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OSTEOCHONDRITIS DISSECANS AND SIMILAR LESIONS OF THE TALUS

Report of Fifty-Five Cases with Special Reference to Etiology and Treatment.

By

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Even since Franz König propounded his theory of osteochondritis dissecans in 1888, that articular lesion has continued to occasion interest and numerous papers on it have been published. Opinions as to the etiology have varied in the course of the years. Broadly speaking we can distinguish two main views: traumatic etiology, and spontaneous necrosis with no traumatic factor. These different theories have been extensively treated in the Scandinavian literature by Gösta Jansson (1930), M. Aage Nielsen (1933), John Hellström & Karl Östling (1934), S. Ribbing (1937) and Hugo Aronsson (1942). We shall not, therefore go into them here.

So far as we can gather from the available literature, fifty cases of osteochondritis dissecans of the talus have been published (see table I). Our series comprises 55 cases and throws light on the problems involved, thus justifying the presentation of this paper. The cases were collected at the Department of Orthopedics, Karolinska Institutet, the Department of Surgery and the Military Department of Surgery, Karolinska Sjukhuset. The extent of the present series is due in great part to a first-class radio-diagnosis. (Ray & Coughlin (1947), among others, have emphasized that frontal and lateral roentgenograms may not suffice to disclose osteochondritis dissecans of the talus; in some cases the condition can be demonstrated only by oblique views).

The series, which is summarized in table II, comprised 16 females

TABLE 1
Previously Published Cases of Osteochondritis Dissecans of the Talus.

Author	Year	Medial Lesions		Lateral Lesions	
		Trauma	No Trauma	Trauma	No Trauma
Kappis, Max	1922				
Wolff, Aage O.	1926		2		
Breitländer	1927		1		
Cordes	1927		1		
Harms	1927	1			
Vogel, K.	1927		1		
Axhausen, G.	1928		1 + 1		
Deutschländer, Carl	1928			1	
Garcia Diaz	1928		1		
Löwen, A.	1929		2		
Sidler, A.	1929/30		1		
Radicke	1930		1		
Fairbanks, H. A. T.	1933	1			
Hellström & Östling	1934			1	
Burr, R. C.	1939	2			
Myhre, Helge	1939	2	1		
Hoffmann (see Myhre).			1		
Brickey, Paulet, Grow.	1940			1	
Mensor, M. C., Melody, G. F.	1941	1			
Aronsson, Hugo	1942	1	3		
Cobey, Milton C.	1943	1		2	
Vaughan, C. E., Stapleton, J. G.	1947	1	2	1	
Ray & Coughlin	1947	7	4	3	
Marks, Kenneth L.	1952			1	
		17	23	10	

and 39 females ranging in age from 9 to 65 years, nearly one half being in the 20–30 year age group.

The observation period in the 51 who were followed-up varied between one and twelve years.

Twenty-four patients sought treatment very soon after the onset of symptoms, and the others after periods of up to 14 years.

Five patients had bilateral involvement: in one the lesions were lateral, in three medial, and in the fifth lateral on one side and medial on the other. Of the unilateral cases, the lesions were medial in 34 and lateral in 16, involving the right ankle in 28 and the left in twenty-two. Since the medial and lateral lesions seem to have different pathogeneses and prognoses, we have found it appropriate to deal with them separately.

I. LESIONS OF THE MEDIAL ANGLE OF THE TALUS

Trauma.–Forty-One Cases. Of these cases, 16 had no history of trauma and 11 had sought treatment for fresh traumata, but were found on roentgenologic examination to have lesions of long standing. In the remaining 14 cases, there were traumata of such long standing that they might have been etiologically implicated in the osteochondritis dissecans. Six of these 14 patients spoke of a trauma of such mild character that no treatment had been required and the capacity for

TABLE II
Personal Series.

Case No.	Sex	Age at Initial Examination	Age at Onset of Symptons	Trauma = + No Trauma = –	Follow-up Time in Years	Ankle Affected	Localization Lateral = l Medial = m	Operated = + Unoperated = –	Symptom-Free = + Not Symptom-Free = –	X-Ray Progression = + X-Ray Healing = – X-Ray Status Quo = ±
1.	m	65	65	+	2	L	l	—	—	±
2.	m	23	23	+	6	R	l	+	+	—
3.	m	22	21	+	2	L	l	+	—	—
4.	m	37	37	+	7	L & R	l	—	—	±
5.	m	20	17	—	3	L	m	+	+	±
6.	m	23	21	+	4	L	m	—	—	+
7.	m	21	19	+	8	R	l	—	—	±
8.	f	55	52	+	6	L	m	—	—	—
9.	m	21	21	+	2	R	m	—	+	
10.	m	25	23	+	2	R	m	—	+	—
11.	m	22	19	+	2	R	m	—	+	±
12.	m	36	22	+	8	R	m	—	—	—
13.	m	26	26	+	8	L	m	—	+	±
14.	m	21	19	+	6	L	l	—	—	—
15.	m	65	65	+	3	L	m	—	+	±
16.	f	49	30	—	5	C & R	m+?	—	—	±
17.	m	17	17	+	2	R	m	—	+	±
18.	m	21	21	+	1	R	l	+	—	+
19.	f	33	33	+	8	R	m	—	+	—
20.	f	35	25	+	0	R	m	—		
21.	m	18	17	—	3	R	l	+	—	—
22.	f	47	47	+	7	L	m	—	+	
23.	m	52	50	—	10	R	m	—	+	—
24.	m	24	22	+	4	R	l	—	+	—
25.	m	27	27	+	8	R	l	—	—	—
26.	f	38	37	—	2	L	m	—	+	±
27.	f	42	42	+	2	L	m	—	+	

TABLE II
(continued)

Case No.	Sex	Age at Initial Examination	Age at Onset of Symptoms	Trauma = + No Trauma + −	Follow-up Time in Years	Ankle Affected	Localization Lateral = l Medial = m	Operated = + Unoperated = −	Symptom-Free = + Not Symptom-Free = −	X-Ray Progression = + X-Ray Healing = + + X-Ray Status Quo = ±
28.	m	26	21	+	12	L	m	—	—	—
29.	f	22	21	+	3	L	l	+	—	—
30.	m	50	47	+	10	Hö	m	—	—	+
31.	k	25	20	+	3	Hö	m	+	+	—
32.	m	30	29	—	0	Vä	m	—		
33.	m	17	15	—	4	Vä	m	—	—	±
34.	m	34	34	—	3	Hö	m	+	+	—
35.	k	9	9	+	11	Vä	m	—	+	—
36.	m	23	23	+	3	Vä	m	+	+	—
37.	m	24	24	+	2	Hö	l	—	—	+
38.	m	41	41	+	0	Hö	m	—		
39.	m	52	42	+	1	Hö+Vä	m	—	+	±
40.	k	14	14	—	7	Hö+Vä	m	±	±	±
41.	m	24	24	+	2	Vä	l	+	+	
42.	k	20	19	+	5	Hö	l	—	+	+
43.	m	25	24	—	3	Vä	m	—	—	±
44.	m	51	51	+	1	Vä	m	—	—	—
45.	k	29	22	+	0	Hö	m	—		
46.	k	12	11	—	1	Vä	m	—	+	—
47.	m	39	39	+	1	Hö	l	+	+	
48.	m	23	20	+	2	Vä	m	+	—	—
49.	k	15	14	—	2	Vä	m	+	+	±
50.	m	21	21	+	1	Vä	l	—	—	
51.	m	17	17	+	9	Hö	l	—	+	—
52.	k	50	49	—	8	Hö+Vä	m	—	—	—
53.	m	22	22	+	2	Vä	m	—	+	±
54.	m	47	47	+	½	Hö	m	—		
55.	m	47	46	—	3	Hö	m	—	—	±

work had not been appreciably impaired. The other eight spoke of a major trauma that had caused incapacity for work of varying duration. These did not differ in any way from the others.

Symptoms, treatment and follow-up.

Of the previously *symptomless* cases that were discovered by chance at roentgenologic examination for acute traumata, one was submitted

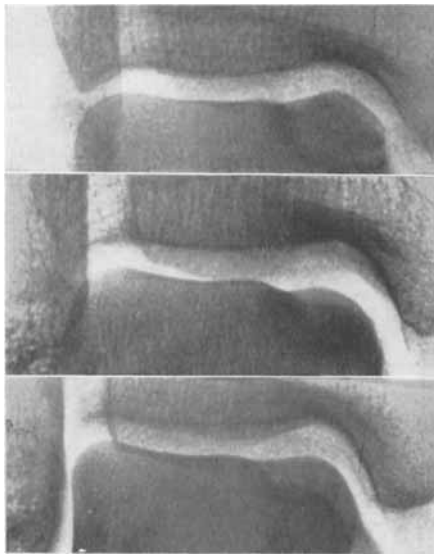

Fig. 1 a.
Roentgenograms from 1945, prior to operation (top), from 1947 (middle) and from 1950 (bottom).

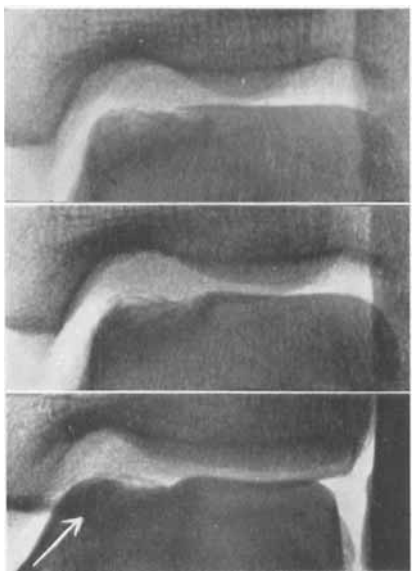

Fig. 1 b.
Roentgenograms from 1945 (top), 1947 (middle) and from 1950 (bottom).

for operation. Ten patients underwent follow-up examinations and eight of them, including the operated case, were found to be symptom-free; the other two had mild symptoms of arthrosis type.

Symptoms of locking were found in four, all of whom had roentgenologically demonstrable loose bodies. One of these four was operated on, and at the follow-up proved to be symptom-free. Also symptom-free at the follow-up was one patient in whom the loose body had moved out and had been placed on the medial side of the talus; the other two still had symptoms of locking.

Twenty-six had *symptoms of arthrosis type,* i.e., pain on weight bearing and movement. Five patients in this group underwent operation; five were submitted to fixation and the others to symptomatic treatment. At the follow-up four of the five surgically treated cases were symptom-free and one had unchanged symptoms. Of the 21 non-operated patients in this group, 18 reported for follow-up examination and nine of them were symptom-free. One of the patients in this group had bilateral involvement; one of her ankles was operated on, and at the follow-up she was symptom-free on the operated side but still had symptoms on the untreated side. A brief case report follows.

Case 40. Female, aged 14. For one year there had been pain on weight bearing and movement, as well as swelling of both ankles. There was no trauma. Clinical findings: moderate swelling over the talocrural joints and tenderness on palpation. –Roentgenologic findings, November, 1945: Bilateral osteochondritis dissecans with medial crateriform deformation of the superior margin of the talus.–Since the symptoms were most pronounced in the right ankle, the latter was subjected, in December, 1945, to arthrotomy and excision of the pathologic area. At repeated follow-up examinations, the last in April, 1952, the patient was symptom-free in the operated joint but still had symptoms of arthrosis in the contralateral joint. A follow-up roentgenogram in September, 1950 disclosed a regular operation defect medially in the superior margin of the right astragalus (see fig. 1 a), but an irregular defect, surrounded by sclerosis, on the left (see fig. 1 b).

Roentgenologic Development.–At the roentgenologic follow-up examination five of the seven operated cases showed more or less advanced filling of the defect and levelling of the edges (see fig. 2), but the two others exhibited no change.

Twelve of the non-operated cases showed unchanged roentgenologic findings (the respective follow-up times were 1, 2, 2, 2, 2, 3, 3, 3, 5, 5, 7 and 8 years). Roentgenologic signs of healing were present in ten cases (respective follow-up times: 2, 2, 2, 7, 8, 8, 9, 10, 12 and 12 years). (See fig. 3).

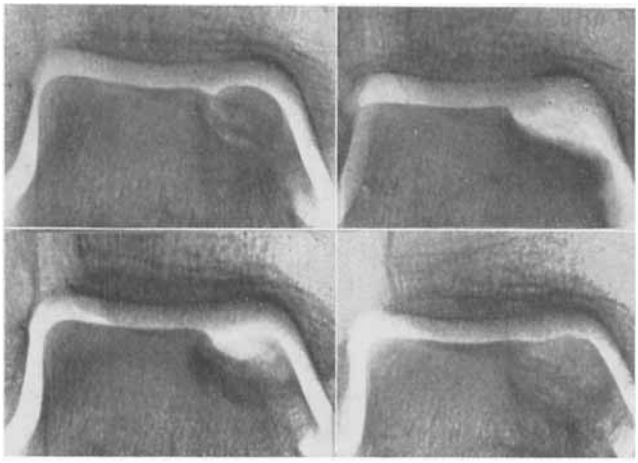

Fig. 2.
Roentgenograms from 1947 prior to operation (top left) and after operation (top right); from 1948 (bottom left), and from 1950 (bottom right).

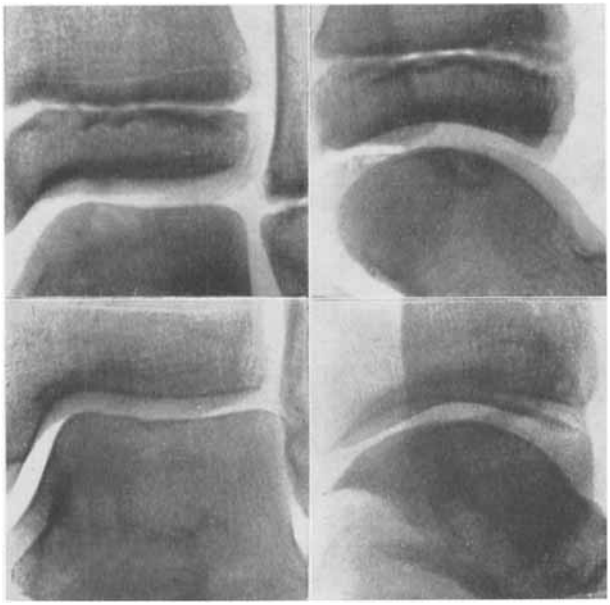

Fig. 3.
The upper roentgenograms are from 1939 and the lower from 1950. (Non-operated case).

Roentgenologically demonstrable progression was found in only three cases (follow-up times 4, 7 and 11 years respectively). The other cases in this group were not examined roentgenologically at the follow-up.

TABLE III

	Without Symptoms	With Symptoms
Roentgenologically unchanged	8	4
Roentgenologically worse	0	3
Roentgenologically healed	5	5

Table III shows the roentgenologic findings at the follow-up in relation to the subjective symptoms.

Twelve of the 41 medial lesions were associated with roentgenologically demonstrable arthrosis in the talocrural joint. In five of these cases progression had occurred during the follow-up period.

II. LESIONS OF THE LATERAL ANGLE OF THE TALUS

This group numbered 19 patients (including one with bilateral involvement)–16 males and three females.

Trauma.–Only one of the 19 cases had no history of trauma. In seven cases the patients had undergone roentgenologic examination immediately after a severe adduction trauma. The roentgenograms showed flake fracture of the lateral articular surface of the talus. In the further course roentgenograms disclosed osteochondritis dissecans at the same site.

Case 29. Male, aged 22. In May, 1951 he sustained a severe adduction trauma to the right ankle. Clinical findings: Swelling and hematoma discoloration below it and anterior to the lateral malleolus.–Roentgenologic findings: At the superior lateral margin of the talus a thin chip of cortical bone, about 1 cm long, was avulsed and displaced about 1 mm upward.–The patient was submitted to plaster fixation for one month. The condition improved, though pain still persisted on weight bearing and movements of the ankle. Roentgenologic findings, April, 1952: Osteochondritis dissecans with decalcification round the fracture area (see fig. 4). –Due to persisting symptoms, an arthrotomy was performed on May 7, 1952, with excision of the osteocartilaginous fragment from the right talus and removal of a biopsy specimen from the joint capsule. Pathologic diagnosis (Engfeldt): (5 a). The section showed spongy bone tissue as well as some ordinary articular cartilage and an area of cellular fibrous cartilaginoid tissue, which in parts showed opening out and regressive changes. (5 b). Joint capsule tissue that was moderately fibrous and, on the inner surface, in parts coarsely papillate. The parts nearest to the articular cavity were highly vascularized and congested. This tissue was in parts

fibrous, in other parts looser and slightly edematous. Quite a number of histiocytic elements and a few isolated collections of lymphocytes were observed.

At a follow-up examination six months after the operation, the patient was symptom-free.

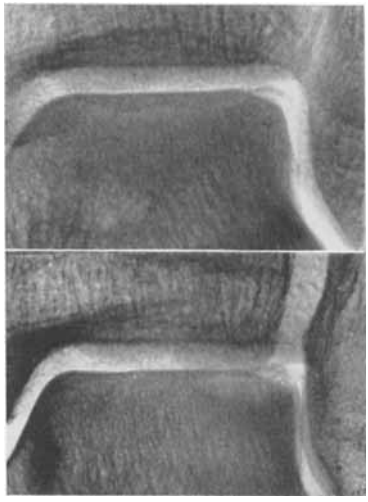

Fig. 4.
The upper roentgenogram is from 1951 and the lower from 1952.

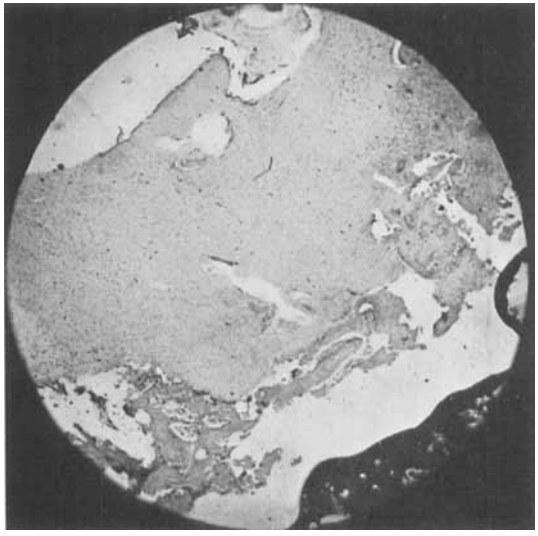

Fig. 5 a.

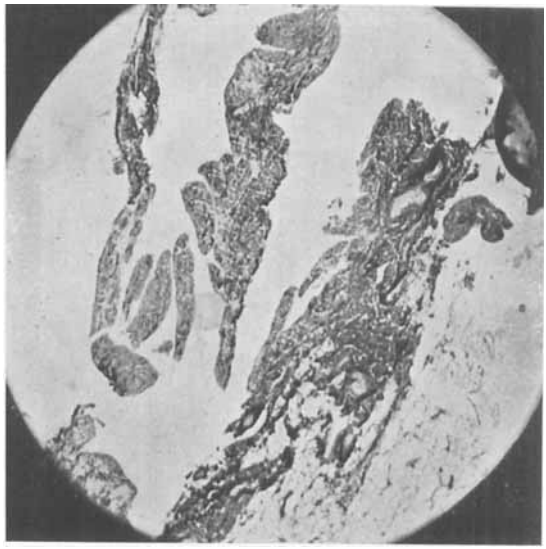

Fig. 5 b.

Case 41. Male, aged 25. On Sept. 27, 1950, the patient sustained a severe adduction trauma to the left ankle. Roentgenologic findings, Sept. 27: On the superior lateral margin of the astragalus, a small fragment of bone was avulsed and displaced about 1 mm upward (see fig. 6 a). Treatment consisted in plaster fixation for five weeks. This notwithstanding, there was pain on weight bearing and movement, as well as swelling and locking of the joint. Objective examination

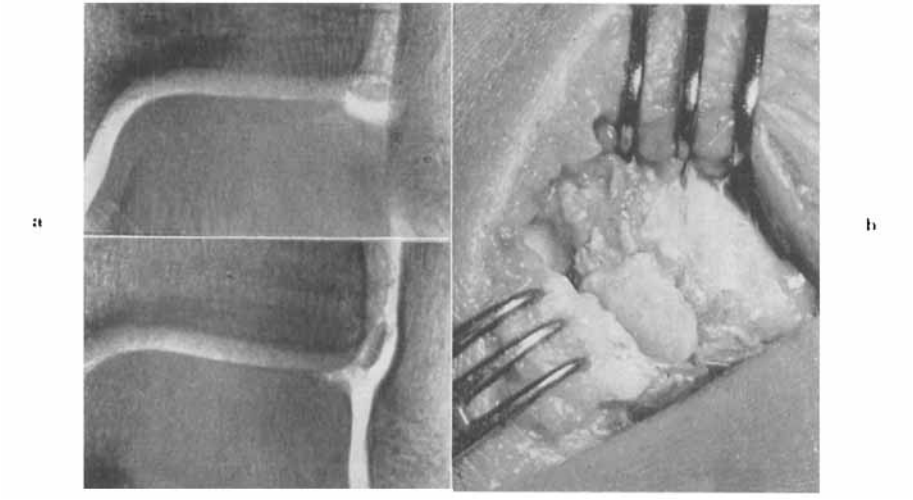

Fig. 6.
(a) Upper roentgenogram from 1950 and lower from 1951.
(b) Photograph at operation.

disclosed slight limitation of the dorsal extension and tenderness to pressure over the anterior part of the lateral malleolus. Roentgenologic findings, Dec. 14, 1951 (14½ months after the trauma): The small fragment of bone from the lateral margin of the talus was displaced somewhat proximally and rotated about 90 degrees. The fracture surface of the talus was somewhat more regular but still not quite smooth. The level of the cartilage was unchanged.–Operation, Jan. 3, 1952: At the angle of the talus was a depression with an irregular surface. The loose body was located some distance posteriorly, and not until the anterior portion of the syndesmosis had been divided was it possible to separate fibula and tibia sufficiently for removal of the loose body. The latter measured 10 by 7 mm and was covered by cartilaginoid tissue on all sides (see fig. 6 b).–At follow-up examination six months later, the patient was symptom-free.

Eleven patients had a history of trauma but had not been examined at the time it occurred. One of them was a case of relatively mild distortion; the other ten concerned severe traumata. It was impossible, however, to reconstruct the traumata in detail afterwards.

Subjective Symptoms.–Each of these 19 patients had symptoms of arthrosis type after the acute distortion symptoms had subsided. In two of them locking of the joint had occurred as well.

Treatment.–Seven of the above 19 were submitted to operation (arthrotomy and excision of the osteochondritic focus). Four of these seven were quite symptom-free (respective follow-up times: 5 years, and 6, 6 and 3 months), and three had very slight symptoms (observation times 2, 3 and 4 years respectively). Of the twelve non-operated cases, three were symptom-free at follow-up examinations after 5, 7 and 10 years respectively; two of the latter showed roentgenologic healing (see fig. 7), but in the third case there were still conspicuous roentgenologic changes at two follow-ups with an interval of five years (see fig. 8). Nine had moderate symptoms which did not appreciably affect the capacity for work. Of these cases, roentgenologic examination showed unchanged conditions in five, incipient healing in two and progression in two.

Operative Technic.–The talocrural joint was opened anteriorly with a transverse incision in the skin and, after retraction of the tendons a longitudinal incision in the capsule. Pronounced plantar flexion of the foot enabled the changed area and the loose bodies to be exposed and removed.

DISCUSSION

As will be seen from the present series, certain differences exist between the medial and lateral localizations in this condition. Traumata had occurred in all the lateral cases except one, and in most of

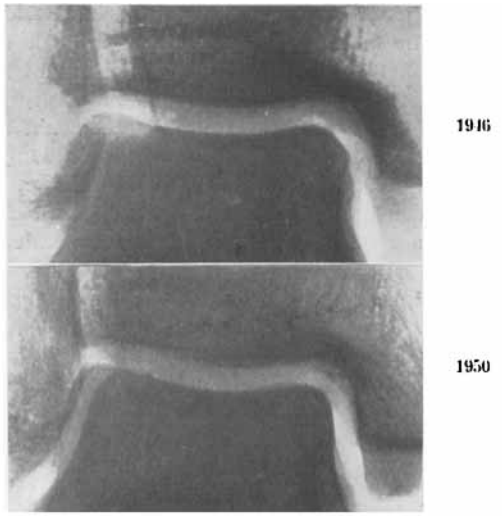

Fig. 7.
Roentgenogram from 1946 (top) and from 1950 (bottom).

them had been very severe; whereas in many of the medical cases there was no history of trauma, and where a trauma had occurred it was often so slight as to be of very doubtful etiologic significance. On going through the cases in the literature–40 medial and 10 lateral– we found that 17 of the former and all of the latter cases had a history of more or less severe trauma (see table I).

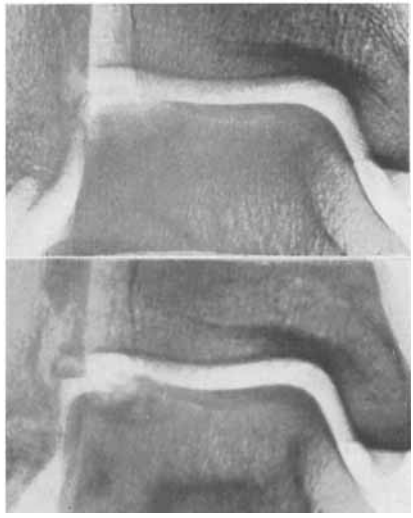

Fig. 8.
Upper roentgenogram from 1945 and lower from 1950.

These findings led us in 1950 to conclude that the lateral lesions often have a traumatic etiology, even though we were unable at that time to present conclusive evidence. In the continued analysis and amplification of the series, we collected seven cases in which fractures at the lateral margin of the trochlea tali had assumed an appearance resembling osteochondritis dissecans. Definite adduction trauma were present in each of these seven cases.–Marks (1952) has described a similar case. We feel, therefore, that we have now furnished likely evidence of a traumatic etiology, as a rule, in so called lateral osteochondritis dissecans.

We have gone through the Karolinska Sjukhuset files containing roentgenograms of talocrural joints in cases of acute traumata, but among those with traumatic lesions (about 4,000 cases) we have not found any with similar damage at the medial superior margin of the talus. Indeed, such a finding was scarcely to be expected in view of the anatomic conditions. The medial superior edge of the talus forms an obtuse angle, whereas the lateral edge forms a right angle or even an acute angle (see fig. 4). In a first-degree adduction trauma the lateral superior margin of the talus may, therefore, be forced against the lateral malleolus and result in the avulsion of a flake of bone and cartilage. An abduction trauma is not likely to produce a similar lesion of the medial superior margin of the talus. In view of this circumstance, together with the fact that such a large proportion of cases with medial osteochondritis dissecans have no history of trauma, we consider that in such cases traumata are generally of minor significance or none at all.

The medial and lateral forms differ, too, in the symptomatology. Whereas the latter forms, in this series, were generally associated with pronounced symptoms, most of the medial ones produced only very slight symptoms. This circumstance is also reflected in the number of operations, for which the indications were mainly symptomatic: surgical intervention was resorted to in seven of 19 lateral cases but in only seven of 41 medial ones. These differences have not been evident in the small series reported by other authors.

Some of the non-operated patients showed healing and subsequent freedom from symptoms. In a few others, although the lesions healed, there were persisting symptoms of arthrosis, as well as roentgenologic evidence of increasing arthrosis changes. All of the operated patients showed local healing, and most of them became symptom-free. Those who had post-operative symptoms also presented roentgenologic signs of increasing arthrosis changes. From these findings the conclusion that conservative treatment ought not to be unduly prolonged if there

are no roentgenologic signs of healing seems justified, since the risk of arthrosis lesions probably increases. A further argument for surgical intervention is that the operation is relatively minor with our method. We have found the more extensive operations of other authors (e.g. Ray and Coughlin) to be unnecessary.

SUMMARY

1. In osteochondritis dissecans of the talus, the medial and lateral localizations appear to differ both from the etiologic and from the therapeutic point of view.

2. The medial forms seem to have an atraumatic etiology; the lateral forms, on the contrary, apparently follow trauma to the bone.

3. The symptoms are notably of arthrosis type (painful weight-bearing) and are probably caused by a state of irritation due to the degenerative process. Loose bodies produce symptoms of locking.

4. Symptom-free cases should not be submitted to surgical treatment. Cases with loose bodies and symptoms of locking should be operated on, and likewise those with long-standing symptoms associated with roentgenologic evidence of necrosis. The symptoms appear to be more pronounced in lateral than in medial involvement, and spontaneous healing seems to be rarer in the former, so that operative treatment should be considered earlier. The incidence of arthrosis deformans is strikingly high even in cases where the traumata have been of mild character–which finding should point to earlier operative treatment.

RESUME

1. Dans l'ostéonchondrite disséquante de l'astragale, les localisations médiales et latérales différent d'un point de vue étiologique et thérapeutique.

2. Les formes médiales semblent avoir une étiologie atraumatique; les formes latérales, au contraire, sont apparemment dues à un trauma de l'os.

3. Les symptômes sont plutôt du type de l'arthrose (support de poids douloureux) et sont probablement causés par une irritation due à un processus dégénératif. Des fragments détachés peuvent bloquer l'articulation.

4. Les cas dans lesquels aucun symptôme ne se manifeste ne doivent pas être soumis à un traitement chirurgical. Les cas dans lesquels des fragments se sont détachés et bloquent l'articulation doivent être opérés, de même que ceux chez lesquels des symptômes persistants

se sont manifestés, s'associant à des nécroses dont l'existence a été reconnue à l'examen radiologique. Les symptômes semblent être plus prononcés dans les cas de localisations latérales que dans ceux où elle est médiale et la guérison spontanée semble plus rare dans les premiers, pour lesquels il y a donc lieu d'envisager très tôt un traitement opératoire. L'existence d'arthrose déformante est très élevée, même dans les cas où le traumatisme a été d'un caractère bénin – ce qui semble bien indiquer qu'il y a lieu d'instituer rapidement un traitement chirurgical.

ZUSAMMENFASSUNG

1. Bei der Osteochondritis dissecans des Talus scheint die mediale und die laterale Lokalisation sich in ursächlicher und therapeutischer Hinsicht verschieden zu verhalten.

2. Den medialen Formen scheint eine atraumatische Ursache zu grunde zu liegen, während die lateralen Formen anscheinend den Folgezustand eines Knochentraumas darstellen.

3. Die Symptome weisen vor allem die Kennzeichen der Arthrose auf (Belastungsschmerzen) und werden wahrscheinlich von dem durch den degenerativen Prozess bedingten Reizzustand hervorgerufen. Freie Gelenkskörper geben Veranlassung zu Einklemmungssymptomen.

4. Fälle ohne Symptome sollten nicht operativ behandelt werden. Fälle mit freien Gelenkskörpern und Einklemmungssymptomen soll man operieren, ebenso Fälle, die zusammen mit langdauernden Symptomen röntgenologische Zeichen von Nekrose aufweisen. Die Symptome scheinen bei lateralem Sitz der Erkrankung ausgesprochener zu sein als beim medialen und Spontanheilung scheint im ersteren Falle seltener vorzukommen, so dass früheres operatives Eingreifen bei der lateralen Form angezeigt erscheint. Das Vorkommen von deformierender Arthrose ist erstaunlich häufig selbst in Fällen in denen das Trauma verhältnismässig leicht war – eine Beobachtung, die möglicherweise auf die Notwendigkeit frühzeitigen Operierens hinweist.

REFERENCES

Aronsson, H.: Über Osteochondritis dissecans im Fussgelenk. Zbl. chir. 69, 312–323, 1942.

Axhausen, G.: Zum Vorkommen des Abgrenzungvorganges am Fussgelenk. Zbl. chir. 55, 322, 1928.

Breitländer: Osteochondritis diss. tali. Arch. klin. chir. 148, 149, 1927.

Brickey, P. A. and *Grow, J. B.:* Osteochondritis diss. Amer. J. Surg. 48, 463, 1940.

Burr, R. C.: Osteochondritis diss. Canad. med. Ass. J. 41, 232, 1939.

Cobey, M. C.: Osteochondritis diss. of the Astragalus. Milit. Surg. 93, 184, 1943.

Cordes: Osteochondritis diss. im Sprunggelenk. Zbl. chir. s. 1252, 1927.

Deutschländer, C.: Zur Kenntnis der gelenknahen Nekrosenherde im subchondralen Knochenmarkraum. Zbl. chir. 55, 469, 1928.

Diaz, Garcia: Un cas d'osteochondite de l'astragale. Bull. mém. Soc. nat. chir. 54, 986, 1928. (Ref. Z. org. Chir. 43, 750, 1928).

Fairbanks, H. A. T.: Osteochondritis dissecans. Brit. J. Surg. 21, 67, 1933.

Harms: Osteochondritis diss. an der Talusrolle. Zbl. Chir. s. 1017. 1927.

Hellström, John and *Östling, Karl:* Ein klinischer Beitrag zur Kenntnis der Osteochondritis dissecans. Acta chir. scand. 75, 273, 1934.

Hoffmann: cited by Myhre.

Jansson, Gösta: Osteochondritis dissecans (König) vom röntgenologischen Gesichtspunkt aus betrachtet. Acta Radiol. 11, 33, 1930.

Kappis, M.: Osteochondritis dissecans und traumat. Gelenkmäuse. Dtsch. Zsch. Chir. 171, 13, 1922.

König: Über freie Körper in den Gelenken. Dtsch. Zsch. Chir. 27, 90, 1887–88.

Läwen, A.: Über osteochondritis dissecans am Talocruralgelenk und ihre operative Behandling. Zbl. Chir. s. 2498, 1929.

Mensor, M. C. S. and *Melody, G. F.:* Osteochondritis dissecans of ankle joint. The use of Tumography as a diagnostic–. J. Bone. Joint Surg. 23, 903, 1941.

Marks, K. L.: Flake fracture of the talus progressing to osteochondritis dissecans. J. Bone and Joint Surg. 34 B. 90, 1952.

Myhre, H.: Osteochondritis dissecans trochlea tali. Acta Radiol. 20, 272, 1939.

Nielsen, M. Aage: Osteochondritis dissecans capit. humeri. Acta osthopedica scand. 4, 307, 1933.

Radicke: Beitr. klin. chir. 150, 1930.

Ray, R. B. and *Coughlin, E. J.:* Osteochondritis dissecans of the talus. J. Bone, Joint Surg. 29, 697, 1947.

Ribbing, S.: Studion über hereditäre, multiple Epiphysenstörungen. Acta Radiol. suppl. 34, 1937.

Röden, S., Tillegård, P. and *Unander-Scharin, L.:* Osteochondritis dissecans tali. Nordisk Medicin. 45, 324, 1951.

Sidler, A.: Über Gelenkknorpellösung und Gelenkmausbildung im Oberen Sprunggelenk. Zsch. Orthop. Chir. 52, 302, 1929–30.

Vaughan, C. E. and *Staphelton, J. G.:* Osteochondritis dissecans of the Ankle. Radiology. 49, 72, 1947.

Vogel, K.: Ein Fall von Knochenerweichung (Köhler, Keinböck u. a.) im Sprungbeinhals. Zbl. chir. 2510, 1927.

Wolff, Aage O.: Osteochondritis dissecans i talocruralleddet (Dansk radiol. Selskabs Forhandlingar 1925). Hospitals Tidende 1926.