we shall still be able, at the end of three months, to use successfully the movements of which I spoke; and, on the other hand, if permanent anchylosis should occur, such a result would be less unfortunate for the patient than persistency of lateral mobility. For the latter exposes the knee, at every moment, to new sprains and, consequently, to renewed arthrites, which are more troublesome than complete anchylosis.

There will probably be, in all three patients, a final indication, that of favouring the restoration of movements and diminishing prolonged rigidity. But as the means appropriate to that are the same as in cases of spontaneous arthritis, I will speak of them when treating of the latter.

LECTURE XXXV.

ACUTE AND SUBACUTE SPONTANEOUS ARTHRITIS OF THE KNEE.

I. First patient affected with acute arthritis of the right knee, gonorrhœal, with contracture of the flexors—Straightening of the limb under ether—Afterwards, discovery of lateral mobility and crepitation—Explanation of these two symptoms. II. Second patient affected with single acute arthritis, probably rheumatic, of the right knee. III. Formation of a complete anchylosis in both cases, notwithstanding the efforts made to prevent it—Study of the lesions—Congestion—Plastic deposits, whence the name plastic or anchylosing arthritis—Explanation of the anchylosis by the establishment of adherences after a struggle between the tendency towards resolution and the adhesive tendency. IV. Therapeutical indications based upon these ideas.

GENTLEMEN: You have seen for more than six months in ward Ste-Catherine two women who have often given me occasion to speak to you of the acute and subacute forms of spontaneous arthritis of the knee. Both are now getting well with anchylosis. As cases of this kind are not rare, and raise thorny questions of science and practice, I propose to-day to recall the principal details of these two observations and the reflections which they have suggested.

I. *Acute arthritis of the right knee, gonorrhœal, with contracture of the flexors.*—One of them, 25 years old, occupying bed No. 24, ward Sainte-Catherine, was admitted the 29th of December, 1871, into the medical service of my colleague M. Pidoux. She had been taken a few days before with sharp pains in the right knee, accompanied by slight fever and loss of appetite. When she was brought to the hospital, the pains were still very severe; not only was she unable to make any movement, but the knee was flexed to a right angle with the thigh, and she was unable to straighten it, the slightest attempt to do so increasing her pain. At the same time the skin was hot, the pulse at 90, and there was sleeplessness, and very little appetite. In a word,

the intensity of the local inflammatory symptoms and the persistency of this slight febrile condition indicated that the arthritis belonged to the acute form. Furthermore, M. Pidoux had discovered that this arthritis was single, and that the patient had a purulent urethritis and vaginitis which authorized him to consider the disease as of gonorrhœal origin.[1] The 17th of February, that is, more than six weeks afterwards, the general condition had improved, but the local symptoms remained about the same, and M. Pidoux asked me to take the patient into my service. I then found that the knee was flexed at a right angle, in consequence of which the patient was forced to lie upon the corresponding side, that it was swollen and felt very hot to the hand, and that the least pressure, and of course the slightest attempt to move it, caused very severe pains. When I asked the patient to point out the chief seat of these pains, she always indicated the inner side of the knee, the part which corresponds to the passage of the internal saphenous nerve and to the insertions of the internal lateral ligament. On account of the flexed position of the limb, I was not able at first to determine whether there was any effusion of liquid; however, if there was, it was not very abundant, for I did not find any fluctuation.

I further recognized that for the moment the patient had no other joint affected, and that she was without fever. It was then a single arthritis which had at first been acute, and which, in consideration of the disappearance of the febrile phenomena, might be considered as having passed to the subacute state. Was this arthritis to be called rheumatic? Strictly speaking, yes; for by this rather vague word *rheumatism* we wish to designate a general cause, the essence of which is unknown, which affects the synovial, fibrous, and muscular tissues. Furthermore, gonorrhœa also was present, and whatever may be the way in which the production of gonorrhœal arthritis is explained, it is certain that, in its symptoms and consequences, it resembles certain forms of rheumatic arthritis, especially that in which the disease is single, or very marked and prolonged in one articulation, whilst the others are but slightly affected, and in a very temporary manner. Moreover, if any doubts might have existed as to the rheumatic nature of the affection, they would have been destroyed when, a few months later, in June, we saw this patient affected with pains in several other joints, especially those of the shoulders and left elbow. As to the right elbow, it had long been completely anchylosed by fusion in consequence of a traumatic lesion during childhood.

On making my etiological examination I noticed upon the neck the scars of two ganglionary abscesses, and upon the borders of the eyelids a little alopecia and redness coinciding with slight specks left by keratitis during childhood. The patient, although apparently of a good constitution now, had then the scrofulous temperament. The

[1] I agree with those who think that gonorrhœal arthritis is rheumatic. But clinically it deserves mention and a special description for the following reason, which is absolutely inexplicable: it is localized much more frequently than ordinary acute rheumatism in a single articulation, and there goes beyond the congestive form and takes on the plastic and anchylosing form, of which I shall speak in this lecture.

local symptoms do not in any manner authorize me to admit a scrofulous arthritis, but these antecedents might make me fear, in case this acute arthritis should become a chronic one, that it might pass to the fungoid form or white swelling.

However that may be, there was when I first examined this woman one main indication to meet: that of straightening the knee, thus applying the excellent precepts given by Bonnet[1] for the substitution of a good for a bad posture in diseases of the articulations. The patient was then anæsthetized by means of ether, and I straightened the limb very easily by my hands alone, placed it in a wire frame and kept it in place by means of a cushion, an anterior splint, and five straps. Since then the pains have not been so violent. They reappeared however from time to time, especially when the patient moved a little too much, and after we ourselves had made an examination or sought to give motion to the articulation, which we knew to be threatened with anchylosis. At a certain moment during these explorations we discovered lateral mobility, as in the man suffering from a sprain of whom I have previously spoken, and at the same time a loud crepitation which appeared to me to be caused by the friction of the bony surfaces. In a word, after about two months of treatment, the arthritis had passed to the chronic state with the two chief symptoms which I have just mentioned.

To what were these two symptoms due? I again repeat that we do not well understand all the lesions of the beginning of acute and subacute arthritis, because we have not had occasion to study them on the cadaver, and because the only information we possess has been furnished by experiments on animals, and especially by those which Prof. Richet has described in his works on white swelling. Now these experiments probably do not reproduce all the lesions which occur in the living man, and especially those which would explain the symptoms in question. I shall then give you only very probable opinions, warning you that I cannot verify them by direct observation.

As for the abnormal lateral mobility I am disposed to attribute it, like that which I observe after violent sprains, or in patients with white swelling, to a lack of resistance in the lateral ligaments. But I do not disguise from myself that in an articulation where the synovial membrane is thickening and advances, as I shall soon tell you, toward fibrous transformation, it would seem as if the ligaments ought to ollow a similar course, and increase their resistance by a thickening and condensation of their tissue. We should then have to admit that here the ligaments have a tendency, in consequence of the arthritis, to lose in part their fibrous character, while the synovial membrane tends to assume this character. It would be strange; but after all it is not impossible.

And it is because it seems to me strange that I offer you another explanation; perhaps the lateral mobility is caused by the semilunar

[1] Bonnet, Traité des Maladies des Articulations; Lyon, 1845. Thérapeutique des Maladies articulares; Paris, 1853.

fibro-cartilages being softened, thinned, and about to disappear. For I can understand that if these intermediate bodies were lacking, the two bones would approach one another, the ligaments would slacken and lose the tension which, during extension of the limb, was the principal obstacle to lateral mobility. What authorizes me to offer you this supposition is the fact, that, in all articular diseases which have been a little prolonged, the trouble in nutrition which follows causes, by a mechanism which we do not understand, the destruction of the diarthrodial cartilages. Now as the fibro-cartilages have a similar structure, I presume that, as moreover we see it in white swelling, their cells open, are destroyed, and disappear, and that as the cartilaginous portion is disassociated and absorbed, the fibrous portion either disappears itself by absorption, or is no longer thick and firm enough to fill the space between the two principal bones of the articulation.

I have now to explain the second symptom, the crepitation. I can attribute it to nothing else than to that destruction of the diarthrodial cartilages of which I have just spoken, a partial destruction undoubtedly, but nevertheless occupying the whole thickness in certain points, and thus permitting during lateral motion the rubbing of the bones which we felt. I repeat that this singular destruction of the diarthrodial cartilages, first noticed in white swelling, then in dry, deforming arthritis, seems to occur in almost all arthrites when they have a certain intensity or last for a long time.

I omit, for the moment, the later phenomena which we observed and the present condition of the patient, for, in these respects, she resembles another woman, No. 3, whose antecedents I will now recall, and will then complete the account of the two observations.

II. This second patient, No. 3, is 23 years old, and, like the preceding one, has had no children; she knows no cause to which to attribute the very painful affection of the knee from which she was suffering when admitted to the hospital the 3d of April, 1872. The disease then had lasted a week, and was accompanied by a slight febrile movement. The local symptoms were a notable swelling, a small effusion within the joint, considerable heat felt by the hand, very sharp pain on the slightest movement, and much spontaneous pain during the day and especially during the night. In a word, the general and local symptoms were those of moderate acute arthritis. In her case there was neither the permanent flexion nor the lateral motion which we found in the other. We have treated this woman by immobility in a wire splint, several applications of wet cups, several blisters, purges, opiates, and sometimes, when the pains were very severe, by the subcutaneous injections of the hydrochlorate of morphine. You have noticed frequent renewals of pain and even of fever, which made me fear suppuration; but this has not occurred, and after several weeks all these symptoms were so much better that I have no longer had this fear, and have considered the disease as having passed to the chronic state.

III. Since about the 15th of June the two cases have been so similar that I can complete their history at the same time. Finding myself in presence of a disease which had kept the articulation immovable

and threatened to end by anchylosis, I tried to prevent this result and obtain a cure with preservation of the functions. With this object I communicated a few movements to the limb every morning, and advised the patients to do the same. The manœuvre was repeated in the evening by the interne of the service. But see what happened: notwithstanding all their efforts, the patients were not able to bend the knee; their muscles did not contract, and the only movements which took place were in the hip and thigh. As for those which we communicated during about a minute each time, they were very limited and caused pain which lasted for quite a long time thereafter. At the end of a week these pains increased, so that it became necessary to stop all motion and return to poultices. A few days later we recommenced, with the same result. I therefore had to give up my attempts and let the articulations rest.

To-day, the end of July, in both patients the patella is united to the femur; the first (No. 24) has lost all movement of the tibia upon the femur; the second (No. 3) has still a few movements, but they are very limited, and I expect to see them disappear entirely. In both of them we shall have subacute arthritis terminating, after passage to the chronic condition, in complete anchylosis. I have shown you, in addition, that since the beginning of the disease the femur has seemed to be tumefied to a great distance above the articulation. To-day, when I compare it with that of the opposite side, I find a swelling similar to that which we have often seen after simple and compound fractures, and after epiphysary osteitis, a swelling which we call hyperostosis.

Let us now see, gentlemen, 1st, what have been the anatomical lesions in these two women; 2d, why, in spite of all our efforts, the cure did not take place with preservation of shape and functions.

1st. As for the lesions, it is certain that in the synovial membrane they consisted of a hyperæmia and inflammatory exudations, some of them deposited in the membrane itself and making it rigid, others on its inner surface in the form of fibrinous and neo-membranous flakes, which became vascular and were transformed into a fibro-cellular or fibrous tissue. It is for the sake of better characterizing this capital lesion and to distinguish it from those of fungoid and dry arthrites that you have often heard me employ the expression *plastic arthritis*.

While these lesions are being produced in the synovial membrane, what is taking place in the other constituent parts of the articulation? That is what autopsies have not yet well cleared up. I presume, as I have already said, that the diarthrodial and inter-articular cartilages are disorganized and thinned, and perhaps have disappeared. It is more than a presumption in the case of the first patient; for we have not been able to otherwise explain the crepitation which we found in her at a certain time. But is this lesion entirely similar to that which we find in white swelling and dry arthritis, or is it different? Do Brodie's ulcerations and Redfern's velvety change occur here?

Or is it by the histological lesions, the knowledge of which we owe to M. Ranvier, especially by the proliferation of the superficial cells, the segmentation of the fundamental substance, and the opening of the

capsules into the articular cavity, that the supposed destructions commenced? I cannot answer these questions, because the authors I mention have not made a sufficient number of autopsies to be able to say whether the lesions which they describe in dry arthritis are also found in anchylosing plastic arthritis, or at what period of the disease they appear.

So also for the ligaments; are they softened and destroyed, as we might have supposed, in the first patient who has had lateral mobility from the beginning? Are they not, on the contrary, thickened and rigid, in consequence of their participation in the plastic phlegmasia? On this point also I am in doubt.

And the bones? I have noticed nothing in the patella or the tibia, but I told you that the femur seemed notably hyperostosed in both patients. Is it the periosteum alone which has been invaded by propinquity, and which has furnished new layers of bone by a periostitis, likewise plastic? Or has the entire thickness of the bone been affected, passing to the condition of plastic or condensing osteitis? I do not know. But on this subject I offer you a final reflection which has already been mentioned *àpropos* of osteitis. I have often seen hyperostosis follow traumatic and spontaneous arthrites when they had not taken on the fungoid character and the tendency to chronic suppuration, or, which amounts to the same thing, when the patients were not scrofulous. I therefore believe that in cases where we have doubts as to the nature and the tendencies of an arthritis, the certain appearance of hypertrophying osteitis is an argument in favour of the opinion that this arthritis is plastic rather than fungoid and suppurating.

2d. How and why was this complete anchylosis established? Two chief incontestable reasons explain this termination: First, the false membranes formed adhesions like those we find on serous membranes after inflammation; these adhesions, becoming more and more firm and rigid, have diminished little by little the synovial cavity and opposed change in the position of the articular surfaces. Secondly, prolonged immobility has favoured these adhesions. This immobility itself was due both to the pain which permitted neither voluntary nor communicated movements, and to the muscular insufficiency which was itself the consequence of these pains. For there are produced during the course of painful arthrites remarkable physiological and anatomical modifications of the muscles. They cease to contract voluntarily, and assume a prolonged contracture which completes the immobility in the position either of flexion or of extension. You remember that in one of our patients we had to straighten the knee after having overcome the resistance of the contractured flexors by anæsthesia. When the first effect, contracture, has lasted a certain length of time the peri-articular muscles atrophy, then pass to the fibrous and fatty condition which characterizes retraction. The longer these lesions of the muscles last, the more do they favour the establishment of the anchylosis by allowing the formation and organization of the adherences of which I have just spoken.

It is now the moment, gentlemen, to define our opinions upon the

influence of immobility in the production of anchylosis. It does not by itself produce it, only when combined with a plastic arthritis. But it may happen that this latter is caused by the immobility itself. Recall in this connection the distinction which I have often made between large and small articulations, at least so far as traumatic articular diseases are concerned. Immobility alone rarely causes plastic arthritis in the large joints, and if the latter occurs it is due to external violence. The contrary is the case in the small articulations. Immobility alone may there cause plastic arthritis and consecutive anchylosis.

These results do not seem to me to agree with M. Charcot's. In his recent works this author has described an arthritis of the large articulations in paralytics; but the immobility does not seem to me, in such cases, to be the sole cause of the articular phlegmasia. The troubles of the nervous system undoubtedly contribute to a certain extent, and the conditions are not the same as those of immobility after great traumatisms.

But let us return to the mode of production of the anchylosis of the knee in our patients. Is there no reason to lay it to the charge of other causes than those of which I have just spoken? I think there is, but I am not sufficiently acquainted with all the alterations that have occurred, to affirm it. It may be, for example, that the diarthrodial and semilunar cartilages having been completely absorbed, the surfaces of bone thus laid bare and attacked by plastic osteitis have united by a mechanism analogous to that of the formation of a callus. There would then be established an anchylosis by fusion of the bones. It may also be that the cartilages not having been absorbed, fusion has taken place between them, and that thus the anchylosis may be by cartilaginous fusion, a form which is much rarer, but which has been observed, and of which I have seen an example. Or it may be that the cartilages having been preserved, false membranes have formed upon the opposing surfaces, and that solid adhesions have taken place between them. The anchylosis would then be called fibrous.

We are unable to recognize clinically in the living patient with which of these forms indicated by pathological anatomy we have to deal. This would be a source of regret if therapeutics could do anything for complete anchylosis, that in which all movement has disappeared. But I am of those who believe that in such cases nothing should be done. The indication, either to oppose anchylosis itself, or to substitute a good for a bad posture, exists only in those cases in which some movements remain and the anchylosis is incomplete. Now, anchylosis may be incomplete in two ways: first, by the thickening and lack of extensibility of the synovial membrane, consecutive to the cellulo-fibrous, and even fibrous transformation of the plastic materials deposited in it, it is then incomplete anchylosis by rigidity; second, by the establishment of adhesions which are still extensible and susceptible of resolution between the opposite points of the synovial membrane, in which case the anchylosis is called cellular or adhesive. You understand, finally, that the incomplete anchylosis may be at the same time adhesive and by rigidity.

I now reach the second question, a very interesting one clinically, that of knowing why complete anchyloses are produced. It is first because the arthritis has passed beyond the limits of that which is simply congestive, as happens quite often in the traumatic variety following contusions and sprains, and because by passing these limits it becomes plastic and adhesive. But all plastic arthrites do not end in complete anchylosis: a goodly number end in the rigidity and cellular adhesions of which I have just spoken, and which we finally overcome. What takes place in such cases? The congestion disappears, the inflammatory products infiltrated in the thickness of the synovial membrane, and which have given it its rigidity during a certain time, do not go very far in their fibrous transformation, and may be reabsorbed; the false membranes, if there have been any, are also reabsorbed; the synovial membrane again becomes supple, and the articulation returns to its normal condition.

In our two patients the plastic products, instead of being reabsorbed, have advanced further and further in their organization, have made the synovial membrane fibrous, and have formed adhesions, while at the same time, in all probability, the cartilages and the ligaments have undergone the alterations we have mentioned. The capital difference then is this: the plastic arthritis instead of terminating by resolution, has terminated by adhesion, and consequently, by a profound transformation of the normal anatomical conditions.

But in making this explanation I only move the difficulty back. Why, in fact, this unfavourable termination rather than the first? Here I can no longer answer with pathological anatomy, we must turn to pathogeny, that is, to that which is the most obscure and the most difficult, and yet the most real in our science. These women have got their infirmity because they have had a very intense inflammation of a peculiar kind. The intensity has caused the primitive congestion and the consecutive exudations to be more marked than they are in other cases; the nature or the special mode of inflammation has been such that the tendency has been towards organization rather than absorption of the plastic products. Has there been a special cause? It is probable; but we do not know what it is. We say, since we can do no better, that this cause has been rheumatic; we say, for one of the patients at least, that the rheumatism has been gonorrhœal. But since rheumatism produces also arthrites which are simply congestive, or plastic non-anchylosing arthrites, or still others, it remains to know why it has taken the form which we have observed upon our two patients. Probably we must here accuse, as we are so often forced to do, a peculiar aptitude, an idiosyncrasy behind which lies—we should not hide it—our inability to explain the relation between the etiology, the intensity of the lesions, and the tendency of these lesions to advance in one direction rather than in another.

Let us confine ourselves to the deductions which are applicable to the clinic. These deductions are the following: when you have recognized that an arthritis, whether traumatic or spontaneous, is neither suppurating, nor fungoid, nor dropsical, nor dry, do not forget that it is plastic, and that consequently it has a certain tendency towards an-

chylosis, a tendency which you must combat by exciting or favouring resolution, and preventing adhesion.

IV. *Therapeutical indications.*—They result from what precedes, and belong to three periods of the disease. In the first, that of the beginning, the intensity of the phlegmasia must be opposed by rest in a good position, antiphlogistics (leeches, cupping) derivatives upon the intestinal canal, narcotics to quiet pain.

In the second, you must try to provoke and aid the absorption of the plastic products. It is still rest and immobility which meet this indication; revulsives upon the skin, blisters, punctate cauterization may also aid it.

In the third, you must try to make the adhesions, which are still thought to be soft, and the rigidity, which is not yet invincible, yield. It is then that it is proper to try massage and communicated movements. But here we find ourselves between two dangers: that of provoking, by these movements, a return of the phlegmasia, and that, by not employing them, of allowing the anchylosis to form. We are obliged to feel our way. If the manœuvres cause only temporary pain it is proper to persevere; if, on the contrary, they provoke continuous pain, with return of the swelling, of the articular effusion, and of heat to the touch, they must be stopped. Perhaps a little later they may be borne; if they are not it is best to abstain entirely and to abandon the arthritis to the chances of anchylosis, as we have had to do for our two patients. At this same period if the articulation has become indolent enough to allow the patient to leave his bed and walk on crutches, baths and douches of sulphurous or thermal waters, such as those of Néris, Bourbonne, and Plombières, would also be very useful.

LECTURE XXXVI.

CHRONIC ARTHRITES OF THE KNEE.—HYDRARTHROSIS.

Dropsical arthritis or hydrarthrosis—Lesions supposed to exist, but inappreciable by physical signs—Probable congestive form—Enlargement of the patella, explained by a hypertrophying osteitis—Prognosis—Long duration, possible relapse—No tendency to suppuration and to anchylosis—Therapeutical indications: 1st curative treatment: compression, blisters, puncture, actual cautery, injection of iodine; 2d prophylactic treatment.

GENTLEMEN: We have at this moment in the wards several patients suffering from chronic affections of the knee. When passing their beds every morning, I indicate by a word what there is that is characteristic in each one of them, and I recall to you the questions which should always preoccupy us when in presence of affections of this kind: shall we have resolution, anchylosis, suppuration, or the infirmity of

dry arthritis? I cannot speak to you of all these patients: I shall take only the three principal types: hydrarthrosis, white swelling, and dry arthritis. I begin with the hydrarthrosis.

Dropsical arthritis or hydrarthrosis.—The patient in No. 20 is a man thirty years old, jeweller, of a medium constitution, but in whose antecedents we find no indications of scrofula. He told us that on several occasions he had pains in the shoulders, arm, and left knee, which however have never been swollen. These pains, although apparently rheumatic, had not been accompanied by fever nor by such an alteration of health that we could explain them by an acute articular rheumatism.

He entered our wards for the first time three years ago for a swelling in the right knee, the cause of which was unknown. He remained six weeks, and left us almost cured, with a rubber knee-cap, which we advised him to wear during the day and to remove at night. He was able to resume work and continue it until a month ago. However, he has always felt at times, especially when a little fatigued and when the weather was damp, pains in this knee. Finally, a month and a half ago, after a long walk, the pain became more intense, more prolonged, a new swelling appeared, and the patient was obliged to return to us.

He has been here four weeks. You noticed at first that the right knee was swollen, that the depressions on each side of the patella were replaced by a tumefaction appreciable by the eye, that the region was not hot to the hand, and that, by pressing with both hands upon the sides, while the index finger of the right hand pressed back the patella, fluctuation was distinctly felt. There was further the sensation that the liquid was not separated from the skin by a thick layer, and on examining the prolongations of the synovial cavity, especially the upper one, we did not feel any thickening.

The diagnosis was not difficult; first, it was certainly an arthritis, since the swelling, the pain, the difficulty in movement, the heat from time to time could be attributed neither to a simple neuralgia,[1] nor to a cancer, nor to any other disease. But we had next to make the anatomical and the etiological diagnosis of this arthritis.

As to the first, the thing was evident; it was certainly a chronic arthritis, but with considerable effusion, so considerable that it was allowable to make use of it to characterize the disease as our authors have done by employing the word *hydrarthrosis.* You have often heard me pronounce this word for the present patient, and for those who have been similarly affected. But I prefer generally the name *dropsical arthritis* or *arthritis with effusion,* because under this name of hydrarthrosis are comprised two things: an essential effusion or one without lesion, like that which is formed during anasarca, and an

[1] I have often spoken of patients who, with out any anterior disease, or after a traumatic or a rheumatic arthritis, had a very sharp continuous pain in the side of the knee, with exacerbation during walking or without known cause, and without any appreciable swelling. The arthritis had no lesion, or only a congestion inappreciable by our senses; but it was more painful than this simple lesion would have made one suppose. I call it neuralgic arthritis or exaggerated sensibility of the knee.