LECTURE XXXVIII.

DRY ARTHRITIS OF THE KNEE.

I. Case of a patient affected with dry arthritis of both knees. II. On the left the arthritis is at the same time dropsical and deforming, knock-kneed—Anatomical explanation of the symptoms by synovial congestion, probable wearing away of the cartilages, absorption, after rarefaction, of the cancellous tissue of the outer condyle of the femur—Ultimate course of the disease; its incurability—The name dry arthritis is the only one we have to express this group of symptoms. III. In the right knee, considerable increase in size of the end of the femur, very marked lateral mobility, loud crackling—Subluxation of the tibia—Explanation of these symptoms by the formation of osteophytes, destruction of the ligaments, eburnation of the articular surfaces. IV. Cases of other patients affected with dry arthritis of the knee—Principal varieties of this disease.

GENTLEMEN: I. We have at No. 25, ward Ste. Vierge, a man 58 years old, whom we shall not keep very long, although both his knees are diseased, for we cannot cure him. He has been suffering for seven years, without apparent cause, or at least without his being able to attribute it to any traumatic lesion. He only knows that his left knee has often been swollen, and that he has been treated for dropsy of it. The right one does not appear to have been the seat of a similar effusion, but the patient has also suffered in it almost constantly, sometimes a little more, sometimes a little less, without the pains having ever been excessive and accompanied by fever; but the patient has often been obliged to enter the hospitals, leaving them, after a month or two, ameliorated but not cured. He has walked with more and more difficulty, and finally was no longer able to do it without the aid of crutches, and even then with much difficulty and fatigue. We find the following condition of affairs.

II. In the left knee, a very evident effusion, without enlargement of the patella, without hyperostosis of the femur, without appreciable thickening of the synovial membrane, without movable or immovable foreign body; no lateral mobility; voluntary movements but slightly limited, notwithstanding the pains; communicated movements of flexion and extension almost as free as in the normal condition; perception by the hand and ear of very marked cracklings during both voluntary and communicated movements; finally we find that deformity which is known by the name knock-knee, and which consists in the deviation of the leg outwards, and in the very marked prominence of the inner condyle of the femur.

What is the anatomical explanation of these symptoms, what will be the ultimate course of the disease, and what name should we give it?

I explain the effusion, as I did in simple hydrarthrosis, by a congestion of the synovial membrane with exaggeration of its secretory function, and diminution of its power of absorption. But I suppose, for autopsies have shown it in several cases of this kind, that the congestion is not general, and that it occupies more particularly the folds known as the synovial fringes.

I attribute the sensation and sound of crackling to a lesion which has often been found in the autopsies, that is, the loss of polish and the partial destruction of the diarthrodial cartilages. I do not know exactly to what degree this destruction has advanced, but recalling the lesions described by Redfern and M. Broca, lesions which I have already had occasion to mention when speaking of the other varieties of arthritis, I believe that the arrangement in parallel fibres which constitutes the velvety condition, and its coincidence with a partial disappearance of the cartilage, cause the crepitation which now occupies us. I know that pathological anatomy leaves a gap here, for I have never had, and no one, so far as I know, has ever had the opportunity to dissect subjects in whom this symptom had predominated, and to see exactly what lesion had produced it. Moreover you might raise the objection that I have already spoken of—possible velvety change in patients who had offered no crepitation. That undoubtedly was due to this, that the movements, limited by the pain, by the rigidity of the synovial membrane, by commencing adhesions, and above all by muscular contracture, were not sufficiently extended to cause this loud friction; for I now call your attention to the fact, as I shall undoubtedly do again, that one of the characters by which this patient's arthritis differs from those of which I have heretofore spoken, is that the muscles are not contractured and immovable, or are so to a much less degree.

As to the deformity, we must not consider it congenital, as the knock-knee often is. For the patient tells us that his knee was always well shaped, and that the deformity appeared only five or six years ago and has increased little by little. It is then accidental, and I cannot explain it otherwise than by a rather vaguely described lesion of which I, for my part, have not thus far been able to give an anatomical demonstration. I refer to the sinking of the outer condyle caused by absorption of its substance, absorption undoubtedly prepared by rarefaction. I have already often spoken to you of rarefaction of the cancellous tissue; I told you that it sometimes accompanied one of the varieties of osteitis, but that it might also occur without osteitis, that then it coincided with an infiltration of fat, and that it was a result of age. I spoke to you of fractures made possible by this rarefaction, but I have not yet had occasion to show you the concomitant disappearance of a large part of the rarefied cancellous tissue. It is precisely that which seems to me to have taken place in this patient. The same thing probably occurs in other patients affected with this or some other accidental deformity, hence the name deforming arthritis used by some authors.

What will be the ultimate course and termination of this disease? Observe well two principal things: 1st. The arthritis, although it has

lasted a long time, has not suppurated; 2d. It has not caused anchylosis, even incomplete. Now by the phenomena which we have found, by what has taken place in the right knee, of which I shall presently speak, by the age of the patient, by what the clinic and pathological anatomy have taught us about this subject, I am convinced that the disease will go on in the same way. The arthritis will persist, without terminating either by suppuration or by anchylosis. Why so? Because there is, in the phlegmasia of this articulation, a nature, a mode, as they still say, a tendency, as I have also said, which does not lead to these results. The articulation will not suppurate, because it is not fungoid, and because the patient's constitution is not scrofulous; it will not anchylose, because there is neither the rigidity by thickening, nor the false membranes which characterize plastic arthritis, and perhaps also because, as the muscles continue to act a little, the movements, slight as they may be, will prevent the rigidity and adhesions from establishing themselves to the degree necessary to produce anchylosis. I can carry the explanation no further. It is evident that here we touch upon the question of the intimate nature of the disease, and, as in all questions of this kind, we are stopped by the unknown.[1]

And now what name shall we give this affection? After what I have just said, it will be neither that of congestive arthritis, nor that of plastic arthritis, nor that of fungoid arthritis. It might, strictly speaking, be that of dropsical arthritis, for there is liquid in the articulation. But no, I shall not employ this expression either, for it would give you false ideas upon the prognosis and treatment. In fact, to these words, dropsical arthritis and hydrarthrosis, is attached the idea of curable anatomical lesions, of temporary physiological troubles, of probable cure, in a word, and of efforts to be made by the surgeon to obtain this cure. But here you have a congestion which has become permanent, and perhaps subsequent inappreciable transformations into cartilaginous tissue about the synovial fringes; you have irremediable lesions of the diarthrodial cartilages and the epiphysis. None of that will disappear. The tendency is not to get well, but rather to get worse by the development of other disorders of which the present lesions are only the prelude, and of which the right knee will show us examples.

There is only one word, in the present state of surgery, to express both the lesions and the very curious nature of this disease—it is that of dry or deforming arthritis. Undoubtedly it will seem strange to you that I call dry arthritis a disease which has an effusion for one of its manifestations. But I have to use a word which will make you feel that it is not an ordinary hydrarthrosis, and I have no other

[1] An apparent contradiction may be found between the characters which I give to dry arthritis of the knee, and those which I gave, page 54, to the arthritis of the tarsalgia of adolescents. But there I made only a comparison; I did not wish to establish a complete identity. If the tarsal arthritis of adolescents resembles dry arthritis by the lesion of the cartilages, it differs from it by its accompanying reaction upon the muscles, by possibility of anchylosis, and by its curability after probable repair of the ulcerated cartilages.

than those I have just used. But I shall say more upon this point after having spoken of the right knee.

III. The physical symptom of the right knee which first strikes us, is a considerable increase in size, affecting especially the lower extremity of the femur which at the same time is rounded. Placing your hand upon this swelling you find it very hard, and, as it were, lobulated, all the hard points have the consistency of bone and are evidently united with the femur. There are similar, but much fewer, roughnesses upon the tibia. The patient can make a few movements of flexion; but it is not a muscular resistance which limits them, for, taking the limb in both hands, you can give it these movements very easily; at the same time you feel and hear a loud crackling; very extended abnormal lateral movements can also be made; and, finally, by analyzing carefully the situation of the tibial epiphysis, we see that it is placed too far behind, that it cannot be brought forward, in a word, that it is in that state of displacement which is often called subluxation.

I ask for this knee the same three questions as for the other.

And first, what is the anatomical explanation of the different symptoms? Of course the idea may at first occur that the swelling of the femur is due to an osteo-sarcoma. But if it had been that disease, during the time it has lasted it would have extended to the shaft of the femur and would have softened. Is not then this hardness that of a hyperostosis? I have avoided using this word; for hyperostosis, as we understand it, occupies the shaft and the whole thickness of the compact portion of the long bones, while here the bony swelling corresponds to the cancellous extremity, and seems to occupy the external layers rather than the parenchyma of the epiphysis. Now, we have here, gentlemen, a special lesion with which the labors of modern pathological anatomy have made us acquainted, that is, ossifications of the edge of the cartilage and the neighbouring periosteum, perhaps even of that portion of the synovial membrane which covers the latter. These ossifications are analogous to those which, in the same variety of arthritis, we sometimes find upon the inner face of the synovial membrane in the place of the fringes, and to those which sometimes become loose in the articulation and form a variety of movable foreign bodies. In a word, these are rounded concretions or stalactites, osteophytes, as they are still called, of complex origin, due to an abnormal ossifying power of the articular cartilages and of the synovial membrane. What makes them very remarkable and uncommon in this patient is that they are much more abundant and agglomerated than they generally are in cases of this kind.

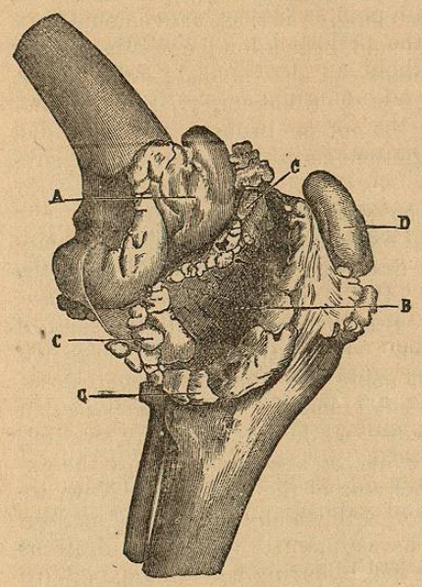
Figure 21, for example, which I borrow from M. Duplay, represents osteophytes much less agglomerated than those of our patient.

As for the crackling, I explain that also by a lesion of the cartilages; I think even that it is no longer a question here either of the velvety condition, or of the wearing away indicated by Prof. Cruveilhier in 1824.[1] I think there is a more advanced lesion, an extensive destruction of these same cartilages, and friction of the bony articular

[1] Cruveilhier, Archives de Médecine, 1824, 1re serie, tome ii.

surfaces which have become hard and eburnated. This eburnation is perhaps explained, as M. Ranvier admits, by calcification of some of the cartilaginous cells, and more probably by a hypertrophy limited to the compact sub-cartilaginous layer, which, instead of disappearing, as in fungoid arthritis, has undergone a process of condensation, in which, however, neither the parenchyma of the cancellous nor that of the compact tissue has participated; this would be the continuation of the process of ossification which takes place at the periphery, without, although for absolutely unknown reasons, the participation of the interstitial portions of the bone. In any case, you recognize here a peculiar lesion which has been described by Cruveilhier[1] under the name of eburnation or eburexostosis of the articular surfaces.

Fig. 21.



Osteophytes. A. Condyles of the femur. B. Articular surface of tibia. C. C. C. Osteophytes. D. Patella.

But should we not also attribute the crackling to lesions of the synovial membrane? I told you that there was no effusion. Might there not then be a condition of dryness which, during movement, would give crepitation? I admit that, studying the origin of this word dry arthritis, I have examined specimens which have been shown me, to see how far the synovial membrane participated in the dryness indicated; but I have never found it dry enough to account for the crackling. Indeed there was always enough humidity on its inner surface to make the expression seem to me unjustified.

Are there no other lesions of the synovial membrane in such cases? I do not speak of the congestion which probably occurred at the beginning, and which undoubtedly has now disappeared. I allude only to the fibro-cartilaginous and even bony transformations which have been pointed out as possible in the synovial fringes. If such transformations have taken place we can understand that they might contribute to the production of crackling. But there is no sign which authorizes me to affirm the existence of this lesion.

Lateral mobility is too marked in this patient for me to think of seeking its explanation, or at least its sole explanation, in the destruction of the semilunar fibro-cartilages. I do not doubt the softening and perhaps destruction of the lateral and even of the crucial ligaments. But has it been a simple absorption, or a fatty degeneration?

[1] Cruveilhier, Bulletins de la Société anatomique, 1826, tome i. p. 195.

This point has not been thoroughly studied. I only know that M. Duplay[1] makes fatty transformation of the ligaments an almost constant lesion in diseases of this kind.

As for the subluxation, it is also explained by the weakening of the means of union, and especially of the posterior ligament which would have been affected by lesions the same as those of the lateral and crucial ligaments.

In short, gentlemen, we have the explanation of the symptoms in a series of lesions affecting all parts of the joint, and consisting in: congestion of the synovial membrane, destruction of the ligaments, new ossification by means of certain points of the synovial membrane, of the articular surfaces, and of the periosteum, without false membranes leading to anchylosis, without any tendency to suppuration, without condensing osteitis of the adjoining diaphyses.

What name then shall we give the disease? As before, I am not authorized to use any of the denominations which I employed in the precedent lectures. I need one which indicates these three principal characteristics: no anchylosis, no suppuration, long duration and incurability (for there is still less reason to hope for a cure in this latter knee than in the other, because there is a too profound modification of the normal anatomical condition). I still have only the name dry arthritis to express all this. This name has the advantage of indicating a real characteristic, the persistence of an inflammatory condition which from time to time becomes subacute. It has certainly the disadvantage of exaggerating a symptom, the dryness of the synovial membrane, and of not sufficiently indicating the three characteristic tendencies I mentioned. I use it, however, because I have no other, and because I should not easily find one which would better express all the negative and positive characteristics of this variety of arthritis.

IV. Observation of the two knees of this patient has already shown us that dry arthritis presents different degrees or characters. Recall the patients I have shown you at different times in the lectures of this and the preceding years, and you will see that there are still other varieties.

We often see at our consultation, and sometimes in our wards, patients verging on old age, whose only symptoms are articular pain and cracklings. I have often pointed out arthrites of this kind after fractures of the thigh, contusions and sprains of the knee, after subacute rheumatisms, and almost always in patients who are more than fifty years old.

I showed, in 1868 and 1869, ward Ste. Catherine, No. 19, a woman 46 years old, whose left knee, following an attack of rheumatism with hydrarthrosis, had been painful for several years and the seat of an infirmity, the principal cause of which was an excessive mobility which seemed to indicate a destruction of all the means of union. You remember that the lateral mobility was very great, and that I was even able with my hands to move the tibia outwards, inwards, forwards and backwards, so as to produce a subluxation in these

[1] Follin et Duplay, Traité de Pathologie, tome ii.

different directions. You remember that all these movements caused a dry sound which was heard by those standing about the bed, and which must have been caused by the contact of very hard surfaces, probably the eburnated articular ones. At that time the hydrarthrosis was slight. It should be noted also that I had made, a few months before, an injection of the tincture of iodine, which, strange to say, had not caused acute arthritis, or, consequently, the anchylosis I sought, as if the articulation had lost, at the same time with its principal anatomical dispositions, its aptitude to inflame under the influence of a severe irritation. The name dry or deforming arthritis was then the only one that I could give to this disease.

Hence I concluded that, clinically, we have to distinguish at least four varieties of dry arthritis: a first and very common one, in which the crepitation and the moderate but habitual pains are the dominant phenomena, and in which, doubtless, the lesions affect only the synovial membrane, which is congested, and the diarthrodial cartilages, which are eroded and scratched; a second, in which a temporary or permanent synovial effusion is added to the preceding lesions and symptoms; a third, in which there is lateral mobility and more or less subluxation indicating the destruction of the lateral ligaments; and a fourth, in which, with or without destruction of the lateral ligaments, we find osteophytes, either in the synovial membrane, or about the diarthrodial cartilages; furthermore, different degrees of deformity by partial osteo-malacia of the articular extremities may be found in all these cases.

Etiology.—I have carefully questioned the old man, the present patient, and have found no cause to which I could refer the affection of his knees. But as both articulations were attacked at the same time, and as we are always disposed to explain multiple arthritis by a rheumatic diathesis, I am willing to admit, although the patient has had no other rheumatic manifestation, that the disease is due to this general cause. I should, however, tell you that you must expect to see dry arthritis appear in subjects who are not rheumatic. In those cases where it is absolutely single a certain complaisance is needed to admit the rheumatic diathesis. Also when it follows a traumatic lesion, a wound, a contusion, a sprain, a fracture, this same cause cannot be invoked. That is why I am still obliged to appeal to an unknown cause in those cases in which rheumatism cannot be legitimately admitted. Is it not also necessary, even in those cases where the latter is admissible, to suppose a particular form of the diathesis which gives rise to these manifestations so special and so different from those by which rheumatism generally betrays itself?

On account of what precedes, I regret that our learned colleague, M. Charcot, has given the anatomical description of dry arthritis under the title of chronic rheumatism[1] On the one hand, the arthritis in question is not always rheumatic; and, on the other hand, rheumatism, as I have told you, gives rise to other forms of curable chronic arthritis (plastic and dropsical). There is then a disadvantage in giving

[1] Charcot, Leçons sur les Maladies des Vieillards et les Maladies chroniques. Paris, 1868.

a description of chronic rheumatism, which leaves the impression that this disease leads inevitably to the incurability of dry arthritis.

If you wish to confine yourselves to the information furnished by the clinic, do not forget, gentlemen, that dry arthritis of the knee, of which thus far the anatomical characteristics have been studied far more than the symptomatology and the etiology, presents, with reference to its origin, two forms: it is primitive or consecutive. When it is primitive, it may be rheumatic, as I told you, or of another nature which remains unknown to us. But most frequently it is consecutive to one of the forms, with which you are acquainted, of acute, subacute, or chronic arthritis. You see it, I repeat, after neighbouring contusions, sprains, fractures, which have given rise to one of the arthrites which I have just mentioned. You see it also after pure rheumatic arthritis, which at first takes on the characters of plastic arthritis, and then terminates by the lesions of dry arthritis instead of by resolution or anchylosis. Finally, you see it after arthritis, which has been at first purely dropsical. You may also see it, as M. Charcot's work testifies, after gouty arthritis, that in which the initial lesion is the invasion of the cartilaginous cells, and the other constituent parts of the articulation, by an excess of uric acid and urate of soda contained in the blood.[1]

You will more rarely see fungoid arthritis followed by dry arthritis, because, as you know, it tends rather to terminate by suppuration and sometimes by anchylosis, and also because it belongs to the youthful period of life, and because there is one almost necessary condition for the development of dry arthritis, that is, senility.

Indeed, gentlemen, and it is with this final consideration that I shall terminate the etiology, the tendency of arthritis to end neither by resolution, nor by anchylosis, nor by suppuration, and to be accompanied in certain cases by ligamentous destructions and osteophytes, is rarely seen before the age of fifty years, and developed especially in old age. But it happens in this, as in the rarefaction of the cancellous tissue, that certain subjects have this pathological aptitude a little earlier, say from forty to fifty years of age, and in consequence of a premature local senility.

Treatment.—I have nothing useful to say about the treatment. We have only to treat the new inflammatory attack by rest and poultices. As soon as it ends we shall advise compression of the dropsical knee with a flannel band, and a roller bandage, including a posterior splint for the right knee, which is so movable, and we shall let the patient resume the use of his crutches, without which he cannot now get along. If he asks, on account of his infirmity, which is only too real, admission to Bicêtre,[2] I shall obtain it for him.

[1] While normally only traces of uric acid exist in the blood, from 5 to 17 centigrammes are found in 1000 grammes of blood during an attack of the gout.

[2] Hospital for indigent, infirm, and incurable old men, situated just outside Paris.