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MORTON'S DISEASE

A Clinical and Patho-anatomical Study

By

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HISTORICAL SURVEY

By Morton's disease is meant severe throbbing, burning pain and tenderness on the plantar surface of the foot, usually over the 3rd and 4th metatarsophalangeal joints, with pain radiating along the respective toes. The pain occurs when the foot is in use, e.g. when walking, and diminishes when the foot is at rest and the shoe is removed.

In 1876 *T. G. Morton*, whose name the disease bears, described the condition as "a peculiar and painful affection of the 4th metatarsophalangeal articulation". In Morton's opinion the symptoms were due to compression of branches of the external plantar nerve running to the lateral surface of the 4th toe, between the head of the 5th and the neck of the 4th metatarsals. He observed this painful condition in 12 patients, of whom 11 were women. In 3 he achieved complete freedom from pain by resecting the 4th metatarsophalangeal joint.

Tubby (1896), and *Tubby* and *Jones* (1898) described the disease very carefully. They classified it into three grades according to the severity of the symptoms. The most severe grade was treated by resection of the metatarsal head. In two of their operated cases the nerve was inspected, and showed

[1] Read at the *Nordisk Ortopedisk Forening*'s meeting in Helsingfors, June 1949.

"a well marked state of neuritis". They believed that the disease was due to pressure on a communicating nerve running between the branches of the lateral and medial plantar nerves, which run under the head of the 4th metatarsal. They also believed that the heads of the 1st and 4th metatarsals are subject to considerable pressure when the foot is in use.

Excision of the metatarsal head has also been recommended by other authors.

Morton's disease has been regarded by many as a condition caused by an abnormal strain on the anterior arch; they have cured the pain by non-surgical reconstruction (arch supports and orthopedic footwear). (*Pollosson* (1889), *Woodruff* (1890), *Bradford* (1891), *Guthrie* (1892), *Gibney* (1894), *Bose* (1894), *Goldthwait* (1894), *Lamacq* (1896), *Hohman* (1948) and others).

At one time it was supposed to be caused by inflammatory changes in the metatarsophalangeal joint, bursae or ligaments. (*Frank* (1904), *Hertzler* (1926), *D. J. Morton* (1935) and others).

In 1893, *Hoadly* gave a detailed account of 6 cases of metatarsalgia. In one of these, which had not responded to conservative treatment, he exposed the digital branches of the lateral plantar nerve of the 4th toe and found a small neuroma. He resected the nerve. A promt and complete cure was obtained.

Hoadly's observations do not seem to have aroused great interest amongst his contemporaries, however, for it was not until 40 years later, in 1935, that resection of the nerve was again advised as a treatment for *Morton's disease. Sir Harold Stiles,* the English surgeon, was operated on for long-standing metatarsalgia. The nerve between the 3rd and 4th toes was resected.

After *Betts's* investigation (1940), resection of the nerve became the generally accepted procedure. He was the first to make a histological examination of the resected nerves; he examined nerves from 10 cases. *Betts* interprets the disease in the following way. "The fourth nerve is formed by the internal plantar, with a communicating branch from the external

plantar, each coming round from opposite sides of the belly of the flexor brevis and crossing obliquely before they unite. Two to three centimetres distally the nerve divides, to pass to the adjacent sides of the 3rd and 4th toes. The nerve lies immediately on the transverse ligament, which is a very firm structure". When the foot is in action, the flexor brevis contracts and the nerve is stretched. The thickening of the nerve is due to chronic irritation. Histologically, *Betts* found "a great increase in the fibrous tissue elements of the nerve".

In 1943, *McElvenny* made a histological examination of five of twelve resected nerve specimens and said that "they appeared to be either neurofibromata or angio-neurofibromata".

Baker and *Kuhn* (1944) abolised the pain in 14 cases by resection of the nerve. The histological picture was said to resemble that of an amputation neuroma.

King (1946) made a histological examination in five cases. He suggested the term "sclerosing neuroma" and drew an analogy with keloid. He attributed the condition to chronic trauma.

Bickel and *Dockerty* (1947) operated on 18 cases, and gave a detailed patho-anatomical report. They found oedema, fibrosis, demyelination and cyst formation in the nerves, and endarteritis in the vessels. Further they found a correlation between the length of the history and the severity of the changes. They say, "There is considerable evidence that trauma from weight-bearing in small or otherwise ill-fitting shoes is responsible for the pathogenesis of the lesion".

Nissen (1948) examined 27 cases. He rejected chronic trauma of the interdigital nerve as the cause. He believed that "the primary lesion is one of local vascular degeneration leading to a variety of changes in and around the cutaneous nerve. An ischaemic lesion".

THE AUTHORS' MATERIAL

A. Clinical Data.

The material comprised 17 patients, 13 women and 4 men. In 16 the condition was unilateral, and in 1, which in this series has been counted as two cases, bilateral, thus giving 18 cases in all. In all cases the interdigital nerve was resected and examined patho-anatomically.

The patient's age at the time of operation is shown in tables 1 and 2: 12 patients, i.e. two-thirds of the material, were aged between 30 and 42 years. The remaining 6 cases were between 42 and 58 years. The youngest patient was aged 30 years.

TABLE 1
Cases of Morton's Disease.

Sex	Age	Past history	Fibrosis	Endarteritis
F	34	36 months	0	0
F	34	24 „	(+)	
F	58	48 „	+	
F	52	456 „	+	
M	54	12 „	+	
F	52	7 „	+	+++
M	40	84 „	+	
F	36	12 „	+	(+)
F	57	60 „	++	+++
F	42	120 „	++	
F	37	36 „	++	+++
F	36	4 „	++	+++
F	31	60 „	++	++
F	46	36 „	+++	+
M	36	3 „	+++	+++
M	36	120 „	+++	+++
F	30	12 „	+++	+++
F	42	96 „	++++	

Length of history (see table 1). Only 3 patients had had symptoms for less than 1 year. 8 patients had had symptoms from 1 to 4 years; 6 for over 5 years. The longest history was found in a patient aged 58 years, who had had symptoms for 38 years.

Occupation. 10 of the female patients were housewives, 1 was a laundress and 2 did no work at all. 2 of the men were factory hands, 1 was in the fire brigade and 1 was a teacher.

1 patient attributed the condition to a distortion of the foot, which had been treated for two months in plaster-of-paris, and 1 to a fracture of the 5th metatarsal which had also been treated for two months in plaster-of-paris. In both cases the symptoms developed as soon as the plaster-of-paris was removed. 2 patients attributed the condition to advanced pregnancy.

Symptoms. 14 patients complained of aching and burning pain between the 3rd and 4th toes: 2 of similar pain between the 2nd and 3rd toes: and 2 of pain between the 4th and 5th toes. The pain always radiated to the adjacent toes, and, in two cases also up the leg. Several patients complained of numbness and a feeling of cramp in the affected toes. Usually the patients described the sensation as like stepping onto something sharp. The pain always began when the foot came into action and increased in intensity the more the patient walked. Intermittence of the pain was also reported. Many patients were at times unable to go out of doors. The pain disappeared almost instantaneously if the shoes were removed and the foot placed at rest. Walking either barefoot or in slippers was less painful than walking in hard shoes. The complaint was constant and severe, and the patients were seriously inconvenienced.

Objective findings. In two-thirds of the cases the shape of the foot was normal, whereas in the other third the anterior arch was moderately depressed but not contracted. In one case, where the anterior arch was of a transverso-plane type, there was a subluxation at the 4th metatarsophalangeal joint. The skin appeared normal and there were no external sign of circulatory disturbance. The patients stated that they often had severe tenderness in the aching interspace, that it was recurrent and was accompanied by radiating pains or numbness in the adjacent toes; there seemed to be tenderness in the region of the neighbouring metatarsophalangeal articulation,

though it is difficult to demarcate this tenderness very distinctly. In one case there was slight swelling on the dorsal aspect of the affected interspace. Sensory tests were made in some cases, but they revealed nothing abnormal.

Previous treatment. Usually the patients expect relief from an arch support; they visit shops specialising in such appliances and buy arch supports of various types. When these have proved ineffective they seek medical advice. Cases, in our series, in whom the correct diagnosis had not been made, had been classified as e.g. Buerger's disease, polyarthritis, sciatica, arthrosis of the metatarsophalangeal articulation, etc., and treated in accordance with these diagnoses.

Treatment. After conservative treatment, which in no case had any effect, had been tried, the interdigital nerve at the site of the pain was resected. In 14 cases this was the 3rd, in 2 the 2nd, and in 2 the 4th interdigital nerve. A longitudinal incision was made on the plantar aspect of the foot corresponding to the anterior arch. From 1-2 cm. of the nerve corresponding to the bifurcation was resected. Macroscopically the nerves appeared to be thickened to the size of a slate pencil, or in some cases to the size of a pea, whilst in others there was no appreciable thickening.

Follow-up examinations were made in all cases. The period of observation varied from 3 months to 4 years, most were examined between 1 and 2 years after operation.

The results were as follows:

Subjectively completely cured	14	(77.8 %)
Subjectively improved	3	(16.7 %)
Subjectively unchanged	1	(5.5 %)

The recalcitrant case was the one already mentioned (see page 4), with a painful luxation in the metatarsophalangeal joint of the 4th toe, in addition to the Morton's disease. In this case, however, the radiating pain in the toes disappeared after the operation, and the character of the remaining symptoms indicated that they were due to the untreated luxation.

At the follow-up examination, the site of operation and the

whole front part of the foot were carefully inspected and palpated, and a sensory test was made on the toes. Previous authors have been afraid that the operation scar on the sole of the foot might inconvenience the patient. Our experience in this material shows, however, that in the majority of the cases the scar was barely noticeable or palpable. In one half of the material, consisting of those observed for the longest time, no definite sensory disturbance could be detected, whereas in the other half there was sensory impairment, which the patients had usually not noticed. No swelling suggesting an amputation neuroma was observed.

When diagnosing Morton's disease, it is necessary to exclude cases with the usual types of anterior arch trouble. In these cases the complaints are located more diffusely in the whole of the anterior arch; there is no radiating pain in the toes; and the pain often persists after the foot has been put to rest. We have called this pain metatarsalgia. Cases with metatarsalgia usually have a contracted, depressed, anterior arch with tender corns under the medial metatarsal heads.

The favourable results of resection of the nerve in Morton's disease encouraged us to try the operation in cases of metatarsalgia which had proved difficult to treat. The nerve was resected in 5 cases of metatarsalgia; all were followed up, and the result was unsatisfactory in all.

In order to show the relative frequency of these cases of metatarsalgia and of cases of Morton's disease, the number of cases registered at the Orthopedic Clinic of the Karolinska Institutet during the years 1945—47 under the diagnosis "pes transversoplanus" and Morton's disease are shown:

Pes transversoplanus	740 cases
Morton's disease	32 cases

Cases of transversoplanus were more than twenty times as frequent as cases of Morton's disease. During this period 8 of the 32 cases of Morton's disease were operated on. 6 of them were examined patho-anatomically and are included in this material.

B. Patho-anatomical examination.

The patho-anatomical changes in the nerves excised from our 18 cases of Morton's disease can be divided into four main groups (1) fibrosis, (2) endoneural oedema, (3) demyelination of the nerve and (4) vascular changes.

(1) *Fibrosis.* This is very variable in degree. In some cases there was macroscopic swelling of the nerve, limited to the part lying between the capsules of the metatarsophalangeal articulations. In some cases the swelling was as large as a pea or bean, and in these the hypertrophied nerve was more or less adherent to the adjacent structures. In other cases the nerve was not appreciably swollen. Microscopically the fibrosis was found to be perineural. In the mild cases there was merely slight thickening and condensation of the perineural capsule, and the nerve bundles were only slightly separated; the nerve was separated from the neighbouring arteries, synovial sheaths and joint capsules by loose interstitial tissue or adipose tissue. In cases where the fibrosis was more marked, the nerve bundles were separated by a compact mass of connective tissue, which also bound the nerve to the surrounding structures and embedded the larger trunks of the interdigital arteries. In some cases the connective tissue mass enclosed small bursal cavities, probably derived from the neighbouring synovial sheaths.

Endoneural fibrosis was seen, but was not particularly marked. No proliferation of Schwann's cells, nor infiltration with inflammatory cells, either endoneural or perineural, was seen in any of the cases.

In order to be able to demonstrate the variable degree of fibrosis, we have graded the changes +, ++ and +++, as in figure 1 a. These degrees of fibrosis were found in all but one case, in which the nerve was converted into a bean-sized fibrous nodule (figure 1 b).

(2) *Endoneural oedema.* In a number of cases there was oedema within the nerve bundles segregating the nerve fibres (fig. 2). This varies considerably in degree, both from case to case and in the different bundles of the same nerve. We have

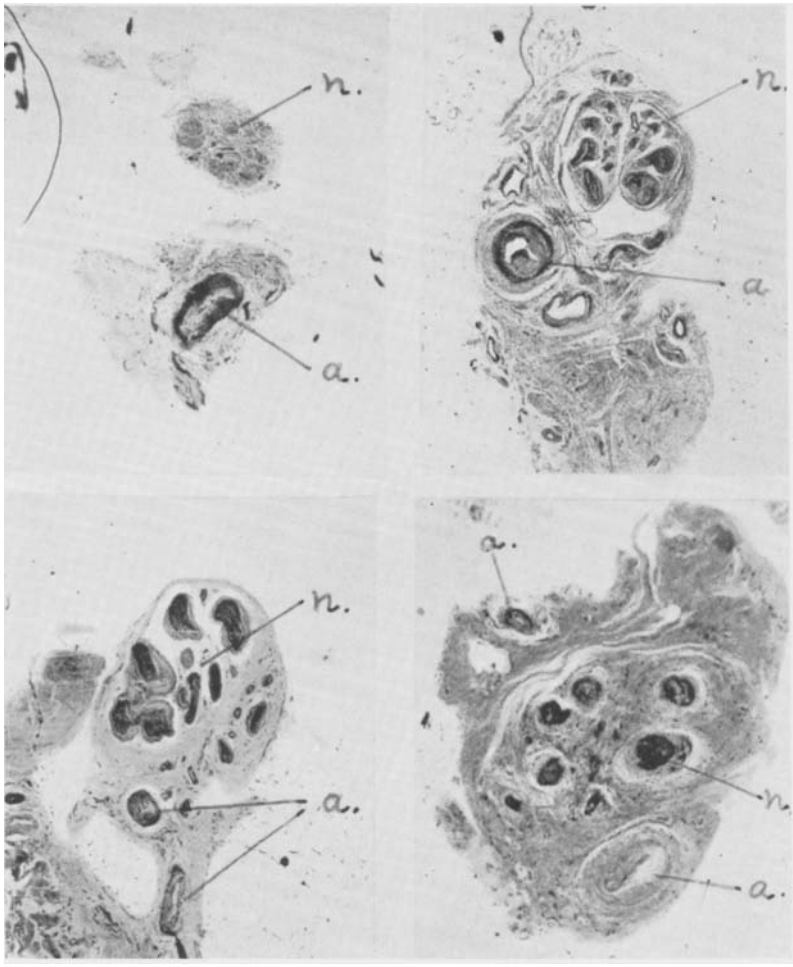

Fig. 1a.

Varying degree of perineural fibrosis. Cross sections of interdigital neurovascular trunks (Smith-Quinley stain). Myelinated nerve bundles and most of the vessels stain rather deeply black, connective tissue a paler shade. Upper left picture shows a specimen in apparently normal condition, nerve (n) free from the interdigital artery (a). The other pictures show fibrosis designated + to +++. The nerves are fused on to the vessels by connective tissue, which also separates the nerve bundles (Magnified about 12 ×).

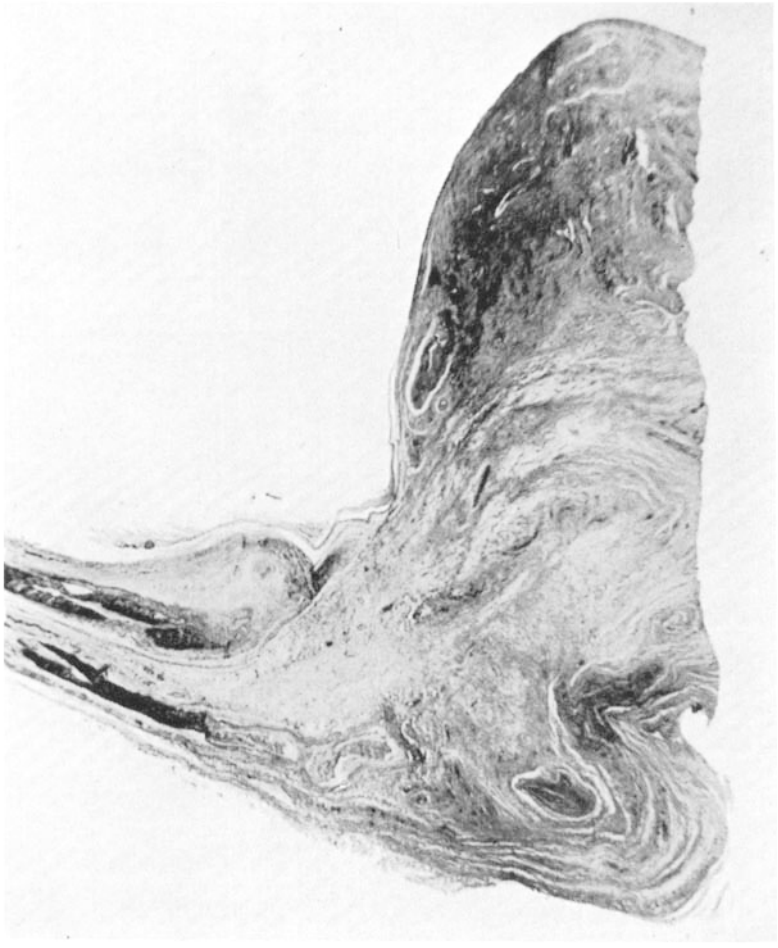

Fig. 1b.

The greatest degree of fibrosis found in the present series. Longitudinal section of one-half of a bean-sized nodule, with the nerve entering on the left. The nerve bundles are partly demyelinated and widely separated by dense connective tissue. This degree of fibrosis is designated ++++ (Smith-Quinley myelin stain, same magnification as figure 1a).

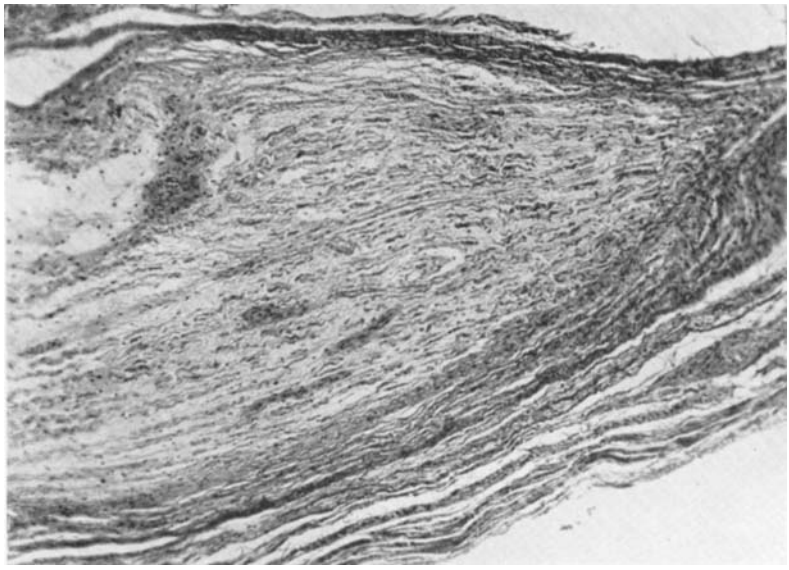

Fig. 2.
Endoneural oedema Oblique longitudinal section of nerve bundle with moderate uerineural fibrosis. (Mallory stain).

not encountered any oedema in the perineural connective tissue.

(3) *Demyelination.* We have demonstrated this phenomenon by Smith-Quinley staining of paraffin-embedded sections. It appeared as a more or less marked loss of staining of the myelin sheaths. Unfortunately the material did not permit an exhaustive examination with frozen sections and fat stains. No signs of early myelin decomposition were found. Demyelination varies considerably from case to case and within bundles of the same nerve. There was a certain degree of parallelism between the intraneural oedema and demyelination, but it was not constant. No parallelism was found between the degree of fibrosis and the demyelination.

(4) *Arterial changes.* These were found mainly in the larger arteries, the interdigital trunks and their larger branches down to a diameter of about 75 μ; they consisted in pro-

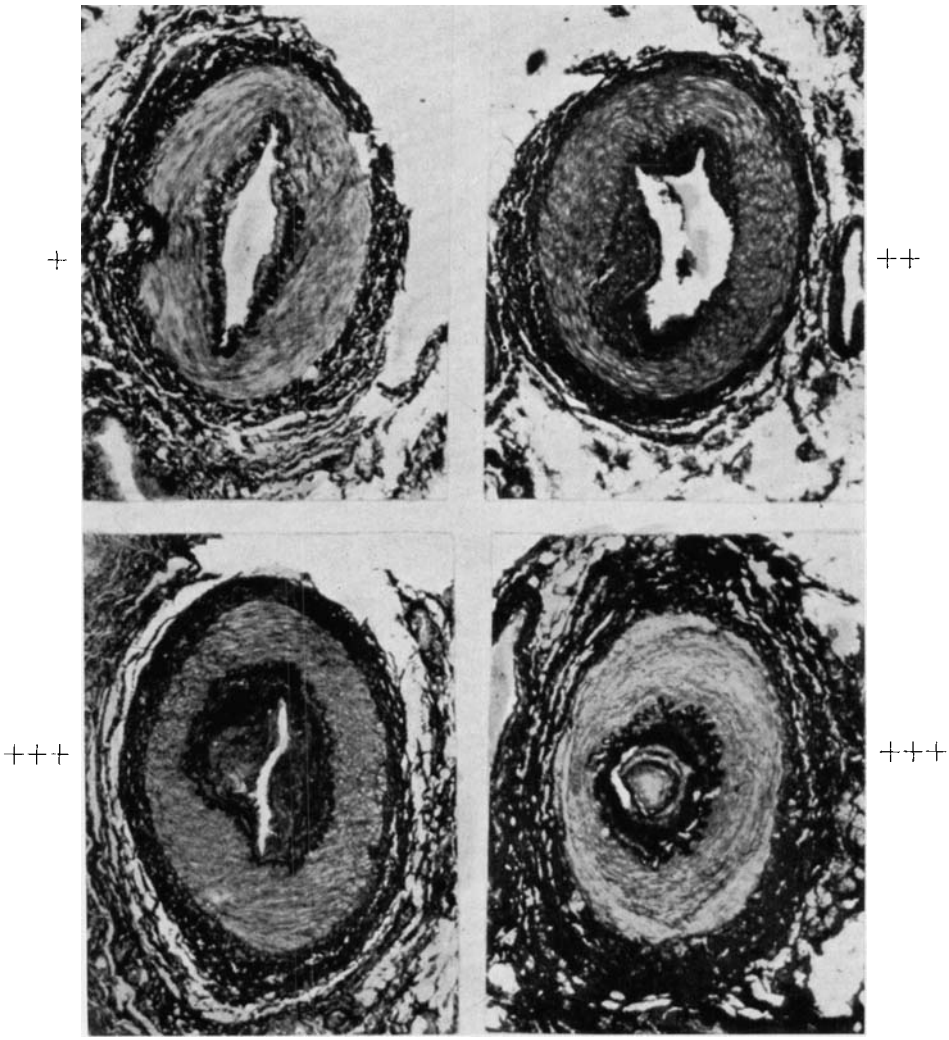

Fig. 3.

Various degrees of endarteritis. Upper left picture shows slight change, designated +, it consists mainly in hyperplasia of the internal elastic membrane. The upper right picture shows more advanced changes with an intimal cushion rich in elastic tissue (++). The two lower pictures show severe endarteritis (+++), the right one with an obstructive connective-tissue plug and small recanalisation lumina.
(Elastin-v. Gieson stain).

liferation of the subintimal layer and its elastic structures. If the change was slight, there was a hyperplasia of the tunica elastica with multiplicity of its lamellae; if more advanced, there was formation of connective-tissue cushions, very rich in elastic tissue, so that the lumen became irregularly constricted and split-shaped. In some cases the remaining lumen was filled with a fibrous mass less rich in elastic tissue; in this mass were endothelium-covered lumina resembling recanalization lumina in thromboses. Recent thromboses were, however, never seen. The arterial changes varied in different sections of the same specimen. The smaller arteries, especially the intraneural, were usually unchanged, and an insignificant change was found in the subintimal layer of such vessels in only a few cases. Neither intimal changes of an atheromatous type nor any calcification were found in the material. No changes were found in the media. In the cases with marked perineural fibrosis, where the arteries were embedded in the connective tissue, the tunica adventitia was condensed; otherwise its appearance was normal. Inflammatory infiltration of the artery was never encountered. Figure 3 shows the different grades of arterial change, +, + + and + + +.

The veins showed in many cases mild changes consisting of superficial intimal cushions rich in elastic fibres.

All these changes varied considerably. In many cases there was no visible fibrosis, and in others it was only slight. In about one half of the cases, however, it was well marked and even in some cases severe. It is difficult to estimate the frequency of arterial changes because, as we have pointed out, the changes can usually only be found in the larger arteries, and, unfortunately, these have been seen in the biopsy specimens of only 11 out of 18 cases. In most of them however, the larger arteries did show changes.

Bickel and *Dockerty* state that the degree of pathological change is proportional to the length of the history. This, however, was not true of our material. Table 1 shows the cases arranged in order of the severity of the fibrosis. It will be seen that there are cases with a very long history and only a slight

fibrosis, and vice versa. On the other hand there is some parallelism between the arterial changes and severe fibrosis.

In order to ascertain how far these observed changes are characteristic for subjects with the clinical picture of Morton's disease, we examined the interdigital nerves and arteries in a series of post-mortem cases of more or less the same age as these cases of Morton's disease. The bodies were those of healty subjects who had died suddenly from various causes (material from the Department of Forensic Medicine), and of hospital patients who had died from various diseases, e.g. tuberculosis in quite a number of cases, surgical conditions, blood diseases, acute infections, etc. Cases of arterio-sclerosis or other cardio-vascular disorders were excluded. As far as it was possible to ascertain from the hospital records and other information, none of them had ever had any foot disease of the Morton type. In each case the interdigital nerves, together with the surrounding connective tissue and arteries, especially from the parts where the nerves pass between the metatarsophalangeal articulations, were dissected out of the left foot. Three or four arterial nerve trunks were examined in each case, and the examination always included the trunk between the 3rd and 4th toe. At microscopy, special attention was paid to the four changes mentioned above, i.e. fibrosis, endarteritis, endo-neural oedema and demyelination. Fibrosis and endarteritis were graded as for Morton's disease. Table 2 shows 18 Morton's and 22 control cases, arranged according to age. The control cases were classed with the severest grade of change found in any of the excised neurovascular trunks. The trunks most affected were usually those between the 2nd and 3rd and the 3rd and 4th toes.

Table 2 shows a high incidence of both fibrosis and end-arteritis in the control material. Admittedly, the fibrosis was never so marked as in the few Morton cases with the most marked changes, but nevertheless it is comparable with that seen in most of the cases. The arterial changes were equally marked, both as regards type and degree of severity in the control material, and in individual cases they were even more

TABLE 2

Sex	Age	Fibrosis	Oedema	Demyelination	Endarteritis
Cases of Morton's Disease.					
F	58	+	0	+	
F	57	++	+	+	+++
M	54	+	+	+	
F	52	+	+	+	
F	52	+	+	+	+++
F	46	+++	+	+	+
F	42	++++	+	+	
F	42	++	0	(+)	
M	40	+	+	+	
F	37	++	0	+	+++
M	36	+++	+	+	+++
F	36	+	0	0	(+)
M	36	+++	+	+	+++
F	36	++	+	+	+++
F	34	+	0	(+)	
F	34	0	0	0	0
F	31	++	+	0	++
F	30	+++	0	+	+++
Controls.					
M	63	++	0	0	++
M	51	++	0	0	+++
F	50	++	0	0	+++
F	50	(+)	0	0	+++
F	48	++	0	+	+++
F	45	+	0	0	+++
F	44	++	+	0	+++
M	42	+	(+)	0	+++
M	42	+	0	0	+
F	42	++	0	0	++
F	39	++	+	+	+++
M	37	+	0	0	+
F	35	++	+	+	0
F	32	(+)	0	0	+
F	31	(+)	0	0	(+)
F	30	++	+	0	+++
M	30	0	0	0	(+)
M	29	++	(+)	+	+++
M	28	+	0	0	(+)
M	19	+++	0	0	++
M	16	0	0	0	(+)

severe than in any of the Morton's cases. In the control material the endarteritis was more constant in cases aged over 40 years, although it occurred also in one or two below 40. The endoneural oedema and the demyelination were much less frequent in the control material, occurring only occasionally. In table 3 the frequency of the different phenomena in the controls and in Morton's cases are compared. It will be seen that the frequency of arterial changes was so similar that the difference is negligible. There was also a similarity in the frequency of fibrosis but, as we have pointed out, in some cases of Morton's disease the fibrosis was much more severe than in the control material. Intraneural oedema and demyelination, however, were definitely more common with Morton's disease cases than in the control material, and even the degree of these changes (not shown in Table 2) was more marked with Morton's disease.

TABLE 3

	Cases of Morton's Disease	Controls
Fibrosis $\geqq$++	10/18 = 55.5 %	11/21 = 52.4 %
Endarteritis $\geqq$++	8/11 = 72.7 %	13/21 = 61.9 %
Oedema	11/18 = 61.1 %	5/21 = 23.8 %
Demyelination . . .	15/18 = 83.3 %	4/21 = 19.0 %

TABLE 4

Metatarsalgia.

Sex	Age	Fibrosis	Oedema	Demyelination	Endarteritis
F	46	+	0	0	+
F	41	+	0	0	+++
F	36	+	+	0	+++
F	33	+	0	0	++
F	18	0	0	0	0

As supplement to these control examinations, excised digital nerves taken from five patients suffering from metatarsalgia (see page 7.), were examined. Table 4 shows the changes present in this small material. It will be noticed that

in these cases there were also considerable arterial changes, but fibrosis and the other phenomena were practically absent.

DISCUSSION

In the literature there have been two schools of thought on the pathogenesis of Morton's disease. One, to which, among others, *Morton, Tubby and Jones, Bickel and Dockerty, Betts* and *McElvenny* belong, believes that the condition is caused by chronic trauma to the interdigital nerve, brought about by pressure or crushing. The other, led by *Nissen,* believes that it is primarily an endarteritis in the interdigital artery which leads secondarily to ischemic changes in the nerve.

Our patho-anatomical examinations have shown that chronic endarteritis is a frequent phenomenon in Morton's disease. At the same time, examination of a control material showed that endarteritis of this type occurs with approximately the same frequency and the same degree of severity in persons who have no disease of the feet. It is quite possible that endarteritis in the interdigital arteries causes fibrosis of the nerves, but this process seems to be so common in this region of the body that one asks oneself whether, after a certain age, it is not physiological. In most cases it does not give rise to any painful symptoms. Further, there is no evident relation in our material between these phenomena and the duration and severity of the symptoms. One must, therefore, presume that the pain in the Morton's cases is caused by one or more additional factors—these factors being either quite independent of the endarteritis-fibrosis complex or in direct conjunction with it. The fact that Morton's symptoms subside after excision of the nerve does not necessarily mean that the cause of the pain is a change in the nerve itself. It could mean merely that excision of the pain track abolishes the symptoms. It is interesting to find, however, that the most usual age for the onset of Morton's disease coincides with the time when, to judge from the control material, interdigital arteritis also

begins to be common. It is also worth while pointing out that intraneural oedema and demyelination seem to be more common in the Morton's cases than in the control cases. This might indicate that the pain is in some way connected with some specially accentuated disturbance of the circulation or metabolism of the nerves in these cases.

Apart from the problem which particularly interests us in this investigation, viz. the pathology of Morton's disease, we find the frequent occurrence of endarteritis in this region of the body of very great interest. Our small material is, of course, only sufficient to indicate the approximate frequency of the phenomenon and the general type of the changes. It may be said that the changes have certain characteristics in common with Bürger's endarteritis obliterans, but rapid thrombosis and inflammatory infiltration, which are both part of the classical Bürger picture, are not seen. A ready suggestion is that the arterial changes in question are closely allied to the strain placed upon the foot. In order to gain support for this theory, it would be necessary to carry out a more comprehensive investigation on autopsy material, with a view to estimating still more closely the frequency of the changes at various ages and their sites, and to comparing the findings with those in the interdigital neurovascular trunks of the hand, etc.

SUMMARY

The clinical picture, treatment, results of treatment and the patho-anatomy in 18 cases of Morton's disease are surveyed. Morton's disease has a typical clinical picture distinct from other painful conditions in the anterior part of the foot. It is characterised by: pain when the foot is in use, tenderness, usually felt between the metatarsophalangeal articulations of the 3rd and 4th toes, and radiating pain, often with cramp and numbmess, in the adjacent toes. The pain disappears immediately the foot is placed at rest. Resection of the interdigital nerve gives complete freedom from pain and does not

seem to cause any complications in the form of amputation neuromata or cicatricial tissue.

The patho-anatomical examination showed that four main changes are found in the nerve: perineural fibrosis, endarteritis in the interdigital arteries, endoneural oedema and demyelination of the nerves.

Examination of the interdigital neurovascular trunks in the feet of a control material of autopsy cases, who had not had any symptoms of Morton's disease, showed that perineural fibrosis and endarteritis occurred with the same frequency as in the Morton's cases, but that intraneural oedema and demyelination were considerably less frequent in the control material. Fibrosis was not so marked in the control material as in some of the cases of Morton's disease. There was, however, no parallelism between the degree of fibrosis and either the duration or the severity of the Morton's symptoms.

The theory expounded by Nissen, that Morton's disease depends on a promary endarteritis and a secondary fibrosis as a consequence of circulatory disturbances, cannot be said to give a full explanation. Our control examinations showed that after the age of 40 years the endarteritis-fibrosis complex is so common in the interdigital neurovascular trunks of the foot, that one wonders whether it may not after all be physiological (weight bearing?); Morton's disease on the other hand is comparatively rare. Some special factors, so far unknown, and possibly connected with endarteritis or fibrosis, must be presumed for the appearance of the pain complex. It is possible that the high incidence of intraneural oedema and demyelination characteristic of the Morton cases may provide a clue for further investigations into the true pathogenesis of the disease.

RESUME

Description du tableau clinique, du traitement, des résultats thérapeutiques et de l'anatomie pathologique de 18 cas de la maladie de Morton.

La maladie de Morton a un tableau clinique typique, distinct de toute autre affection de la partie antérieure du pied.

Il se caractérise par les symptômes suivants: douleur à la marche, sensibilité douloreuse généralement ressentie entre les articulations métatarso-phalangiennes des 3ème et 4ème orteils, et douleur irradiante souvent accompagnée de crampes et d'engourdissement dans les orteils adjacents. La douleur disparaît dès que le pied est mis au repos. La résection du nerf interdigital supprime immédiatement toute douleur et ne semble par donner lieu à des complications sous forme de névrome d'amputation ni d'hyperplasie fibreuse.

L'examen anatomo-pathologique décela quatre modifica- principales du nerf : fibrose périneurale, endartérite des artères interdigitaux, oedème endoneural et démyélinisation des nerfs.

L'examen des nerfs et vaisseaux interdigitaux du pied sur un matériel de contrôle constitué par des cas d'autopsie de sujets n'ayant présenté aucun des symptômes de la maladie de Morton prouva que l'on rencontre la fibrose périneurale et l'endartérite aussi fréquemment que dans les cas de la maladie de Morton. Par contre, les cas d'oedème intraneural et de démyélinisation sont beaucoup moins fréquents dans le matériel de contrôle. La fibrose n'est pas aussi marquée dans le matériel de contrôle que dans certains des cas de la maladie de Morton. Il n'y a cependant aucun parallèlisme entre le degré de la fibrose, la durée ou la gravité des symptômes de la maladie de Morton.

La théorie exposée par Nissen, suivant laquelle la maladie de Morton est due à une endartérite primaire et à une fibrose secondaire, conséquences de troubles circulatoires, ne paraît pas donner une explication entièrement satisfaisante. Nos examens de contrôle montrent qu'au-delà de 40 ans le complexe endartérite-fibrose est si commun dans les nerfs et vaisseaux interdigitaux qu'on se demande si, après tout, ce n'est pas un phénomène physiologique (poids à porter). La maladie de Morton est par ailleurs relativement rare. Un ou plusieurs facteurs particuliers, inconnus jusqu'à prèsent, se rattachant à l'endartérite ou à la fibrose, suscitent peut-être le complexe douloureux. Il est possible que la forte incidence de l'oedème intraneural et de la démyélinisation qui caractérisent les

cas de la maladie de Morton puissent guider les recherches tendant à établir la véritable pathogenèse de la maladie.

ZUSAMMENFASSUNG

Das klinische Bild, die Behandlung, die Behandlungsresultate und die pathologische Anatomie in 18 Fällen von Mortons Krankheit werden zusammengestellt und besprochen. Mortons Krankheit zeigt ein typisches klinisches Krankheitsbild, das sich von anderen schmerzhaften Zuständen im Vorfusse deutlich unterscheidet. Es ist charakterisiert durch Schmerzen, die beim Gebrauche des Fusses auftreten, und durch Schmerzhaftigkeit, die gewöhnlich zwischen den Metatarsophalangeal-Gelenken der 3. und 4. Zehen gefühlt wird, ferner durch ausstrahlende Schmerzen, oft verbunden mit Krämpfen und Parästhesien, in den erwähnten Zehen. Der Schmerz verschwindet, sobald der Fuss ruhig gehalten wird. Resektion des Interdigitalnerven führt zur vollständigen Befreiung vom Schmerz und scheint keinerlei Komplikationen in Form von Amputationsneuromen oder Narbengewebe hervorzurufen.

Die pathologisch anatomische Untersuchung erwies, dass hauptsächlich viererlei Veränderungen am Nerven vorgefunden werden: Perineurale Fibrose, Endarteriitis der Interdigitalarterien, endoneurales Ödem und Markscheidenverlust des Nerven.

Untersuchungen des interdigitalen neurovaskulären Bündels an den Füssen eines Kontrollmaterials von Autopsiefällen, die keinerlei Symptome von Mortons Krankheit hatten, zeigten, dass perineurale Fibrose und Endarteriitis ebenso oft zu beobachten waren, als in Fällen von Mortonscher Krankheit, dass aber das intraneurale Ödem und der Markscheidenverlust bedeutend seltener im Kontrollmaterial zu sehen waren. Fibrose war nicht so ausgeprägt im Kontrollmaterial wie in einigen Fällen vor Mortons Krankheit. Man konnte jedoch keinen Parallelismus zwischen dem Grade der Fibrose und der Dauer und Schwere der Krankheitssymptome feststellen.

Nissens Theorie, dass Mortons Krankheit von einer primären Endarteriitis ausgeht, die eine sekundäre Fibrose auf Grund der Zirkulationsstörungen zur Folge hat, kann nicht

als eine voll zureichende Erklärung angesehen werden. Unsere Kontrolluntersuchungen zeigten, dass sich nach dem vierzigsten Lebensjahre der Endarteriitis-Fibrose Komplex in den interdigitalen neurovaskulären Bündeln so allgemein nachweisen lässt, dass man sich fragt, ob es sich hier nicht um physiologische Veränderungen (Belastung?) handelt. Mortons Krankheit ist immerhin eine seltene Erscheinung. Irgendein besonderer Faktor, vorläufig unbekannt und möglicherweise mit der Endarteriitis und Fibrose verbunden, muss als Ursache für das Entstehen der Schmerzen angenommen werden. Möglicherweise kann das häufige Vorkommen von intraneuralem Ödem und Markscheidenverlust, so charakteristisch für Mortons Krankheit, einen Hinweis für die weitere Untersuchung der wirklichen Pathogenese der Erkrankung geben.

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