 patients with poor limb nutrition.

Lumbar Ganglionectomy. The quickest and most certain method of improving the blood supply to the limb is lumbar ganglionectomy. Release of normal tone increases the blood flow even if there is no evidence of abnormal vasoconstriction. In order to be sure of denervating the whole limb it is necessary to remove the first three lumbar ganglia and the intervening chain (Hall Ratcliffe and Jepson, 1950). Of 52 Type 3 patients in this series, 47 were improved by the operation.

Paravertebral Block with 10 per cent. Phenol. In patients in whom lumbar ganglionectomy was contra-indicated on the grounds of age or general condition, chemical destruction of the ganglia has proved of value. The method advocated by Haxton (1949) has been employed. Of 18 Type 2 patients, 14 were improved by this method.

Vitamin E (α-tocopherol) Therapy. α-tocopherol therapy has been employed in cases where operative procedures were considered inadvisable. Patients were given daily doses of synthetic α-tocopherol (Ephynal, Roche). A controlled series has been reported by Hall Ratcliffe (1949). Of 41 Type 2 patients fulfilling the experimental criteria, 34 were improved, while of 25 controls only 5 were improved.

Tenotomy of the Tendo Achillis. This procedure has been carried out in Type 3 patients in whom the reference areas of the pain show beyond doubt that claudication in the gastrocnemius or soleus is the limiting factor. The disability is slight and in most cases the gait is improved; the slight limp caused by the tenotomy replaces the severe limp of the claudication.

The operation was performed on 18 Type 3 patients and all were improved.

Division of the External Popliteal Nerve. Three Type 3 patients had no calf pain, the reference area being that of the anterior tibial-peroneal group. The external popliteal nerve was infiltrated with 2 per cent. novocain, after which the pain was relieved and all were able to walk more than half a mile in comfort. They each agreed that the foot-drop was infinitely preferable to the claudication pain and that the nerve should be divided. This was done and so far they have remained satisfied.

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LOXTON: Advances in Treatment of Rheumatoid Arthritis

RECENT ADVANCES IN THE TREATMENT OF RHEUMATOID ARTHRITIS

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Since the announcement by Hench and his collaborators at the Mayo Clinic that cortisone (17-hydroxy-11-dehydrocorticosterone, Compound E) and ACTH (pituitary adenocorticotrophic hormone) can produce dramatic improvement in patients suffering from rheumatoid arthritis, the medical and lay press have shown a burst of interest in the treatment of this tragic and ancient disease. The general physician and endocrinologist, who in the past were often loath to admit cases to their beds, now all seem keen ‘to have a go.’ It is therefore important that the problems arising from these new forms of treatment should be viewed with a proper sense of perspective. To do this a brief description of modern views on the aetiology, diagnosis and natural history of the disease is necessary. We must also reconsider our methods of assessing improvement and cure.

Aetiology

The cause of rheumatoid arthritis is unknown, though most workers agree that heredity and physical or emotional strain may be contributory factors. The disease is commoner in women than in men, in the poor than in the rich, in cold wet climates than in warm dry climates. We shall discuss not only these factors, but also the following suggested causes: focal sepsis, bacterial or virus infection, bacterial or virus sensitivity, vitamin deficiency, endocrinological factors and the general adaptation syndrome of Selye.

Focal sepsis if always sought is not commonly found. Finding and clearing it rarely cures. It cannot therefore be regarded as a constant aetiological factor.

The disease has many of the characteristics of a chronic infection, fever, malaise, wasting and a