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AETIOLOGY AND MANAGEMENT OF LESIONS OF THE FEET IN DIABETES

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It is generally held that there is an association between diabetes and arterial disease. Joslin *et al.* (1940) state that "arteriosclerosis is excessive and has become the major cause of death among diabetics generally," and this view has many supporters. Opinion, however, differs on the nature of the arterial lesion, which is regarded by some—for example, Warren and Le Compte (1952), Barach and Lowy (1952), and Katz and Stamler (1953)—as atherosclerosis, and by others—for example, Lundbaek (1954)—as a specific form of angiopathy associated with long-standing diabetes.

Histologically it would appear to be impossible to differentiate between non-diabetic and diabetic arterial disease, and, although Lundbaek and Posborg Petersen (1953) have found statistically significant differences in the calcium, cephalin, and lecithin content of the coronary arteries in non-diabetic and diabetic subjects, these differences are admitted by them to be small. Lundbaek bases his belief on the existence of specific diabetic angiopathy largely on statistical grounds, sex incidence, and the presence of such associated lesions as retinopathy and the renal lesions described by Kimmelstiel and Wilson (1936).

Object of the Survey

The present investigation is particularly concerned with peripheral vascular disease of the lower extremities, and was carried out with the prime object of increasing our knowledge and understanding of the aetiology and treatment of lesions of the feet in diabetes; the results obtained, however, may have some bearing on the wider issue of the nature of the arterial lesion in diabetes.

In a recent survey of patients attending the diabetic clinic at King's College Hospital during the period 1950 to 1953, one of us (M. M. M.) found symptoms and signs of occlusive peripheral vascular disease affecting the lower extremities in 146 out of a total of 3,788.

These 146 cases have been analysed in order to determine the relationship between this condition and age, sex, and duration of diabetes.

Table I shows that symptoms and signs of peripheral arterial disease of the lower extremities were entirely

absent below the age of 40, and that there were only three cases between the ages of 40 and 49; thereafter the incidence rose sharply, being greatest above the age of 60. The same trend was observed in both sexes, although the incidence was greater in men.

TABLE I.—Incidence of Peripheral Occlusive Arterial Disease in Diabetes with Respect to Age and Sex

Age Group	Males per 100 Diabetics	Females per 100 Diabetics	Ratio Male/Female
20-29 ..	—	—	—
30-39 ..	—	—	—
40-49 ..	2	1	2:1
50-59 ..	4	2	2:1
60-69 ..	11	4	2.75:1
70+ ..	23	5	4.6:1

These results expressed as a percentage incidence of the diabetic population attending the clinic show that peripheral arterial disease was twice as common in patients of 70 and over as it was in the 60 to 69 age group.

Table II shows the relative sex incidence of occlusive vascular disease; it will be seen that male diabetics were much more commonly affected than females, especially in the older age groups.

TABLE II.—Age and Sex Distribution of Peripheral Occlusive Arterial Disease in a Population of Diabetics Attending King's College Hospital

Age Group	Male Distribution		Female Distribution		Total		Incidence in Diabetic Population
	Arterial Disease	Diabetic Population Used	Arterial Disease	Diabetic Population Used	Arterial Disease	Diabetic Population	
20-29 ..	—	186	—	221	—	407	—
30-39 ..	—	222	—	203	—	425	—
40-49 ..	4	227	2	226	6	453	1.3%
50-59 ..	11	280	9	531	20	811	2.5%
60-69 ..	31	292	28	762	59	1,054	5.6%
70+ ..	36	159	25	479	61	638	10.5%
Total ..	82	1,366	64	2,422	146	3,788	3.9%
Percentage ..	56	36	44	64			

Peripheral Arterial Disease

It is significant that the incidence of symptoms and signs of peripheral arterial disease (Table III) appears to bear no relationship to the duration of the diabetes, the number of patients being approximately the same in all groups.

TABLE III.—Incidence of Peripheral Occlusive Arterial Disease in Diabetics with Respect to Duration of Diabetes

Duration of Diabetes in Years	No. of Patients
Less than 1	20
1-4	26
5-9	29
10-14	25
15-19	26
20 and over	20
Total	146

In a recent follow-up of some 250 diabetics in whom the disease began before the age of 15 and in whom it had been present for not less than 10 years, and in many for 20 or more, one of us (W. O.) was unable to find a single lesion attributable to occlusive vascular disease; this, of course, does not exclude the presence of such disease. In the great majority of these young diabetics the disease was severe and its control variable. In short, old age seems to be the common factor in these cases, and the duration, severity, and control of the diabetes of secondary importance. It would not, however, be true to say that lesions of the feet occur only in diabetics of 60 or over. Sepsis, perforating ulcers, and even gangrene are by no means uncommon below this age, but it has been assumed too readily that such conditions are due to peripheral arterial disease alone or combined with a lowered resistance to staphylococcal infection. Careful examination shows that the great majority of these comparatively young patients have a quite adequate blood supply as judged by peripheral arterial pulsation, oscillometry, and skin temperature tests, the common defect being diabetic neuropathy.

The importance of neuropathy in this connexion became apparent as a result of a detailed investigation of the incidence and manifestations of neuropathy in the diabetic department of King's College Hospital (Martin, 1952), in which there was observed to be a high incidence of septic and apparently ischaemic lesions in patients with severe neuropathy. Neuropathy, unlike retinopathy and most other diabetic complications, was found to be related less to duration than to control of the diabetes: this led to a reassessment of our older patients in whom lesions of the feet were often associated with diabetes of comparatively short duration. To our surprise we found that in most cases the increased incidence of lesions of the feet in diabetics could be explained by the superimposition of neuropathy on the degree of arterial disease common to non-diabetics of the same age and sex; the appreciation of this is essential, in our opinion, to a proper understanding of the aetiology and treatment of the diabetic foot.

Nature of Diabetic Neuropathy

In order to appreciate the part played by neuropathy in the production of lesions of the feet in diabetes, it is essential to understand something of the nature and manifestations of diabetic neuropathy.

Diabetic neuropathy is a degenerative condition of peripheral nerves occurring as a complication peculiar to the diabetic state. It affects mainly the nerves to the lower extremities at their periphery and is much more common than the published reports suggest.

Peripheral nerve involvement, although occurring in diabetics of all ages, is more commonly seen in the elderly, in whom the insidious onset of diabetes in middle and later life often results in long delay in diagnosis and treatment.

Neuropathic lesions of the feet are among the common complications for which such elderly diabetics may first seek medical advice.

Severe diabetic neuropathy, with extensive sensory loss and muscular weakness, may not be a frequent finding in smaller diabetic clinics, but less severe involvement of the nervous system characterized by pain, paraesthesiae in the feet and hands, calf tenderness, and vasomotor disturbances are commonly encountered. Although neuritic symptoms in the absence of physical signs have been regarded as distinct from diabetic neuropathy with its more permanent findings of peripheral nerve damage (Rundles, 1945), there is little doubt that they have a common aetiology (Martin, 1952).

The earliest phase in the development of diabetic neuropathy, characterized by diffuse and ill-localized pain, vasomotor disturbances, thermal paraesthesiae, changes in the tendon reflexes, and often alteration in pain sensibility in the feet without much impairment of perception of touch or pressure, is due to widespread degeneration of non-myelinated nerve fibres (Martin, 1952). The impairment of autonomic vasomotor responses and, to some extent, pain conduction due to interruption of the lower sensory neurone predisposes to excessive trauma and lowers tissue resistance (Lewis, 1927). Thus, even when the blood supply to such an area is intact, its tissues may be regarded as relatively ischaemic so far as the local defence and repair are concerned; this is the minimum degree of ischaemia which can be recognized clinically (Learmonth, 1953).

There is now a good deal of evidence that non-myelinated nerve-fibre degeneration occurs in a high proportion of diabetic patients, who, on clinical examination, show no evidence of "clinical" diabetic neuropathy (Martin, 1953). The presence in diabetics of such nerve-fibre disturbance explains the frequent finding of persistently cold feet and their proneness to traumatic lesions.

Cutaneous neuropathic changes commonly start as blisters about the tips of the toes or at the site of a corn or callosity in places constantly exposed to irritation by an ill-fitting shoe. Frequently the deceptive lack of normal sensation, and particularly pain sensation, leads the patient to ignore the lesion and delay treatment until secondary infection with deep penetration of the tissues or severe inflammation is present. The blood supply in many of these cases is quite adequate, and suitable local treatment, combined with good control of the diabetes, usually results in normal healing.

Classification of Lesions of the Feet.—For purposes of description it is convenient to divide lesions of the feet in diabetics into the following types (Oakley, 1954): (1) septic, (2) neuropathic, (3) ischaemic, and (4) combinations of 1, 2, and 3.

1. Septic Lesions

Uncomplicated sepsis produces essentially the same lesion in the diabetic as in the non-diabetic foot, and, apart from control of the diabetes, the treatment is the same, the chief measures being rest, drainage, and antibiotics.

2. Neuropathic Lesions

Loss or impairment of superficial sensation, and especially the appreciation of heat and pain, are important factors in the production of callosities, ulcers, and local gangrene. Loss of deep sensation may cause Charcot joints, while weakness of the intrinsic muscle may produce characteristic deformities which not only favour the formation of chronic ulcers but also add to the difficulties of surgical treatment.

A considerable number of diabetics complaining of their feet prove, on inspection, to have varying degrees of a quite typical deformity. The toes are held dorsiflexed at the metatarso-phalangeal joints with flexion at the inter-phalangeal joints, but these claw toes are never accompanied by the tight plantar fascia and short tendo achillis of fully developed pes cavus (Fig. 1). This deformity is often met with in the presence of an apparently normal circulation, but

is constantly associated with signs of neuropathy. An excessive keratinization of the horny layer of the skin is also usually present, although these last changes are minimized by regular attention. The cocked-up position of the toes is that into which they would be drawn by simultaneous contraction of the long flexors and extensors with the normal balancing force of the intrinsic muscles removed. The association of this deformity with sensory disturbances lends colour to the belief that this deformity is indeed a manifestation of a peripheral motor neuropathy. Proof of this

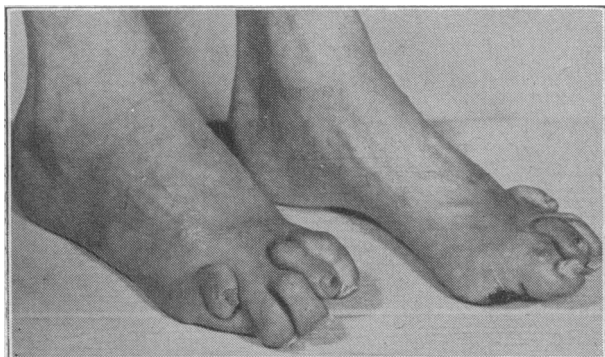


FIG. 1.—Typical deformity of toes associated with diabetic neuropathy.



FIG. 2.—Pressure gangrene of big toes associated with diabetic neuropathy.



FIG. 3.—The same patient as in Fig. 2 after conservative treatment.

contention is difficult, since the established deformity is often so stiff that electromyography is useless, and biopsy, for obvious reasons, is not lightly undertaken.

Such a foot produces symptoms sometimes attributable to ruthless shoe-pressure on a numb foot. Areas of superficial necrosis can appear with dramatic suddenness, but, provided further pressure is avoided, usually heal uneventfully (Figs. 2-3); in the case of the decubitus ulcer on the heel the patient should be confined to bed and the limb suspended until healing is well advanced.

In the presence of stiff and deformed toes the absence of the normal heel-and-toe movements in walking places

an abnormal thrust on the metatarsal heads, and the underlying neuropathic skin passes inexorably through the stage of ugly callosities to the formation of indolent ulcers (Fig. 4).

Treatment

These changes are only hastened by the use of appliances which, unable to affect the shape of the foot, become mere space-occupying lesions in the shoe. It cannot be emphasized too strongly that the use of any appliance inside the shoe in the presence of stiff toes will precipitate the very disasters it is intended to prevent. Appeasement can be obtained only by shoes made to measure, although a rocker bar on the sole may help in mimicking a normal heel-and-toe movement.

Once deep perforating ulceration has occurred, surgical intervention is essential. The object of the operation is to remove the underlying bony pressure point and obtain skin cover without tension. Suture of the wound is permissible only when there is ample skin cover, no sepsis, and an adequate blood supply as judged by the haemorrhage at operation. Fortunately placed incisions, however, with or without primary Thiersch grafting, will allow rapid healing provided that the bone section has been radical enough. The problems presented by the neuropathic foot rarely carry an immediate risk to life, and consideration must be given to the long-term effects of the bone section proposed, since removal of more than one of the three middle toes will always result in further deformity, with the formation of new sites of pressure. If more than one toe has to be removed amputation of all five toes at once leaves a more shapely foot with a far better prospect of useful life. The "moccasin" prosthesis described by Downie (1956) seems to solve the problem of the aftercare of these feet.

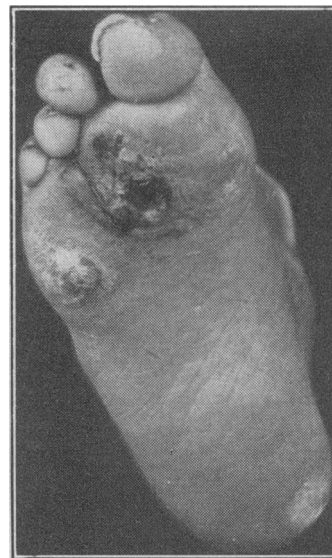


FIG. 4.—Perforating ulcers of sole of foot in diabetic neuropathy.

3. Ischaemic Lesions

The generalized ischaemic lesion unassociated with neuropathy is not so common as is generally supposed, and must be differentiated from the local ischaemic disaster in an essentially neuropathic foot. The recognition of ischaemia is based on the history, clinical examination, and special tests. Pain of a burning character, worse at night and aggravated by heat, is suggestive of ischaemia but by itself does not exclude neuropathy; associated intermittent claudication greatly favours a diagnosis of ischaemia. The foot is typically cold and may be pale or pink, the latter colour being the more often associated with pain. The skin is often smooth, hairless, and shiny, and there may be trophic changes in the nails. Arterial pulsation may be absent at any level below the femorals, but inability to palpate posterior tibial and dorsalis pedis arteries does not necessarily mean that the blood supply to the foot is seriously inadequate. The intelligent use of the oscillometer is often helpful in this connexion, and, although it gives no information about the blood supply below the ankle, it is of considerable value in assessing the level of arterial patency above this level. Skin temperature tests are difficult to interpret, but, as one of us (M. M. M.) has shown, when used in conjunction with intravenous vasodilators such as

tolazoline hydrochloride they may be of considerable value in differentiating between arterial occlusion and vasomotor disturbances associated with neuropathy.

X-ray films of the lower limbs for arterial calcification are of little value in estimating arterial sufficiency, but arteriography provides more accurate and precise information than any other single test; it is, however, not readily available in the majority of cases, and is a somewhat specialized technique. The diagnosis of ischaemia can usually be established without the use of this test.

The two main manifestations of ischaemia in the diabetic foot are pain and gangrene; pain may be associated with gangrene or occur independently as the painful ischaemic foot.

Painful Ischaemic Foot.—This clinical condition is almost confined to the old, and usually affects both feet, although not necessarily to the same degree. The affected foot is typically cold, pink, and intensely painful, especially in bed at night. Gangrene of one or more toes may supervene, but is often absent or long delayed.

Treatment

Treatment is most unsatisfactory; vasodilator drugs are ineffective, the only conservative measures of any value being local cooling, analgesics, and hypnotics. Sympathectomy seems to relieve the pain in some cases, but amputation is the only reliable method of treatment, and is often carried out at the request of the patient.

Gangrene

True ischaemic gangrene usually starts in the distal phalanges of one or more toes and is often painless. Purple discoloration leads to the formation of a terminal black patch which tends to extend proximally, and, so long as there is no infection, causes little discomfort to the patient; severe pain is suggestive of a superadded infection. The lesion may remain localized or may, if untreated, spread so as to involve the skin of the dorsum of the foot; when this occurs local surgical treatment must give place to more radical amputation. The single dry gangrenous toe presents a serious problem, and the decision must be made whether to leave the gangrenous area to separate or to remove the toe.

In our experience the former policy carries with it certain definite disadvantages and dangers. In the first place, the length of time required for separation is always a matter of weeks and often months; secondly, there is the risk of extension of the lesion on to the dorsum of the foot; and, last but not least, infection may supervene. Furthermore, if the patient is confined to bed there is the very real possibility that the combination of pressure and ischaemia may result in ulceration of the heels, while the practice of allowing the patient to be up and about exposes the affected area to the danger of trauma and sepsis. For these reasons it is usually wise, in our opinion, to remove the single gangrenous toe by disarticulation, the wound being left open and allowed to granulate; primary suture is undertaken only if the bleeding at operation indicates the presence of a reasonably good blood supply and if the skin edges can be approximated without tension. In this connexion special mention must be made of ischaemic lesions situated over the heads of the fifth metatarsal; these are commonly due to pressure from tight-fitting shoes.

Such lesions present a special problem, as amputation of the fifth toe together with the head of the metatarsal bone often leaves a raw area for which it is impossible to provide any adequate skin cover, the patient being left with a chronic ulcer for which the only effective treatment is amputation of the leg. It is in such lesions that it is vitally important to distinguish between ischaemic and neuropathic ulceration, for in the latter the blood supply is often good enough to allow of the raw area being covered by a skin graft. In the case of the big toe, skin cover presents less difficulty, as it is usually possible to preserve a flap of skin from the

surface of the toe, which can be used to cover the site of amputation. When more than one toe is involved it is generally better to amputate all five toes by separate racket incisions, the resulting foot being functionally sound and rarely a source of further trouble. When the area of gangrene extends proximally beyond the toes on to the dorsum of the foot, local amputations are likely to prove unsuccessful, and amputation of the leg is the only reliable method of treatment. Various types of transmetatarsal and transtatarsal amputations have been advocated as a means of avoiding amputation of the leg.

Such operations, in our experience, have seldom proved successful, the reason being that, in order to obtain a functional stump, closure of the wound is essential; such closure can very rarely be made without tension or the risk of tension from post-operative haematoma or sepsis. For some years it has been our practice at King's College Hospital to carry out such amputations below the knee. This operation has almost completely replaced the older practice of amputation above the knee in the treatment of diabetic gangrene, and represents one of the most important advances in the surgical treatment of this condition.

Amputation in the Diabetic

Before describing the technique for below-knee amputation, laid down by Silbert (1944), certain general rules of procedure for amputations in the diabetic subject are worthy of consideration.

(a) Expert general anaesthesia is essential, local analgesia being clearly impossible; we have had no experience of freezing in this connexion, but consider it contraindicated by the devitalized state of the ischaemic tissues. Many patients are bad operative risks and so demand the highest anaesthetic skill.

(b) No sort of tourniquet should ever be used on these limbs at any level or in any circumstances.

(c) Pressure points must be protected before, during, and after the period of anaesthesia. The skin of these avascular limbs is very fragile, and the disaster has befallen us of having a patient submit to amputation only to find, on recovering consciousness, that his hitherto good foot had a black heel. All bony points must be supported on a soft foam-rubber pillow throughout the whole period of narcosis; the ordinary "sorbo" mattress on the operating table is not soft enough.

(d) Tension must be avoided during and after operation. This is the only key to success, and conditions the entire design and performance of the operation. We are convinced that it is tension, often due to over-ambitious attempts at skin suture, which precipitates the so-called post-operative spread of gangrene; such tension must at all costs be avoided.

Below-knee Amputation

In the past eighteen months 50 below-knee amputations have been carried out by Silbert's technique in the diabetic department at King's College Hospital without a single re-amputation or a single death attributable to operation.

The operation is done through a circular incision 7 in. (18 cm.) below the knee. The skin is reflected up very gently for 2 in. (5 cm.) and the muscles are divided by a guillotine incision at this level. Blood vessels are ligated and the dissection continued in the subperiosteal layer until a 4-in. (10-cm.) tibial stump is obtained; the fibula is divided at a slightly higher level. The anterior border of the tibial stump is bevelled, and the soft tissues are allowed to fall down over the cut ends of the bones. The stump is bound up in gauze impregnated with petroleum jelly, the skin edges being turned in over the raw area, and the limb supported on a plaster trough extending well up the thigh. On the tenth day the patient is given an anaesthetic, and the stump is inspected; if there is any doubt about sepsis, healing is allowed to take place by granulation, but if, as usually happens, all is well, a delayed primary suture is carried out. Healing may then be expected in about three weeks.

4. Combinations of 1, 2, and 3

The coincidence of sepsis, neuropathy, and ischaemia presents an urgent and desperate problem which merits special consideration.

The sepsis usually arises in the forefoot where shoe-rubbing is most marked, and, since treatment by rest will involve putting the patient to bed with the foot supported on slings to prevent skin necrosis of the heel, gravity encourages the natural tendency of the infective process to spread into the hindfoot. In the surgical treatment of such an abscess really radical incisions arranged to divert the gravitational flow on to the skin of the sole are essential, and enough bone must be removed to allow the edges of the wound to fall together without tension. In our experience the filling-in of large dead spaces by granulation rarely occurs on account of the ischaemia, and the only hope of preserving the foot lies in obtaining early healing of the skin with an absolute minimum of raw area. We have also found that, if the primary drainage, although apparently satisfactory, proves within a few days to have been inadequate, a useful result seldom follows a second attempt at

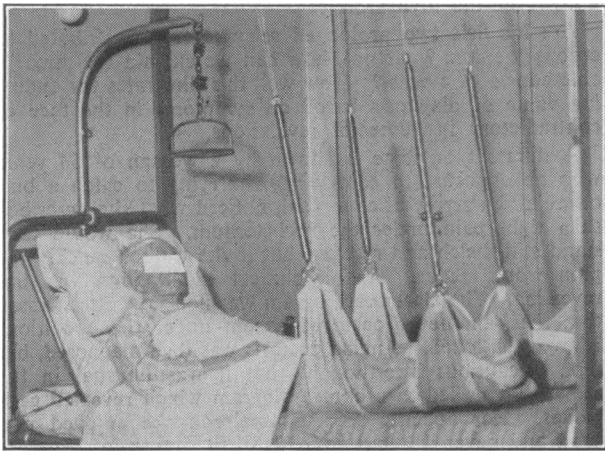


FIG. 5.—Spring suspension of lower limbs.

local drainage. Under these circumstances we are convinced that, if there is not good evidence of healing within ten days, a below-knee amputation should be advised to save the patient an indefinite period of pointless suffering. The treatment of suppurative tenosynovitis or web-space infection is so disappointing that signs of sepsis in an ischaemic toe should be regarded as a most urgent indication for local disarticulation; optimistic expectancy will often cost the patient his foot.

Whether the condition be due to neuropathy, ischaemia, or to a combination of these two factors, there is always the danger that, with the patient in the supine position, pressure on the heels may cause blistering, ulceration, or gangrene. For many years this complication has constituted a serious problem in the treatment of lesions of the feet in diabetics, and not a few limbs have had to be amputated on this account. For the past two years it has been our practice to suspend the lower limbs of all such cases (Oakley, 1954), and as a result we have not since had a single serious heel lesion in any patient. Each limb is swung a few inches above the mattress by means of two long springs, such as are used by the physiotherapists, the tension of the springs being 30 to 50 lb. (13.6 to 22.8 kg.), according to position and weight of the limb (Fig. 5). The slings are specially made of canvas lined with orthopaedic felt; a strap attached to the distal sling is passed round the sole of the foot to prevent the development of an equinus deformity. We regard this as a major advance in the management of diabetics with neuropathic and ischaemic lesions of the feet, and believe that this type of suspension should be used as a routine in the treatment of all such cases.

Discussion

From a long and wide experience of the treatment of lesions of the feet in diabetics we are convinced that the most important factors are the age of the patient, neuropathy, and ischaemia; sepsis is usually a complicating factor, and, in the absence of neuropathy or ischaemia, never gives rise to gangrene. Gangrene itself is of two types: one due to a localized arterial thrombosis resulting from trauma in a foot desensitized by neuropathy, and the other to generalized occlusive arterial disease; this second type may or may not be associated with neuropathy, and sepsis may complicate both types.

When the foot lesion is the result of neuropathy, and there is an adequate blood supply to the foot as a whole, local surgical measures with or without skin grafting give excellent results; when, however, there is generalized occlusive vascular disease, local surgery is attended by the serious risk of chronic ulceration, and more radical surgery, such as below-knee amputation, is likely to be required to cure the condition. The close relationship between age and the incidence of foot lesions in the diabetic, and the absence of any such relationship between duration and severity of diabetes, would appear to cast doubt on the generally accepted view that diabetes is itself a common cause of occlusive peripheral arterial disease. The high incidence of neuropathy seems to us to explain, in part at any rate, why lesions of the feet are more common in diabetics than in non-diabetics of comparable age; the susceptibility of the poorly controlled diabetic to staphylococcal sepsis may also be of some importance in this connexion.

Summary

The incidence of lesions of the feet in the diabetic population attending King's College Hospital Diabetic Clinic has been related to sex, age, and duration of diabetes.

Attention is drawn to the importance of neuropathy, alone and in association with ischaemia, as a factor in the production of localized ulceration and gangrene.

The treatment of lesions due to neuropathy and ischaemia is described, and the value of such operations as removal of all five toes and below-knee amputation are emphasized.

A method of protection of the heels in patients confined to bed is described and illustrated.

Reasons are given for doubting the validity of accepting the high incidence of so-called vascular lesions of the feet in diabetes as evidence that this disease by itself commonly causes peripheral occlusive vascular disease.

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