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STUDIES ON PATELLAR CHONDROMALACIA

An attempt to elucidate its etiology.

In order to be able fully to understand the various aspects of patellar chondromalacia and, above all, be able to conceive the mechanism in the origin and development of this disease, it naturally is necessary to have at least some knowledge of the anatomy and physiology of the articular cartilage. Notwithstanding the investigations that have been carried on for many years, no agreement has been reached yet in the views of certain questions in this connection, and this applies in particular to the nutrition of the cartilage.

While the cartilage in the embryonic stage and early childhood is richly vascularized and while blind ends of a few blood vessels are seen now and then in the "juice spaces" in adults, the articular cartilage normally is devoid of every trace of any blood supply proper. As the hyaline cartilage has a specific structure, namely arcuate formations with the vertex directed towards the articular surface, it may be that this lamellar arrangement offers certain possibilities for the appearance of the so-called "juice channels", through which the nutrition is supposed to take place, owing to some kind of filtration pressure. According to several investigators the intercellular substance is properly to be looked upon as the site of these biological processes, whereas other authors think that no particular localized diffusion or osmosis might be established.

Theoretically, then, nourishment might possibly be supplied to the cartilage in three ways: through the blood vessels at the periphery, in the marginal zone; through connection with the

blood vessels of the bone; or from the vessels of the synovial stratum. Besides, of course, one might reckon also with a combination of two of these ways of supply, or all three.

Among those who consider the synovial stratum the most important means of transporting nourishment to the cartilage, I may mention Leriche and Policard, while Ishido reckons mostly with the marginal vessels and, for instance, Barth with the vessels of the bone. Timbrell and Fischer believe in a combination of all three possibilities, while Axhausen, Wollenberg, Nussbaum, and others think that the deeper layers of the cartilage get their nutrition from the bone, while the upper layers are supplied from the synovial stratum (and it has been claimed that these layers possibly are separated by a distinct border line).

In support of the view that the synovia might be able to nourish the cartilage it has been pointed out that loose bodies may live and grow larger in the joint cavity, and that in necrosis of the head subsequent to fracture of the neck the cartilage may nevertheless remain intact for a number of years after the accident. Regressive changes in the cartilage in infectious lesions might just as well be attributable to metabolic disturbances due to circulatory disorder via the bone substance as via the synovia, which likewise undergoes great changes of an entirely chemical nature.

In connection with these pathological processes in the cartilaginous substance, the cartilage is also undergoing some softening. Its water content, which normally is about 50 %, is increased, and finally the elasticity of the cartilage diminishes—something that naturally is of the greatest practical importance to the funtion. For the articular cartilage does not only smooth off the articular surfaces, completing their congruence and thus reducing the friction; it also acts as a buffer spring, and delays impulses. The elasticity together with the thickness of the cartilage is a decisive factor in this respect. If now one or both of these properties of the cartilage is deranged, it is evident that the cartilage cannot function normally.

Anatomically as well as physiologically the patella is to be

looked upon as a joint socket, and sockets are always lined with a cartilage that is softer than that of the corresponding joint head. The cartilage of the patella, which is strongly developed, is very vascular, and consequently it is more apt to undergo pathological changes when exposed, for instance, to mechanical strain. The pathological processes we find in hyaline cartilage are as a rule localized primarily to the intercellular substance, and it is only secondarily that the cartilage cells themselves undergo alteration.

According to Heine, the simplest degree of degeneration of hyaline cartilage is the so-called swelling of the cartilage. A later stage, according to Weichselbaum, is crevice formation due to partial solution of the matrix, and later, as the intercellular dissolution becomes more or less generalized the crevice formation goes on to a more general "fibril formation". Subsequent to mechanical injuries of the friction type, these fibrils may be "gnawed off" and the cartilage is reduced in thickness. Where the friction is greatest, the resulting defect will be most pronounced too, and when this morbid process concerns the patella it is the central and medial facets that are most involved.

As to the patellar cartilage, it normally is unusually loose in build, and the intercellular substance is mostly dissolved more or less into fibrils. This looseness increases with the years, causing a decrease in the elasticity. As the patellar cartilage is so rich in blood vessels, however, one may readily imagine that the hydraulic and hydrodynamic laws will apply here too like in other cases when kinetic energy is supplied to a fluid-containing encapsulated organ. Through its contact with the articular surfaces of the femur the patellar cartilage has not any great chance of getting out of the way when exposed to a straight anterior blow, whereas it shows tangential displacement in surface sliding. Owing to these anatomical conditions, the patellar cartilage might to some extent perhaps be compared to a water-imbibed delimited organ, to which, as is well-known, the hydrodynamic laws apply.

As in patellar chondromalacia the patellar cartilage has an increasing water content with the increasing degeneration, the

hydrodynamic aspect will assert itself more and more, and thus we have to reckon with a certain vicious circle inasmuch as the changes that have developed increase the explosive effect of the impact, so that the cartilaginous changes in turn are increased distinctly. In patellar chondromalacia we find the just mentioned changes in Aleman's three-stage softening, the finer crack formation, and finally the "fluff formation", sometimes with defects extending down to the bone.

According to this reasoning, then, the patellar cartilage is exceedingly liable to exposure to regressive pathological processes, partly on account of the structure of the tissue itself, partly on account of the anatomical location of the patella that allows a single trauma as well as continual traumatism to assert themselves perceptibly in this part of the body. Pommer has even been able to demonstrate areas of necrosis in the patellar cartilage subsequent to a contusion—a finding which, among others, has been taken to indicate the great significance of traumatic injury to the development of patellar chondromalacia. Slowick has likewise reported two cases of patellar chondromalacia after contusion, in which roentgenography at first showed changes in the bone of the patella, and later these changes disappeared. Also Aleman claims that the type of patellar chondromalacia which he calls post-traumatic is due to exogenous factors—namely, the force from some sort of a blow—and Läwen holds the same view.

With a view to our present knowledge of the biology of articular cartilage, one would perhaps often be inclined to subscribe to this apparently perfectly plain and relatively simple explanation of the origin of patellar chondromalacia. But it is still an unsettled question whether the trauma causes fine structural changes in the cartilage or affects its nutrition, or whether the regressive processes in the cartilage are due primarily to some sort of change in the circulation, independent of a subsequent trauma, or possibly through changes in the biological or purely chemical properties of the synovia.

Often, however, we find patellar chondromalacia in different morbid conditions without traumatic injury as in toxinæmia—

for instance, in tuberculosis—bacteriæmia, or endocrine disturbances with endocrine arthrosis. It seems obvious, therefore, that the trauma does not play as great a rôle in this connection as has been assigned to it. If so, perhaps it will be more correct to reckon with a cooperation of several factors, as it may be difficult in a given case to decide how much effect is to be attributed to the individual etiological factors.

If the kinetic energy of a trauma per surface unit is sufficiently great, of course, it will bring about some changes in the cartilage without it being possible macroscopically to find any sign of chondromalacia. Such a lesion may develop subsequently, or the trauma may bring about a pathological process in the hyaline articular cartilage, which has no capacity for regeneration, and here this process may remain, so to speak, encapsulated, without ever progressing. Thus the difference between these "occult" changes in the cartilage and chondromalacia proper would be entirely quantitative, pretty much in analogy with colliquative necrosis in the fibrocartilage of the menisci and the multiple or solitary ganglia.

If the trauma played the important rôle which some authors have assigned to it, I think, there ought to be found some agreement between the occurrence of exogenous violence against the patella and the appearance of chondromalacia. Clinically, however, the latter condition does not give any particular diagnostic difficulties, at least not when it has reached a macroscopically recognizable stage. So, by establishing the crepitations localized under the patella as pathognomonic of this affection—as pointed out and described thoroughly by Aleman—it is easy to get a survey of the frequency of patellar chondromalacia, and at the same time one may look into the frequency of "conscious" traumata.

I have investigated this question in 640 cases, and I shall refer to the findings recorded in the tables and curves here presented.

As the clinical investigation concerning the frequency of the lesion agrees very well with the observations made by Heine and by Silfverskiöld on autopsy materials, and to some extent also

Frequens of Patellarchondromalaci:

	age 11—50 years			age 11—30 years		
	cases	P. C.	%	cases	P. C.	%
men	202	62	30.7	173	49	28.3
women	266	80	30.1	235	48	20.4

Distribution of P.C. according to work:

Men in the age of 11—50 years. — number of examinated 139.

	no P. C.	P. C.	no. of cases	%
manuel workers	60	30	90	50
brain workers	36	13	49	36.1

Patellarchondromalaci and skating:

	P. C.	no P. C.	% P. C.
skating pers.	119	224	34.6
others	39	45	46.4

Fig. 1.

with Aleman's statistics of arthrotomies, it ought to signify for sure that the establishment of the crepitations described above implies a rather fair possibility of ascertaining the occurrence of patellar chondromalacia, at least after it has developed enough to become visible.

As yet we do not know definitely how long it takes this change in the cartilage to develop after a given trauma. From experimental studies, however, it seems probable that chondromalacia at least takes two, may be four, months for its development.

The earliest age at which patellar chondromalacia could be ascertained in my material was 10 years, while previously this condition had not been observed till the age of 13 years. In these cases, at any rate, it would be a little difficult to consider the

Account of the investigation of Trauma and P.C.

	no. of cases men	no. of cases women
bilateral trauma bilateral P.C.	10	7
bilateral trauma unilateral P.C.	1	3
bilateral trauma no P.C.	16	1
no trauma bilateral P.C.	35	68
no trauma unilateral P.C.	16	2
no trauma no P.C.	108	137
unilateral trauma bilateral P.C.	19	16
unilateral trauma unilateral P.C. on the same side	2	5
unilateral trauma unilateral P.C. on the other side	0	3
unilateral trauma no P.C.	17	45

number of cases examinated: 594.
Agreement between trauma and P.C. — 66.9 %.

Fig. 2.

lesion as the first sign of advancing age. As a rule, however, it may perhaps be right, as pointed out by Silfverskiöld, to hold the view that patellar chondromalacia often is the first sign of a beginning senile change in the organism. As is well-known, chondromalacia is considered a precursory stage of arthrosis

deformans, and the curves for the frequency of the lesion show also a striking parallelism with the curve for arthrosis deformans, only with a shift in the age of about 15 years.

The following schematic recapitulation may serve as a survey of the features pointed out above:

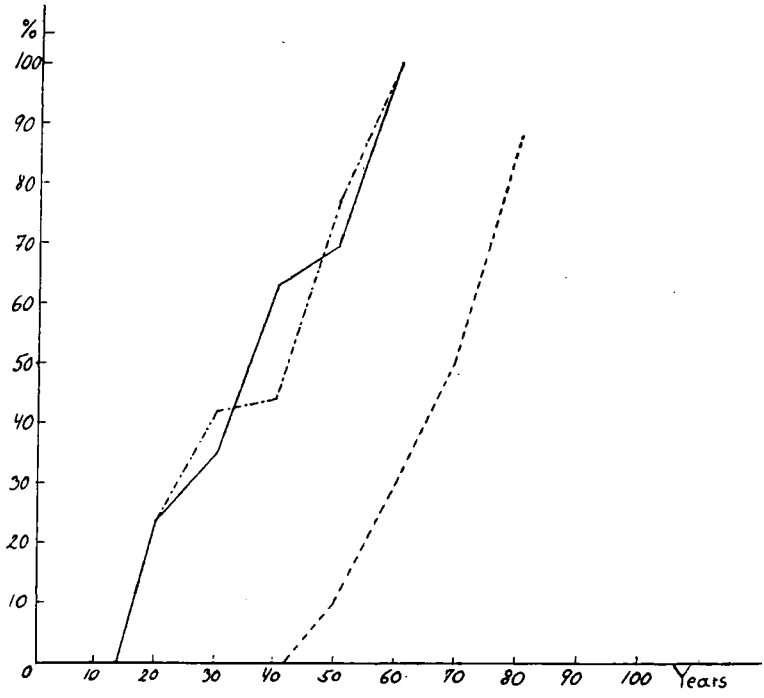

Fig. 3.

———— Frequens of P.C. after Silfverskiölds examinations in dead bodies.
—·—·—·— Frequens of P.C. after Hinricssons clinical examinations.
- - - - - - - - - - Frequens of Arthrosis deformans after Silfverskiölds examination in dead bodies.

From a physiological point of view the patella constitutes a joint socket in which, like in other structures of this kind,

the cartilage is softer than that of the corresponding joint head —a cartilage which normally has an unusually loose structure,

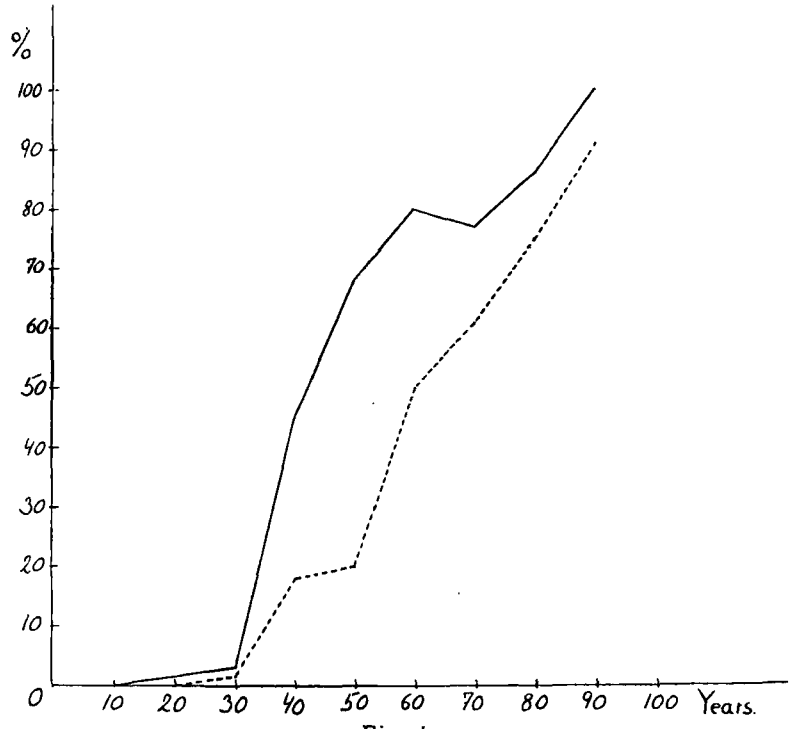

Fig. 4.

After Heine. Frequens of P.C.: ——— in patella ------ in fascies patellaris femoris.

where the intercellular substance most often is dissolved into fibrils, more or less.

On account of its anatomical location the patella is particularly exposed to traumatic injury—as well the single trauma type as the traumatism type.

The decisive point in the effect of a single trauma is mostly its kinetic energy per surface unit. The cartilage which serves, among other things, the purpose of acting like a spring and

distributing any impact over a greater surface has a retarding effect on the propagation of the blow. But, as the cartilage most often is made up by more than 50 % water, it seems probable that the hydrodynamic laws apply to these propagations of energy here like in several internal organs that are rich in fluid, so that the effect on the cartilaginous tissue is increased in this way.

As the patellar cartilage normally is exposed to intermittent pressure, its elastic mass, which is intersected by "juice channels", will alternatingly be compressed and return to its normal state, or in certain cases expand somewhat, so that the synovial fluid is thus pumped about within the cartilaginous substance.

On the background of this view of the physiology of the patellar cartilage, the appearance of patellar chondromalacia ought to be explainable as resulting from a cooperation of various factors, where a sufficiently hard trauma or a sufficiently severe traumatism in certain cases is to be assigned some rôle as an etiological factor.

Investigation of 604 cases has shown, however, that there is a very marked incongruence between the frequency of traumatic injury and the occurrence of patellar chondromalacia. Only about two thirds of the cases (66.9 %) showed an agreement in this respect (this comparison comprises merely the age-classes from 21 to 50 years, about one third of the cases — 34.5 %). Moreover, as is well-known, patellar chondromalacia has often been seen to develop in such morbid conditions as, for instance, toxinæmia and bacteriæmia, and also in endocrine disturbances with the so-called endocrine arthroses, where changes in the metabolism of the cartilage conceivably may result. So it seems quite justified to question whether cooperation of some of these or other factors might not be the direct cause of the development of patellar chondromalacia.

In this connection one may also question whether it is quite appropriate to designate a certain type of chondromalacia as post-traumatic. For, judging from the investigation just men-

tioned, it appears at least as if the single trauma does not play the decisive rôle that has been assigned to it.

This investigation has further established that the frequency of patellar chondromalacia within different age groups is about the same in these clinical studies as in previous pathologic-anatomical investigations, and that there is no demonstrable difference in the sex distribution of this lesion. On the other hand, persons occupied with hard manual work show a higher frequency of this lesion than do the so-called brain workers—namely, 50 % against 36.1 %, respectively.