

# **The etiology and pathology of chronic joint disease / by Newton M. Shaffer.**

## **Contributors**

Shaffer, Newton M. 1846-1928  
Bryant, Thomas, 1828-1914  
Royal College of Surgeons of England

## **Publication/Creation**

New York : G.P. Putnam's Sons, 1878, ©1877.

## **Persistent URL**

<https://wellcomecollection.org/works/upkck3t4>

## **Provider**

Royal College of Surgeons

## **License and attribution**

This material has been provided by This material has been provided by The Royal College of Surgeons of England. The original may be consulted at The Royal College of Surgeons of England. where the originals may be consulted. This work has been identified as being free of known restrictions under copyright law, including all related and neighbouring rights and is being made available under the Creative Commons, Public Domain Mark.

You can copy, modify, distribute and perform the work, even for commercial purposes, without asking permission.



Wellcome Collection  
183 Euston Road  
London NW1 2BE UK  
T +44 (0)20 7611 8722  
E [library@wellcomecollection.org](mailto:library@wellcomecollection.org)  
<https://wellcomecollection.org>

(3.)

A SERIES  
OF  
AMERICAN CLINICAL LECTURES

EDITED BY

E. C. SEGUIN, M. D.

VOLUME III

No. VI

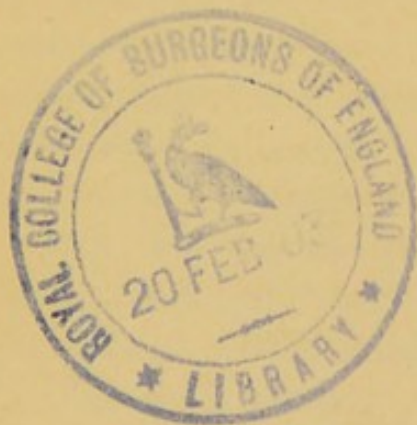
[WHOLE No. 30.]

THE ETIOLOGY AND PATHOLOGY OF  
CHRONIC JOINT DISEASE.

BY

NEWTON M. SHAFFER, M.D.

SURGEON TO THE NEW YORK ORTHOPÆDIC DISPENSARY AND HOSPITAL.  
ORTHOPÆDIC SURGEON TO ST. LUKE'S HOSPITAL.



NEW YORK  
G. P. PUTNAM'S SONS  
182 FIFTH AVENUE

1878

COPYRIGHT BY  
G. P. PUTNAM'S SONS.

1877.



# THE ETIOLOGY AND PATHOLOGY OF CHRONIC JOINT DISEASE.

BY

NEWTON M. SHAFFER, M. D.

Surgeon to the New York Orthopædic Dispensary and Hospital. Orthopædic Surgeon  
to St. Luke's Hospital.

---

ACCOMPANYING chronic diseases of the articulations, there are several interesting and instructive phenomena to which I would like to call your attention, and I wish especially to refer to them, as pertaining to the etiology and pathology of the lesion. In adopting this course, I propose, so far as possible, to give the results of my own observations, which are based upon an experience gained in following many cases in the service of the Orthopædic Dispensary and Hospital, supplementing them, as occasion demands, by illustrations drawn from other sources. The questions involved are very suggestive, and the limits of this lecture will not permit me to do full justice to the subject. I shall therefore strive to be both concise and practical in my remarks.

In order to avoid ambiguity and repetition, I wish to state at the outset, that in using the phrase "chronic joint disease" in my remarks, I refer, unless otherwise stated, to that form of articular disease, of which we may take *morbus coxarius* as a type. The various terms "arthritis," "synovitis," etc., all have



a more limited meaning than the comprehensive appellation I have chosen to designate the lesion under consideration ; for if I interpret the matter rightly, we mean far more in saying that a patient has "joint disease," than by simply stating, for instance, that the individual has an "arthritis." The former carries with it a sense of chronicity which does not pertain to the latter.

From whatever standpoint we may regard the etiology of chronic joint disease, its peculiar clinical history, and its many unfavorable sequelæ will be generally admitted. As usually met with, the symptoms of this form of articular disease are indicative of a progressive lesion, chronic in its nature, not preceded by any acute symptoms, and unaccompanied in its earlier stage by any marked febrile excitement. It is difficult to control, whatever be the treatment employed. It not unfrequently terminates in suppuration,—ultimates in various deformities and atrophy of the limb,—and death ensues in a certain number of cases. The insidious character of the initial symptoms, as followed by the gradual development of the limp, the inconstant character of the pain, the progressive debility of the limb, the impaired mobility of the articulation, the atrophic disturbance of the muscles and their peculiar spasm, are sooner or later succeeded by the more expressive indices of the lesion, which, as it finds expression in deformity and suppuration, gives rise to results which are familiar to all. It is a disease too, in the very large proportion of cases, of early life, occurring during the period of development, when nature's powers of resistance to the invasion of a chronic disease are at their maximum ; but also where in certain conditions of the constitution an exaggerated physiological condition is readily converted into a pathological one : and where, a slight lesion being established, a retrograde takes the place of recuperative action. It is not in short an *acute*



disease in any sense, but one developed in obscurity, and progressing through all its stages attended by symptoms which can only be called *chronic*.

The definition given by Prof. Gross, to chronic inflammation, so well answers that which might be applied to a chronic joint disease, that I quote it here,—“chronic inflammation, however provoked, is generally tardy and sluggish in its movements, creating little constitutional disturbance, but not on this account the less surely and effectually undermining the part and system. In the acute variety, the action is rapid, bold, daring; suffering is severe, and constitutional response loud and unmistakable. In chronic inflammation, on the other hand, the symptoms are, as already stated, often obscure, if not absolutely masked, and the embers of disease never break out into open flame. The disease may continue weeks and months, now stationary, smothered or apparently receding; now advancing and seeming almost ready to assume the acute type.”\* Describing the effects of chronic inflammation, Dr. Gross further says,—“The most common and important of these changes, are suppuration, ulceration, softening, adhesion, contraction, induration and enlargement.”\* Though this is the definition of chronic inflammation, accompanied by a description of its effects, I doubt if the history of chronic joint disease could be better described in so few words.

Subcutaneous local lesions, having traumatism as a cause, and involving non-vital organs (especially the upper and lower extremities) are, as a class, the simplest, so far as treatment and results are concerned, that fall to the lot of the surgeon. They are simply *local lesions*, which run an acute course, and even if neglected and abused, rarely terminate in suppuration. This accident is liable to occur only in those conditions, where

---

\* A System of Surgery, by Prof. S. D. Gross. Vol. I, p. 105.



either from hereditary or acquired causes, there exists an impaired nutrition. These purely local lesions are to be regarded as likely to be followed by serious consequences, as a class, under such circumstances as those which Mr. Paget describes in his "Calamities of Surgery,"\* where a slight lesion may be followed by exaggerated results, and the minor *cause* may easily become the *occasion* for retrograde, rather than a reparative process. Examples of these conditions are met with in daily practice, and the wards of our hospitals afford ample opportunities for demonstrating them. So far as the articulations are concerned, perhaps the typical lesions excited by traumatism, at least the most common, are those produced by sprains. We know how severe they may be, and how chronic they may become. The joint may be stiff and useless; extra, or even intra-capsular adhesions may form as the result of the inflammation excited, by sometimes comparatively slight, and at other times very severe injuries; or the joint may become practically useless, exquisitely sensitive on pressure at certain points, and yet suppuration rarely ensues. None of the characteristic symptoms of chronic joint disease supervene in my own experience, nor has it in the experience of those I consulted. The pathology of this condition has been recently very concisely described by Prof. R. O. Cowling. I quote the following from his paper,—“Languor in the circulation is the prominent feature and the key to most of the conditions. There is mal-nutrition, local in its origin. Plastic deposits resulting in the inflammatory stage, or slowly gathering as the result of the chronic congestion, are not taken up. Tendons, ligaments, and aponeurosis are shortened by continued disuse. The bodies of the muscles waste and then contractility is lost. The nerve structure has not been repaired. New fibres have been de-

---

\* Clinical Lectures and Essays, by Sir J. Paget, page 51.



veloped in adventitious tissue, and they alike cry for good blood and moving blood. With such a condition of affairs, loss of function, pain, and tenderness naturally continue.\*"

In chronic joint disease the condition is far different. There is no plastic material thrown out ; no adventitious tissue is formed. The mal-nutrition is not local in its origin, but general, and in the place of the higher inflammatory products a "regressive metamorphosis" occurs, that ultimates in the formation of a degenerate pus, filled with cheesy flocculi and necrosed cellular tissue. The sprain is the legitimate result of traumatism. The joint disease frequently does not require any blow or fall to excite the local symptoms of the lesion, the morbid elements of which antedate the injury even if it were received. The whole animal economy calls for "good blood and moving blood," not the joint alone. The entire system needs assistance in establishing a normal reaction in the part which is but indicating the general condition. In other words, the joint lesion may frequently be called a symptom ; a symptom of sufficient importance however to require extensive local treatment.

If we take, for example, a case of chronic joint disease at the earliest manifestation of the local symptoms, and treat it *locally*, as we would a fracture or a dislocation, can we assure ourselves that we will arrest the disease? Can we even feel certain that pus will not form? I do not mean to disparage local treatment in joint disease when I say that we cannot. On the other hand, a local lesion in a healthy individual requires no constitutional treatment, and even in the severer injuries of the articulations,—such as fracture near a joint, or a dislocation, where the nutrition of the articulation is materially

---

\*The Pathology and Treatment of Sprains.—*Louisville Medical News*, May, 19th, 1877.



interfered with locally, and its ligamentous and capsular structures lacerated, the symptoms of a chronic suppurative inflammation rarely follow. To summarize,—Experience proves that traumatism excites acute lesions only, as a rule. In those constitutions strong enough to resist and repair the injury, these acute troubles soon subside; under reverse circumstances, they are apt to be followed by a chronic form of inflammation, which may end in suppuration. As applied to joint disease, traumatism is not a prolific cause of chronic inflammation, except in the conditions noted, and then it acts as an *exciting* cause only. We may state still farther. Traumatic joint lesions (excluding incised wounds of the capsule) are not very frequently seen, unless we accept sprain and dislocation as being lesions of this character. When however, these typical traumatic joint lesions occur, they present symptoms that are unmistakable. They no more resemble the ordinary forms of chronic joint disease, in their course and history, than a fracture resembles a chronic osteitis. The comparison may be carried still further. Traumatic joint lesions, as a class, recover (as compared with the chronic) rapidly, while chronic joint disease runs a course of years. Acute joint disease (synovitis,) does not usually, in my experience, degenerate even in strumous individuals into a chronic form of inflammation, while chronic joint disease has an antecedent history, which, like the prodromata of acute hydrocephalus, is vague and indistinct, and it is not until some expressive local symptom presents, that the disease is really recognized. It is chronic from its very outset, and depends on a predisposing condition which has been variously defined and vaguely stated by different authorities. I cannot describe it better, than by stating that the condition is accompanied by a diathesis, that is characterized by an abnormal vulnerability which depends on an acquired or hereditary predisposition to chronic inflammation, the tendency of which is to a regres-



sive metamorphosis, rather than to the formation of plastic elements.\*

In the great majority of children that have presented for examination, both in Dispensary and private practice, with chronic joint disease, the first symptom noted by the parents has not been pain—that which we would expect to find were the cause traumatic—but a limp. This limp would be preceded by a fall in many of the cases, (for how many youngsters are there who have escaped a severe fall,) to which the parents attached a greater or less importance. But in all cases, with a few exceptions, the injury was followed by no acute symptom, nor any manifestation which could be traced to a definite local lesion. In those cases where I have had the opportunity to examine the patient in this stage of the disease, I have found no constitutional disturbance, the thermometer indicating a normal degree of temperature. Pain generally, but not always, can be excited by some of the various movements of the articulation, which are resisted in some of the normal arcs of motion, by a peculiar spasm of the muscle. The patient presents a peculiar attitude, bearing his weight on the sound side, flexion of the joint occurs, and with it other symptoms which depend on the locality affected. This obscure condition is not unfrequently simulated by some slight trouble, or acute peri-articular lesion. On some future occasion, I shall dwell upon this point in detail. I need only say now, that if in such a condition as I have described, a high temperature ( $100.5^{\circ}$  or over) exists, do not make a diagnosis of chronic joint disease. Suppuration in the early stage of any chronic disease is not likely to occur. If you have a high temperature, the chances are that your “joint disease” will disappear in a few days or

---

\* Modified from Birsch-Hirschfeld's definition of Scrofulosis, Vol. XVI of Ziemssen's *Cyclopædia of Medicine*, page 761.



weeks, and without the aid of any extension or other mechanical support. All the prominent symptoms may be present, such as flexed and an adducted thigh (if the hip be suspected) muscular rigidity preventing free movement of the joint, flattening of the natis, obliteration of the gluteo-femoral crease, pain on concussion, pressure, at night, etc.; but a high temperature indicates just that condition which does not exist in chronic joint disease, unless active suppuration be present.  $99^{\circ}$  to  $100^{\circ}$ , (sub lingual) I do not, in children, regard necessarily as an abnormal temperature, though the latter is always suspicious. Still, I have watched and followed several cases where it existed for some weeks, and in one case months, without any sequelæ. It is a safe rule never to make, especially with but one examination, a diagnosis of chronic joint disease, in the early stage, where the temperature is  $101^{\circ}$  or over. A high evening temperature, with a fall to  $99^{\circ}$  or thereabouts in the morning, may indicate (and in joint disease generally does indicate) suppuration. But accompanying this condition are other symptoms which I cannot here stop to point out.

I will relate, as illustrating the diagnostic value of a high temperature under the circumstances just mentioned, the history and result of a case which I have recently seen.

Mary Smith, aged 10, was admitted to St. Luke's Hospital, on October 16, 1877, during the service of Dr. J. L. Little. I happened to be making my usual daily round when Dr. Little examined the patient. He asked my opinion regarding the case, the history of which was as follows: Several months ago the patient had typhoid fever. Eight weeks before her admission, while convalescent, she fell from the bed to the floor, a distance of about two feet, striking upon her right hip. Several days after she complained of pain in the region of the hip, and a few weeks later, the mother noticed a considerable swelling, and great tenderness about the trochanter major. This in-



creased up to the time of admission. Two weeks ago, the patient, who had been limping about, became unable to walk alone. On admission, there was evident a considerable swelling in the region of, and below, the trochanter major, with distinct fluctuation. The limb was flexed on the pelvis at  $30^{\circ}$  and slightly adducted. Upon examining the limb and joint, I found that the movements of the latter were very limited, flexion of the thigh stopping at  $45^{\circ}$ . Adduction and abduction were very slight. Rotation limited, extreme attempts at motion in this direction being accompanied by pelvic movements, and extension could not be executed beyond the  $30^{\circ}$  of flexion. Any sudden motion of limb caused pain. Pain not increased by pressing articular surfaces together. The patient was very greatly emaciated, and the thighs equally atrophied, were about eight inches only in circumference.

A hip joint lesion was so closely simulated by the objective symptoms, that upon a casual inspection, I felt inclined to diagnose a morbus coxarius. A careful examination, and a consideration of the history, caused me think differently, and the temperature being  $102^{\circ}$ , I at once decided that it was an acute extra capsular lesion, an opinion also which Dr. Little held prior to my examination. The subsequent history of this case, the progress of which I noted frequently, I append in full, from notes furnished me by Dr. Alonzo Blauvelt, House Surgeon.

Oct. 17. Patient etherized, and abscess opened freely by Dr. Little, under Lister. About a pint of thick healthy pus evacuated. A digital examination failed to connect the abscess with the joint. Drainage tube inserted and Lister's dressings applied.

Oct. 30. Wound has been dressed every second or third day. Lister's antiseptic precautions and dressings being carefully followed. At this date very slight discharge. Drainage



tube removed. Temperature has ranged from  $99^{\circ}$  to  $101.5^{\circ}$  in A. M., and from  $101^{\circ}$  to  $103^{\circ}$  in P. M. Patient able to move limb in every direction with very little pain. Tonic treatment ordered.

Nov. 15. Wound dressed only about once a week since last record. Entirely healed except a small sinus made by drainage tube. Probe can be passed about  $3\frac{1}{2}$  inches upwards, and a little forwards. Temperature lower. Bals. Peru injected in sinus, and compress over parts. Good motion of joint without pain.

Nov. 30. Sinus entirely healed. Patient allowed to walk around. Temperature nearly normal. Has increased much in flesh. General health improving rapidly.

Dec. 6. Limb can be moved in any direction without causing any pain whatever. Joint motion normal. Patient walks about ward the entire day, with a scarcely perceptible limp. Temperature normal.

I am indebted to my colleague, Dr. Charles McBurney, under whose care the patient now is, for permission to report this interesting case.

To return to the limp. I stated that it might be accompanied by pain. The pain however like the limp is indefinite and vague, and sometimes remits, and not unfrequently changes its location. The parents are apt on these accounts, to call it a "growing pain." But the limp returns. The pain becomes, perhaps suddenly, persistent. It disturbs the patient at night, and is sometimes accompanied by a cry resembling that which occurs in acute hydrocephalus. The child does not fairly awake during these paroxysms. But the cry is peculiar, and the incoherent mutterings are not remembered even if the patient be fully awakened. If the disease be located in the hip, the thigh begins to flex; the joint motions are limited; the muscles begin to waste, and the patient now



walks very badly. Symptomatic fever sooner or later ensues, with a temperature curve that reaches its maximum in the evening, and that touches nearly normal in the morning. Pus is forming, though we may not have external evidence of it for months, when a "cold abscess" will appear. Finally, the patient suffers excruciating pain; the already weakened constitution sympathizes very evidently, the face assumes a care-worn, aged look; and a progressive general atrophy of its various tissues, indicate the extent to which the system is impaired. The abscess discharges copiously an ichorous, shreddy pus. The probe reaches carious bone through the sinuses thus established, and in some cases the patient succumbs, either from exhaustion, or from amyloid degeneration. The very great majority of cases of chronic joint disease, properly treated, do not terminate fatally, but the disease in any event, whether terminating in suppuration and caries, or whether arrested prior to these unfortunate complications, is a very chronic one, requiring a long and persevering treatment, and the result is not, as a rule, a symmetrical limb with good joint motion, but a shortened, atrophied member, useful, no doubt, and strong; but not perfect.

I have tried not to overdraw this sketch, and as illustrated by the average cases of chronic joint disease which have applied to the Dispensary, it is not exaggerated. I wish to ask if it resembles, in any important respect traumatic lesions as applied to the same structures? Does a disease, so essentially progressive, so persistently chronic, so slowly responsive to both local and constitutional treatment, develop from a simple local cause, (so frequently guessed at by the parents, and adopted by the surgeon on this authority,) and which, passing through its primal stages almost unnoticed, or at least, without constitutional disturbance, progresses slowly though surely, until suppuration sends the mercury up to  $101^{\circ}$ — $105^{\circ}$ ?



Joint disease may be either acute or chronic ; when acute, it usually affects the synovial membrane ; when chronic, I believe it most frequently has its origin, especially in the hip joint, in the osseous structure, (epiphysis). The former, sometimes difficult to control, and generally idiopathic, passes through its various stages, and in my own experience, as before remarked, even in strumous children seldom develops into the typical chronic joint disease. Acute inflammation of the synovial membrane is not very frequently seen in the hip-joint. The knee is its favorite seat. When however, it does affect the hip, the lesion can scarcely be mistaken for any other condition. A high temperature, an acutely sensitive joint, a rapid pulse, a "swift atrophy of the muscles," the whole generally preceded by a chill, and with the local symptoms pointing to the affected articulation leave very little room for doubt. A typical case of this somewhat rare affection, and presenting some interesting and peculiar features, occurred in my private practice not long ago. The history is as follows : On Sept. 10th, 1876, I was called to see, in consultation with Dr. A. W. Catlin, of Brooklyn, a little girl, Lucy H., aged 10. Two or three days previously the patient had been to Coney Island, and in returning late in the evening, complained of feeling very chilly. She went to bed immediately upon her arrival home, feeling somewhat ill. During the night she was seized with a violent pain in the hip, and fever rapidly supervened. When examined by Dr. Catlin the following morning, the hip joint (right) was found to be very sensitive, both upon pressure and motion. The thigh was flexed, *abducted* and apparently *longer* than its mate. Temperature  $102.5^{\circ}$ , pulse rapid. Counter irritation and antiphlogistic measures were employed. The malposition remaining, and the lengthening of the limb being not only apparent but *real*, we met in consultation as above stated.

Upon examination, a most peculiar condition of affairs was



made manifest. The rational symptoms all pointed to an acute disease, but the physical strongly suggested a dislocation. The pelvis was tilted downward on the affected side, the trochanter was apparently displaced downward and backward, an actual depression existed in the femoral region, and the limb rotated outward, flexed and abducted, was after careful measurement found to be three-eighths of an inch longer than its fellow. Accompanying this condition, was a marked atrophy of the gluteus maximus and quadriceps extensor cruris. Slight motion produced very severe pain. The temperature was still high, ( $102^{\circ}$ ) and all the indications of fever were present. The acute symptoms were easily accounted for, and pointed to an acute synovitis of the hip-joint. But how with these symptoms were we to account for the actual lengthening? In view of this latter fact, a diagnosis other than acute synovitis was not made.

Two days later we met again, when the pain having modified to a considerable extent, a more thorough examination was permitted. The lengthening was still present, and the position of the thigh was not changed. Palpation and a careful examination of the joint proved that there was no displacement. I then stated my belief that the lengthening was congenital, and could be thus eliminated from our differential diagnosis, and that the trouble was simply an acute synovitis of the hip, with the characteristic muscular atrophy, as mentioned by Sir James Paget.\* The position was, I believe, due to the muscular contraction,† and the "acute atrophy" of the extensors of the leg occasioned the "depression" in the femoral region. As the patient was of decidedly strumous habit, a careful course of treatment was pursued, which ultimated in complete recovery, including a restoration of the atrophied muscles. This length-

---

\*Op. cit., page 209    † See my paper on "Reflex Muscular Contraction and Atrophy in Joint Disease." etc.—*Archives of Clinical Surgery*, June, 1877.



ening of three-eighths of an inch still exists. An examination of the father of the child, subsequently, developed the fact that his *left* leg was one-half inch longer than the right, a condition that he said he had often suspected, from the complaints of his tailor.

A bad hereditary history caused us to fear, during our treatment of this case, that the acute trouble might degenerate into a chronic form of inflammation. But no such complication ensued. The recovery, as stated above, was perfect. I have never seen an acute, suppurative synovitis of the hip. When it occurs at the knee-joint, the symptoms are pronounced. Our standard works on surgery treat fully of this condition. The treatment of the joint lesion not unfrequently terminates in amputation. As generally seen, however, it results from incised wounds of the capsule. I distinctly remember an instructive case of this kind, which I saw in the year 1866. The patient had been discharged from a prominent hospital, because he declined to submit to amputation. I found him in a dark room at the Five Points, surrounded by the most unfavorable sanitary influences. Through sheer pity I promised to attend the boy, (he was twelve years old), though without the faintest idea of doing more than easing his path to the grave. The knee-joint (the site of the disease) was wholly disintegrated. Pus had burrowed underneath the fascia in all directions. I made in all, five free incisions in different parts of the limb, one in the upper part of the thigh, and adapted a simple posterior splint. Stimulants and nourishment were supplied by a neighboring charitable institution. The boy made a good recovery, and had, the last time I saw him, an ankylosed joint in a good position. And this recalls another point to my mind which is worthy of remark. It has sometimes occurred that advanced cases of joint disease, exhausted by hectic, and wasted by suppuration, have been discharged from a hospital "to die at home,"



sometimes at the request of friends. I have seen several such cases afterward with sinuses healed, with health apparent in every expression, and with good, strong, ankylosed joints. I submit these facts simply stating that I have frequently thought, while following advanced cases of profusely suppurating joint disease in hospital practice, that the aggregation of similar cases is contra-indicated. My experience leads me to believe, that, after a lengthened sojourn in a hospital ward, these cases progress much better if removed, provided, of course, that efficient surgical attendance is given, even if the sanitary and hygienic conditions of their homes are not all that could be desired.

I wish now, in contrast with the case of acute synovitis of the hip-joint, (Lucy H.) to relate the history and progress of a case of chronic osteitis, involving the same articulation.

Alice Dormer applied at the Orthopædic Dispensary, on June 24th, 1875. She was then 5 years old, and resided in Brooklyn. Her father died of pneumonia seven months prior to my examination of the child. He had been a healthy man, by occupation a book-keeper. The mother, who came with the patient, was apparently in good health, but had been subject to attacks of acute rheumatism. The patient has four brothers, all in good health, and one sister "in delicate health." One sister died at eighteen months, of acute hydrocephalus. No hereditary disease on either paternal or maternal side. Joint disease and phthisis unknown in the family history. Upon asking the mother if she knew of any cause for the disease, she stated that she did not. No fall or injury, save the one noted below, known. The history developed was as follows: On the 11th of October, 1874, the patient was playing with her brothers as usual. The next day she limped, complained of "cramps" and pain in the limb. She refused to stand on her leg. In a few hours she was walking around again. From this date to Christ-



mas of the same year, she did not limp when she walked. She had no pain during the day, but at night she would sometimes cry out suddenly, and if, on these occasions, the patient was thoroughly awakened, so that the cause of the crying might be ascertained, she would complain of pain in her thigh and knee, especially if the limb was moved. About Christmas she "caught her foot in the bannister," as she came down stairs. The injury was very slight, and the mother attached but little importance to it at the time. The patient cried, however, and complained of her leg and joint. She commenced to limp the next day, and soon after "a pain in the groin" developed. The symptoms now seemed to demand attention, and the mother sought medical advice at a dispensary. A "sprain or growing pain" was diagnosed. Different remedies, domestic and otherwise, were tried until February 25th, 1875, when a Brooklyn surgeon recognized the difficulty, and applied Buck's extension. From that time to the day the patient applied at the Orthopædic Dispensary, she had been kept in bed. The condition of the patient when she applied was as follows: delicate looking, quite anæmic; not well nourished. Dentition regular. Has had none of the diseases incident to childhood, except whooping-cough. The thigh was flexed and adducted, and the joint was very painful on slight motion. Marked atrophy of gluteal muscles on affected side, Obliteration of gluteo-femoral crease. The muscles of the thigh were atrophied also, and the reflex muscular spasm marked. Fluctuation not present. Limbs of equal length.

The patient did not return to the Institution for treatment. An instrument was applied by the family physician in Brooklyn. The mother states that the child grew gradually worse. On October 17th, of the present year, the patient again applied at the Orthopædic Dispensary. Her condition at that time is thus recorded: Very pale and somewhat emaciated. The face shows traces of suffering. The thigh flexed at  $90^{\circ}$ , and adducted at about



30°. Reflex atrophy and contraction very marked—the muscular spasm being so great, that ankylosis is simulated. Temperature 100.5°. Affected limb one inch shorter than its fellow. No pus had formed during the two years that had elapsed since the child was first brought to the Dispensary, or at least none could be detected. Abscess prognosed—child to enter the hospital.

This case illustrates an important point. Obscure symptoms existed before the traumatic element appeared. This, though slight, marked the onset of the more generally recognized symptoms, which might, by many, be deemed those of actual invasion. The disease had been slowly but surely progressing for an indefinite time, and the slight injury received was sufficient, in the peculiar condition of the joint, to produce an effect in no way commensurate with the cause. In my own experience, this is the kind of traumatism which “produces” chronic joint disease.

A brief comparison of the cases of Lucy H., and Alice Dormer will be instructive. In the former there existed a hereditary history that was unfavorable. The lesion was idiopathic, dependant on exposure, partook wholly of an acute character, and terminated favorably. The latter, while pale and anæmic, gave no history in a hereditary sense that was questionable. She was not, however, the victim of traumatism, but nevertheless developed a progressive articular osteitis (epiphysitis) which, in two years, had produced shortening of the femur to the extent of one inch, marked muscular atrophy and spasm, great deformity, and suppuration, if it has not already taken place, is likely to occur at any time. The best result that can be anticipated in this case, is a shortened, atrophied limb, with an ankylosed joint. It is possible, should prolonged suppuration occur, that the history may end with amyloid degeneration, and death.

Through the kindness of my colleague, Dr. James L. Little,



I have recently had the opportunity of observing critically, a very interesting and rare case, occurring in his service at St. Luke's Hospital, the history of which will further illustrate my belief that traumatism is not an important factor in the production of chronic joint disease. I append the history of the case, accompanied by notes made personally at the frequent examinations, to which I subjected the patient.

Edward Gibbs, aged 5, entered St. Luke's Hospital October 4th, 1877. Hereditary history: Maternal grandfather died of phthisis. Paternal side, good, so far as known. Father died from an injury. Mother of patient a thin, anæmic woman, who has been gradually failing in health since the birth of patient. Has lost flesh during the last two years, during which time she has had "a bad cough." The boy in appearance is not strong; has flabby skin, weak eyes; is pale and poorly nourished. Has had measles and whooping-cough, and has recently passed through a prolonged attack of intermittent fever developed in Indianapolis. Has been free from the fever about six months.

On the 23d of September last, the patient fell backward from a wagon in which he was playing, and sustained a severe injury to the hip, which was accompanied by deformity and pain. After a day or two of domestic treatment, (the child was boarding at the house of a stranger,) and several consultations with physicians, none of whom recognized the difficulty, the child on October 4th, as above stated, was taken to St. Luke's Hospital. Dr. Little found the head of the femur dislocated upon the dorsum of the ilium. The dislocation was reduced by manipulation under ether. On the day following I saw the patient and examined him. There was considerable tenderness about the joint, the movements of which were limited by the pain inflicted. The patient walked with a very evident limp, but complained of no pain in doing so. No rigidity or stiffness marked his gait, and no hesitation was manifested in placing



the foot to the floor. His attitude was not unlike that which might arise from a bruise to any part of the limb. Further examination postponed.

Oct. 15. The boy has been moving about the ward at his own pleasure for the past few days, and has daily improved in walking. Examined to-day, especially to ascertain the character of the muscular resistance, and the condition of the joint as regards pain. Patient placed in supine position, and pelvis grasped and steadied with left hand. *Flexion* of thigh slightly resisted; motion nearly normal. With the knee flexed, *rotation inward* gave pain. Motion limited, pain acute, finding expression orally. No reflex muscular spasm, such as is found in chronic joint disease, present. Contraction not persistent. *Abduction* and *adduction* slightly impeded. In prone position, pelvis steadied with left hand. *Extension* resisted quite markedly; not a quick, apprehensive spasm, but a semi-voluntary, elastic, muscular resistance, followed by an exclamation, "Oh!" Flexing the leg at an angle of  $90^{\circ}$ , with the thigh in the above position, and using the leg thus flexed as a lever to produce *rotation inward*, the same result was obtained. Motion limited, the foot passing through the arc of a circle of about  $25^{\circ}$ . *Rotation outward*, markedly limited. Patient sleeps well. Has no pain as he plays about the ward.

Oct. 20. Symptoms have all improved. The same tests as above show improved motion, less pain. Patient *runs* about ward without perceptible limp.

Oct. 21. Case seen by Dr. Buckminster Brown, who visited the hospital with me, and attention was called to the history and condition of patient. The slight remaining resistance to joint motion was developed, and noted by Dr. Brown.

Oct. 27. Gluteo-femoral crease, which had been slightly lower on affected side, normal. No flattening of natis. Severe concussion to sole of foot produces no pain or flinching. No atro-



phy ; faradic reaction of the thigh and gluteal muscles normal.

Nov. 24. Very slight, hardly perceptible muscular resistance. Patient runs, jumps, and plays without the slightest limp or complaint.

Dec. 11. Joint motion normal. Patient discharged, cured.

The lessons taught us by this case are both interesting and important. 1. Without a history of dislocation, the condition made evident by examination would have been well calculated to mislead many in diagnosis. In other words, a condition approximating that induced by the injury received, though excited by a less marked traumatic cause, might have been called "incipient hip-disease," and treated as such by, perhaps, extension apparatus, and in a few weeks the patient would have been "cured," when, as the result abundantly proves no such treatment was required. 2. None of the symptoms especially characteristic of the first stage of a chronic joint lesion were present. 3. At no time subsequent to the reduction of the dislocation, was there any disinclination to walk, or hesitation in using the limb. 4. The peculiar and expressive reflex *spasm* of the muscles was absent, though, as in the "hysterical joint," there was a semi-voluntary muscular resistance, which simulated it. 5. The "swift atrophy of the muscles" mentioned by Paget, as following acute synovitis, was not present, nor was the faradic contractility of the muscles reduced. 6. An extensive, (and a neglected,) traumatic lesion of the hip-joint, in a poorly nourished child, with a very questionable hereditary history, steadily improved from day to day, without any especial treatment, and recovery was rapid and complete. To conclude, we may say that the head of the femur had been dislocated for ten days, and that the capsule of the joint was undoubtedly lacerated and the ligamentum teres ruptured. The patient was only 5 years old, and as likely to develop chronic joint disease as any child that might receive some slight, and al-



most unnoticed, injury, which months afterward is followed by the chronic joint limp that so frequently precedes suppuration, &c. It was, however, a simple, acute, local lesion and repair took place rapidly. But all the conditions that are ordinarily deemed necessary to excite "joint disease" were present, if traumatism alone could develop it in this particular case. The child was even given the opportunity to irritate the joint by all the exercise he wished to take, which was very considerable. On the other hand, a simple fall, unattended by any especial feature and antedating, in many instances, the initial expression of the local symptoms several months, is frequently made responsible for so grave and so serious a lesion, as chronic joint disease. Unfortunately, especially for our patients, traumatism alone is not responsible for chronic joint disease. If it were, rapid and complete recovery would be the rule,—abscess and progressive deformity the exception.

In treating more directly of the pathology of joint disease, I wish rather to view it from a practical and clinical standpoint, as I have its etiology, and as furnishing indications for treatment, than with the object of contributing an essay upon its minute morbid anatomy. Indeed I have nothing to add to the researches of the able investigators, whose contributions to the pathology of joint disease leave but little to be said. Brodie, Barwell, Paget, Billroth, Volkmann, and many others, may be consulted by those who wish especially to study this subject. It is enough for us to know that, for obvious reasons, but few opportunities have been afforded pathologists to record the *initial* changes which occur in the chronic forms of articular disease. The cases on record demonstrate, however, that the primary lesion may occur in almost any of the articular structures, and the facts thus adduced do not, as yet, afford sufficient evidence upon which to base reliable data. Hence it is that we find such a variety of opinions expressed by different



surgical authorities regarding these changes. From a clinical standpoint, however, it matters but little where the initial lesion takes place ; for in those conditions where chronic joint disease is easily excited, the morbid process sooner or later attacks the neighboring structures, and if it proceed unchecked, the entire joint becomes involved.

An opportunity has been afforded me, through the kindness of Dr. Robert Abbe, to examine the pathological condition of a hip joint, one of the structures of which had become the seat of inflammation in a very peculiar manner.

J. Bruce, aged 30, was, on March 2d, 1875, admitted to St. Luke's Hospital, during the service of Dr. James L. Little. The patient was suffering from an aneurism of the femoral artery, for the relief of which Dr. Little ligated the external iliac, on March 9th, 1875. On the 22d day the patient died from exhaustion. While performing the autopsy, and in removing the aneurismal tumor, Dr. Abbe accidentally incised the capsule of the hip joint. A few drops of pus followed the incision. After excising the aneurismal sac, a further exploration of the joint revealed the fact that the ligamentum teres had been the seat of acute inflammation. Its surface, highly injected, was of a brilliant scarlet color. About two drachms of laudable pus was found in the capsule. There was no evidence of disease of the cartilage or bone, and the other joint structures were sound. The patient had complained before the operation of pain in the knee, and the thigh was flexed and adducted to a considerable extent. The presence of the aneurism and the pressure it occasioned upon the subjacent parts, were sufficient to account for both the pain and position of the thigh, and no one had suspected the existence of any hip joint lesion. It would not have been discovered, but for the accident referred to, which disclosed the product of inflammation. Regarding the cause of the lesion thus found, it would indeed be difficult



to offer a perfectly satisfactory explanation. The tumor did not in any way press upon the joint : if it had we should have looked for a lesion in a different structure. There was no history of direct violence. The limb had been held prior to the existence of the pain in the knee, &c., for many weeks in a *flexed* and *adducted* position. This placed the ligamentum teres upon the stretch, and in this way an irritation was produced that might easily be sufficient to produce the inflammation. This is my explanation of it, and it involves a traumatic cause. The evidences of acute inflammation, and laudable pus—each one (where suppuration ensues) the ordinary result of traumatism—were found at the autopsy.

I do not mention this case as forming the basis of my opinion regarding the early changes which occur in joint disease. On the contrary, my clinical observations, records and studies lead me to different conclusions. I offer this interesting case as a contribution to the, as yet, very incomplete literature of the subject.

While no opportunity, other than the case just mentioned, has been afforded me to examine, the post mortem condition of a diseased joint in the *earliest* stage of the lesion, I have, by watching the development of the symptoms, and noting their sequelæ, in well marked and unmistakable cases, and in obtaining reliable histories from intelligent persons, reached conclusions that are based upon, strictly speaking, clinical experience. It is from this standpoint that I submit my views, selecting such cases as may illustrate the points involved, and stating in general terms the results of my observations, which include the records of several hundred cases that have been under my personal care, both in dispensary and private practice. My personal experience with statistics forbids my presenting them. In joint disease especially, they are of value principally to the compiler, so important are the many modifications



of the facts elicited and which cannot be expressed in a few words. Statistics are valuable in reporting results, and in classifying absolutely certain facts, such as sex, age, &c., &c. But beyond this, with no universally accepted definition of the many headings necessary to complete a history, statistical tables on joint disease are, at least, unsatisfactory. I have the records of many cases. Instead of presenting tables, I prefer to state my conclusions.

Of the various structures entering into the formation of those joints which are most frequently the seat of disease, the bone and synovial membrane are the most liable to inflammation. The ligaments, from their intimate association with the synovial membrane, are so closely identified with the latter, that it is practically impossible to diagnose an idiopathic lesion of the former. The cartilage, being non-vascular, is rarely, if ever the seat of an idiopathic inflammation, but when, through traumatic cause it becomes injured, serious trouble is apt to ensue. We have then, practically, these two important structures—the bone and synovial membrane, to which we must look for our primary lesion; or, perhaps we will state the case more clearly if we say that in whatever part of the joint the primary change may take place, it is to one of these essential joint factors to which we must look for our symptoms. The early symptoms of either chronic osteitis or synovitis are, generally, clearly defined, and though, as the morbid process advances the distinctive symptoms may become obscure and conflicting, there are, as I shall hope to demonstrate, certain specific indications which are of value as a means of making a differential diagnosis, and upon which, of course, will depend both our prognosis and treatment.

In chronic synovitis, “we first find swelling and redness of the synovial membrane; it has already undergone some change in the lateral portions of the joint, in the folds and neighboring



sacs ; its tufts are puffed up, very little elongated, but very soft and succulent ; the whole membrane is more readily distinguished from the firm tissues of the capsule, and may be detached with greater facility than normally. At this time the synovia is rarely increased, but is cloudy or even resembles muco-pus. These changes in the synovial membrane gradually increase ; it becomes thicker, more œdematous, softer, redder ; the tufts grow to thick pads, and in places resemble spongy granulations. The surface of the cartilage loses its blue lustre, though it is not yet visibly diseased ; but the synovial outgrowths begin to grow over the cartilages from the sides. and to push in between the two adjacent surfaces of cartilage ; meantime the capsule of the joint is also thickened, and has acquired an evenly, fatty appearance, and is very œdematous ; this swelling and œdema gradually extend to the subcutaneous tissue, and to the skin. From this point, the changes in the cartilage claim most of our attention. The synovial proliferations, in the shape of red granular masses advance gradually over the entire surface of the cartilage, and cover it completely, lying over it like a veil ; if we attempt to remove this veil, we find that in some places it is attached quite firmly by processes entering the cartilage, just as the roots of an ivy vine cling to and insert themselves into the wall against which it grows ; these roots not only elongate, they spread out, and gradually eat up the cartilage, which, when the covering of fungous proliferations is removed, appear first rough here and there, then perforated, and finally disappear altogether ; then the fungous proliferation extends into the bone, and commences to consume this ; the result is fungous caries." (Billroth's *Surg. Pathology*, page 473.)

I have quoted thus at length from an eminent authority upon the progressive changes which occur in chronic synovitis, for the purpose of calling attention to some of the important symptoms following and accompanying this condition, as it



passes through its various stages. This description does not include *hydrops articuli*, which, as the same authority states "is no more apt to become purulent synovitis than is chronic articular rheumatism."

In the early history of this pathological condition, there may be some pain of an indefinite and vague character. Sometimes there is no pain at all. It does not at least exist as an important symptom, and it is most likely to be developed after prolonged exercise. In my own experience it has never been urgent, sleep being undisturbed by "starting pains," and the incoherent, nocturnal cry. A slight swelling may be noted, principally exhibiting itself, for instance, in an obliteration of the fossa on either side of the patella, if the knee be the location of the disease. The patient is apt to limp slightly, especially after exercise, and the limp thus developed is usually the first symptom noted. No pain being present to locate the trouble in the mind of the parents, and the patient sleeping well, the limp is usually supposed to be a "bad habit." The limp increases, the swelling becomes more apparent, the joint begins to assume a semi-flexed position; pressure over the articulation develops sensitive points. The patient now locates a definite pain, and it is usually at this stage, especially in dispensary practice, that professional advice is sought. The changes in the synovial membrane and the modified secretion resulting therefrom, do not produce an effect which limits the motion of the joint, except in the extremes of movement. The resistance felt in gently urging the distal member of the joint into a position of extreme flexion or extension is not unlike that which would result from a simple distension of the capsule, or the altered and thickened state of the synovial membrane. There is no sudden, apprehensive arrest of motion, caused by a quick, spasmodic contraction of the flexors on the one hand, and the extensors on the other; in other words there is no reflex



muscular spasm which arrests movement at a given point, with a quick and decisive check ; or, if this muscular spasm exists at all, it is only to a very limited extent. As the disease advances however and the fungous proliferations fairly invade the bone, this muscular spasm occurs to a marked degree, and with it other symptoms appear which will be described later on.

In primary, chronic articular osteitis, on the other hand, which is only too likely to end in caries, the pathological conditions are of a far different character. The lesion involves, to a greater or lesser extent, the epiphysis of one, or very rarely two, of the important bones which form the articulation. It is however, like the condition described above, viz,—*chronic*, and it may have existed, in a sort of latent state for many months, wholly unsuspected, and at a point not immediately contiguous to the articular cartilage. It would certainly seem that this might be true, for the symptoms, once fairly developed, progress as a rule slowly, but with great persistency, and they are very difficult to control. Remission in the symptoms also, is more apt to occur in chronic osteitis than in chronic synovitis. I saw to-day a case—John Reading, aet.  $3\frac{1}{2}$ , where vague and uncertain symptoms existed all last Summer, He was a little lame at that time, was very restless at night, complained of pain in his hip and knee, and was decidedly disinclined to take exercise. These symptoms all disappeared about September 1st. They again appeared in a couple of weeks, this time accompanied by an inguinal adenitis. Again the child recovered, and ran about without any limp. To-day, (November 22nd), this patient, when examined, though limping only very slightly, was found to have a decided reflex spasm of all the important muscles acting on the hip-joint, very limited joint motion, shortening of the limb (a full quarter of an inch,) marked atrophy of the muscles of the thigh, which



was nearly an inch smaller in circumference than the sound side, and a reduced faradic contractility of the quadriceps extensor and gluteus maximus. The circumference at the calf was the same on either side. But actual pathological observations are wanting on this point, and it is only by calling physiology to our aid that we can reach satisfactory conclusions. In the epiphysis of the long bones, the nutritive processes occur upon which depends the growth of the diaphysis.

These epiphyses are very vascular as compared with the dense structure of the shaft, and they are, of all the joint structures, the most abundantly supplied with nervous tissue. Kölliker remarks\*: "On the articular ends of many bones, such as those of the elbow, knee and knuckle joints, I have noticed the nerves to be more abundant than elsewhere." Again, (page 338), "In the knee I have seen nerves, even in the true synovial membrane, although in general they are rare, and are most distinct in the large vascular processes." In children these epiphyses are in a condition that approximates hyperæmia; and in that peculiar state which predisposes to chronic joint disease it would seem that their true physiological function was sluggishly performed. I cannot do better in this connection than to quote from Mr. Barwell's excellent treatise on "Diseases of the Joints:" "The truth is, that the nutritive activity brought about by the ossifying action in the epiphysis, is very apt to produce a congested condition of the part; and the large bone ends which are found in strumous children are the accompaniment of such tendency to congestion. As the child gets older and stronger, this hyperæmia disappears, and in the great majority of instances is followed by no evil results whatever. It is, however, certain that in a given number of cases the congestion predisposes to inflammation, and the merely passive is followed by an active condition. Thus inflam-

---

\* Manual of Human Histology, Syd. Ed. Vol. I, p. 335.



mation may be set up in an epiphyseal end which was previously in an abnormal state, and such in the greater number of cases is the mode in which the disease now under consideration (strumous articular osteitis) commences. All such attacks are in the beginning very slow, and hang for months, perhaps even years, between a state merely of sluggish functional performance and of active disease." My clinical experience fully sustains this last sentence, and indeed, the entire quotation. When this "sluggish functional performance," the initial stage of chronic articular osteitis, terminates in "active disease," the first symptom noticed by the parents will not be pain but, as in chronic synovitis, a limp. In this one respect the two conditions present the same symptom, though the limp of chronic synovitis resembles that of simple joint fatigue, while that which occurs in articular osteitis is more strictly a debility of the entire limb. Indeed, all the symptoms in chronic articular osteitis, from the very onset, show the marked reflex impression which the disease makes upon the nervous system, while those of chronic synovitis are more strictly *local*. These facts, which, so far as I know, have not been noted before, are not difficult to explain when the neural distribution to the two structures, and the conditions described by Charcot, in his work on "Diseases of the Nervous System" as being necessary to produce trophic changes and muscular spasm, are considered.

Sometimes quite early in the history of articular osteitis there occurs this peculiar, incoherent cry at night to which I have referred already as resembling, in some respects, that which occurs in acute hydrocephalus. I have heard it frequently in chronic joint disease when it was very agonizing. The fact, however, that it rarely awakens the patient, and that if he be awakened he knows or remembers nothing of it, leads me to believe that the irritation of the peripheral epiphyseal nerves is both peculiar and profound—for this reflex expression of the lesion



occurs during the suspension of the will. Then there is the characteristic reflex spasm of the muscles, a spasm which resembles in many important respects that which follows the remote symptoms of injuries of nerves, as described by Dr. S. Weir Mitchell in his valuable treatise.\* Accompanying this reflex spasm is a muscular atrophy not dependant upon *disuse*, which is pronounced and which can be demonstrated by the faradic current. And many of the skin troubles which follow during the local treatment of this lesion, and which have been referred, and which are partly due, to the irritating properties of the adhesive plaster, etc., I have sometimes thought were the analogues of the glossy skin and eczema found after neural lesions. I have not time here to amplify this interesting study. I have already stated my views in brief upon this subject.† I wish, however, to take this occasion to say that I am indebted to my friend, Dr. E. C. Seguin, for his kind assistance and advice in my investigations.

To return to the symptoms of articular osteitis: The debility of the limb increases gradually, and diurnal pain is, as a rule, first developed by any sudden or unexpected twisting of the limb. Even a very slight movement, for which the patient is unprepared, will cause him to cry out. The "starting pains" accompanied by the osteitic cry become in many instances urgent. The muscles acting upon the articulation, when the joint is examined, show a decided tendency to resist, and a marked spasm (it is more than a "contraction") of the muscles arrests the motion at a given point. In the first stage the resistance may be only felt in the extremes of motion; unlike the sensation imparted when the resistance lies in a thickened or distended capsule, it has a peculiar, apprehensive quality. It is sharply defined, and no effort on the part of the surgeon or patient can

---

\* Injuries of Nerves, by Dr. S. Weir Mitchell. pp. 147-153.

† Op. cit.



overcome it without the use of an anæsthetic. These conditions are best noted in the knee-joint, where an effusion, or a thickened capsule is readily observed, and the existence of muscular spasm easily detected. In illustrating these points I will select two cases of knee-joint disease, both of which are under my observation now in private practice.—

Addie D——, aged five and one-half years ; hereditary history shows phthisis on maternal side ; child strumous in appearance, waxy skin, etc. Has had very little illness, except that incident to childhood. Was seen in consultation with Dr. S. W. Dana on July 9th, 1877. At that time the following history was obtained : Last December (1876) the patient complained slightly of her knees, but the trouble was not severe enough to interfere with the patient's health or movements. No swelling was noticed by the parents, nor was the case considered urgent enough to demand the attention of the family physician. A few days before Dr. Dana brought the child to my notice, he had been called in by the father on account of a swelling which had been observed in *each knee-joint*. An examination proved the existence of a very considerable effusion in either joint, accompanied by an evident local rise of temperature, but with no constitutional disturbance. No pain was produced by movement of the joints, by pressure, or concussion. The child could run and play without inconvenience, limping, or pain. It is not unlikely that limping would have been observed had only *one* joint been affected. Passive movements of the affected joints developed no reflex muscular spasm whatever ; an *elastic resistance* limited joint motion in the extremes of flexion and extension. No pain or discomfort at night ; no "starting pains ;" patient sleeps soundly ; no perceptible muscular atrophy. *Diagnosis*.—Chronic synovitis affecting each joint. The bone was excluded from any participation in the disease. As to whether the cartilages were affected is uncertain ; probably



they were slightly on their articular aspects. Looking forward for an indefinite time, and supposing that the disease proceeds unchecked, both cartilage and bone will become involved, and when the latter has been fairly invaded by the disease, there will occur the sharp nocturnal cry, the reflex muscular spasm, and the other symptoms which I have described as pertaining to articular osteitis.

Willie L——, aged four years, applied at my office on September 11th, 1877, with a letter of introduction from Dr. E. D. Hudson, Jr. The patient had been suffering from a chronic disease of the knee-joint for two years. The leg was flexed at an angle of  $90^{\circ}$  with the thigh, and the head of the tibia had become partially subluxated backward. An examination of the joint revealed a smooth and glossy skin drawn tightly over the joint. No fluctuation was anywhere evident; no apparent thickening of the capsule. The ham-string muscles were very tense, and upon careful examination no motion whatever of the articulation could be detected. The joint was practically ankylosed by the muscular spasm; the tendon of the quadriceps femoris being equally tense with the flexors of the leg, and resisting any attempt at motion. That the joint rigidity was due to muscular action alone, was demonstrated by the administration of an anæsthetic, free motion being then easily developed. The muscular atrophy was also great, and the reflex spasm marked, and excited upon the slightest attempts at motion. Nocturnal pain had been a prominent symptom, accompanied by sudden cries and incoherent mutterings at night. *Diagnosis.*—Osteitis, affecting probably the articular aspect of the epiphysis of the tibia, and *prognosis* unfavorable, including a possible suppuration, a partial arrest of development of the tibia, and ankylosis.

To bring out plainly the symptoms characteristic of these two conditions, I will analyze the important symptoms of



each and place them side by side in a differential table:

CHRONIC SYNOVITIS.	CHRONIC ARTICULAR OSTEITIS.
1. Capsule thickened,—Effusion marked.	1. No thickening of capsule evident,—no sense of fluctuation.
2. Natural contour of leg and thigh,—Joint outline obliterated.	2. Muscular atrophy marked,—Joint outline clear and distinct,—Joint appeared large, on account of the diminished size of both thigh and leg.
3. Motion extensive and nearly normal.	3. Motion <i>nil</i> .
4. Resistance to motion <i>elastic</i> , and efforts to overcome it not productive of pain.	4. Joint held perfectly rigid by muscular action alone.
5. No reflex muscular spasm present.	5. Reflex muscular spasm affecting both flexors and extensors.
6. No pain present, nor produced by forcible tests.	6. Acute pain upon the slightest attempts at joint motion.
7. No perceptible limp or hesitation in walking.	7. Unable to walk from pain and deformity.
8. Sleep normal,—No reflex osteitic cry.	8. Incoherent cries and “starting pains” occurring during sleep.
9. Femur and tibia in normal relation to each other.	9. Tibia subluxated backward (partial) by muscular action.
10. Symptoms local, so far as those <i>dependent</i> on the joint lesion are concerned.	10. General and local neural symptoms directly referable to the joint lesion.

A further comparison will be unnecessary. Sufficient is here stated to plainly indicate the essential points involved. *Chronic synovitis*—and especially the fungoid variety—is not, in my experience, attended by reflex muscular spasm to any considerable extent. On the other hand, in *chronic articular osteitis* this spasm is an early and progressive symptom, which gradually increases until ankylosis is simulated. My experience also leads me to the conclusion that the initial lesion occurs more frequently in the epiphysis than in the synovial membrane. Either this is especially true of the hip-joint, or the chronic synovitis remains undiscovered until the morbid process, attacking the bone, gives



rise to the characteristic symptom of articular osteitis, viz., the reflex muscular spasm. Such a condition of the hip-joint, for instance, as that which affected the knees in the case of Addie D—— would certainly have remained undiscovered many months, for it was not until the effusion was observed that any trouble was recognized by the parents. It is much more probable that the synovial membrane is more frequently affected in the knee-joint (this is certainly true of acute synovitis) and that the epiphysis is oftener attacked at the hip. I do not remember to have observed more than two or three cases where I was able to diagnose a fungous synovitis of the hip-joint. In one of these cases the mother of the patient (James Morrow) did not apply to the dispensary (Dec. 20th, 1876) with any thought that a joint disease existed. There was merely a slight and almost imperceptible swelling in the region of the capsule of the hip-joint to which my attention was invited. The history of the boy developed, however, uncertain and vague symptoms of several months' standing, which pointed to the hip-joint—which symptoms had all disappeared upon the appearance of the swelling above referred to. Examination of the articulation developed marked reflex muscular spasm in the extremes of joint motion. The patient could run without a limp, He jumped from a chair without favoring the suspected limb. He could even hop on the affected side with perfect ease. But for the existence of the characteristic reflex spasm and the limited motion (there was no pain, nocturnal or diurnal) I should have thought there was some extra capsular trouble simulating joint inflammation. But this spasm was marked. It was chiefly noted in rotation, both inward and outward, and it possessed all the characteristics of the peculiar resistance which accompanies articular osteitis. After some study, I made a diagnosis of chronic fungoid inflammation of the synovial membrane which had existed for some months, and which was then invading the



bone. With this opinion, an extension instrument was applied. The stiffening of the joint became rapidly progressive. This result did not surprise me; on the contrary it served to confirm my diagnosis, for I have frequently observed that in fungous synovitis of the knee-joint, where tolerably good motion existed, that an immobilizing apparatus rapidly stiffens the articulation. I believe this rigidity results from the fixation, and in this way:—When the joint is allowed to move freely, the spongy granulations are, by friction and pressure, kept in abeyance; but when the joint remains fixed in any one position for a length of time, they become exuberant and sensitive, and motion is thus interfered with.

After a time (to return to the Morrow case) the rigidity became so marked that the apparatus was wholly dispensed with, in order to test the effect of a free use of the articulation. For a time slightly better motion was observed. Pain then became an urgent symptom. But in order to test the matter fully the re-application of the splint was deferred. The joint daily became weaker; the muscular spasm grew more persistent; nocturnal starting pains appeared, and the patient, on November 1st, 1877, was unable to walk a single step alone. The limbs at that time were of equal length, the atrophy marked, and a very slight amount of joint motion only was perceptible. Muscular spasm was very marked, and the faradic reaction of the thigh muscles, which from the first had been modified, showed a still greater reduction. Pain could be developed by very limited force, and all the symptoms of "morbus coxarius" were present. A fact, to which I wish to call attention, was the equal length of the limbs at my last examination of the case (November 10th). The history of the disease covers a period of eighteen months. Had the lesion been one of primary chronic epiphysitis, some shortening would have taken place. The splint was applied for the second time about the 5th of Novem-



ber, since which time the subjective symptoms have been modified, pain being less, and sleep better.

I have said that in the first stage of chronic inflammation involving the joints, but little difficulty occurred in making a differential diagnosis between synovitis and osteitis. My reasons for making this statement are evident in the differential table which I have given, and from my remarks. But many cases present where the articulation is not examined until the disease has existed for a long time. The symptoms here may be conflicting. Evidences of synovial disease may be present, and a decided reflex spasm also exist. My rule under these circumstances is as follows: If the various joint movements, be they limited or nearly normal, are unaccompanied by this peculiar reflex muscular spasm and atrophy, I believe the bone to be free from disease. If, on the contrary, motion be equally extensive, even approximating the extremes, and this spasm occur, the bone is involved. *Marked reflex spasm in chronic joint disease always indicates osteitis.* Its absence, I believe, eliminates the bone, as a rule, from any participation in the lesion. There is a class of cases which form an apparent exception to this rule. I refer to those cases of articular osteitis (either primary or secondary) where early and profuse suppuration occurs. In this condition the reflex muscular atrophy and spasm do not seem proportionate to the extent and character of the lesion. These cases have not occurred very frequently in my experience. They seem to accompany the markedly scrofulous diathesis, and are frequently followed by lardaceous degeneration. I shall speak more concisely of this condition on a future occasion. The opportunities I have had of examining diseased joints after exsection or amputation, confirm my position regarding the relation of the muscular spasm to the lesion. I will mention two or three of the most striking cases, in illustration:



Mr. S—— entered St. Luke's Hospital on Sept. 6th, 1876, with chronic disease of the left knee-joint of several years standing. The limb had become, in consequence of the diseased articulation, a source of much annoyance and trouble to the patient, and various efforts had been made, but without success, to afford relief by conservative measures. A very considerable degree of joint motion existed (to exceed  $90^{\circ}$  of flexion and almost perfect extension). A very perceptible and distinctly audible cartilaginous crepitation existed on passive movement. There was no marked nocturnal pain however, and no decided reflex muscular spasm. It existed, however, slightly. In October, 1876, Dr. Little amputated at the lower third of the thigh. Examination of the joint revealed the characteristic appearance of a fungous degeneration of the synovial membrane in an advanced stage, and the eroded and roughened cartilage had a worm-eaten aspect. The capsule was filled with a pulpy mass of unhealthy granulations, which had invaded the ligaments. The bone was not carefully examined. I should infer that it was slightly diseased, and probably in the head of the tibia. This case is cited to demonstrate that an extensive degeneration of both the synovial membrane and cartilage are compatible with a considerable degree of free and unrestricted motion of the articulation.

Maggie Quinn entered St. Luke's Hospital on Dec. 17, 1875. The patient had sustained an injury of a very uncertain character some six weeks before her admission, which the mother stated had been followed by obscure symptoms about the joint. When examined the motions of the limb were nearly normal, but pain and marked reflex muscular spasm were developed in the extremes of movement, especially when, with the patient in the prone position, and the leg flexed upon the extended thigh, rotation outward or inward was attempted. The child walked with a decided limp, and already a considerable degree of muscular



atrophy had occurred. The muscular spasm increased rapidly, and in three months the joint was seemingly ankylosed. The incoherent nocturnal cry occurred soon after her admission. An extension splint was applied about the middle of January following. An incontinence of urine developed, however, which materially interfered with the perineal straps, and excoriations followed which refused to yield to treatment. In June, 1876, my colleague, Dr. T. T. Sabine, excised the hip-joint. The bone and cartilage were found diseased, and the synovial membrane was, so far as could be ascertained, only slightly affected. I do not remember to have seen a more rapidly progressive or a more persistent reflex muscular spasm than was developed in this case. The pathological specimen obtained sustained my expressed opinion that the primary lesion existed in the bone.

A peculiar circumstance was developed in the after history of this case. Neither the nocturnal osteitic cry nor the reflex spasm of the muscles ceased after the operation. They both existed irregularly, subsequent to the exsection. I forgot to state that the acetabulum was found to be diseased.

This case, operated on so early in its history (a little more than six months after the first appearance of the symptoms), was deemed by me a favorable one for exsection. I regret to add that the sinuses are still unhealed, and that albumen has lately been discovered in the urine.

John Buchanan, aged 3 years, applied at the Orthopædic Dispensary on May 22d, 1877, with extensive disease of the right knee-joint. About one year before, his parents had noticed that the patient limped a little—"dragging the limb." No pain was noticed during the day, but at night the patient would "scream out in his sleep." No swelling of the joint was noticed until after the limp and the nocturnal pain. When the limp appeared the joint commenced to stiffen and to gradually flex. The slightest movement at this time produced very severe pain.



It was with difficulty that the mother could put on the patient's shoe and stocking. The muscles began to waste, and the "stiffness" of the joint became a marked symptom—progressing until no joint motion was apparent. In December following there was a marked increase of the pain. High fever supervened, and the patient began to lose flesh rapidly. Swelling of the joint now occurred. When he applied at the Dispensary, the joint was much enlarged, the tissues surrounding it being thickened and infiltrated. Two sinuses led down to the joint, one above the patella, the other below and to the outer side. An abscess existed on the anterior and outer aspect of the thigh, at about the junction of the middle with the lower third. The muscles of the leg and thigh were greatly atrophied. Any attempt at joint motion produced excruciating pain, and a marked reflex spasm of the muscles existed. The patient was very much reduced in flesh. On May 28th, after consultation with Drs. Markoe, Peters, and Sabine, I amputated just above the abscess, using the abscess wall for the outer flap. The patient recovered rapidly. Examination of the joint revealed the fact that the lesion primarily existed in the head of the tibia, where a cavity was found in the internal tuberosity, involving almost all of that portion of the epiphysis. In the cavity was found a sequestrum as large as a good sized pea. Free communication existed between this cavity and the joint, the cartilage being perforated. Pus had thus found its way into the articulation, producing complete joint disintegration. The condyles of the femur were not involved, a slight erosion only existing on the articular cartilage of the internal condyle. Comment upon this case is, perhaps, unnecessary. I will merely call attention to the initial lesion, which was followed by the early stiffening of the joint, the muscular atrophy and spasm, the absence of swelling, and the osteitic cry, all of which existed before the epiphyseal abscess found exit in the cavity of the joint.



In conclusion I wish to call attention to this peculiar muscular spasm in chronic joint disease. It would be a difficult matter to describe it in such a manner that it would be recognised. Like the physical signs in thoracic disease, its peculiar qualities are best acquired by clinical study. I shall attempt to describe it when I speak of the symptoms of joint disease, in detail. I have elsewhere stated that I believe this spasm to be due to an irritation or inflammation of the peripheral nerves supplying the joint, and that the spasm thus induced produces the various progressive deformities in joint disease.\* Without anticipating what I shall say upon this subject on some future occasion, I may remark that further investigations have only strengthened the position I take in the article referred to. That the trophic changes occurring in joint disease, and the reflex spasm which accompanies it, are due to the same cause I have no doubt. Billroth remarks: "The extent to which the muscles sympathize varies greatly; according to my experience the highest grades of muscular atrophy occur in those cases where there is no supuration of the joints but *caries sicca*, and where the joint disease seems to proceed from osteitis."† In the paper already alluded to I say, "In chronic osteitis, especially if associated with a chondritis, the contraction is firm, tense, and very persistent. It increases with greater or less rapidity, until ankylosis is simulated. The atrophy is slowly but steadily progressive, and the muscles show a marked decrease of faradic reaction. This is especially the case in the dry osteitis." I had not, when I wrote this, seen the above sentence, quoted from Billroth, else I should have referred to it in my paper. The neural symptoms are so marked in some obscure joint lesions as to be suggestive of a primary spinal cord change, a condition which Dr. S. Weir Mitchell calls attention to in his paper on

---

\* Op. cit.

† Op. cit. p. 481.



"Spinal Arthropathies."\* In a letter to me, dated November 5th, he says: "There is a resemblance between the two conditions you speak of," (the muscular spasm and atrophy occurring in joint disease, and that following injuries of nerves), "both sets of contractions and atrophies result from irritative peripheral lesions of nerves, in or out of joints; and in the joints, as in the nerve trunks, the muscular loss is often out of proportion to the joint lesion, and this especially in the knee and shoulder—the small joints scarcely cause it,—but I fancy that more often than we suppose the joint and muscle lesions are common results of a spinal malady." I have often expressed a desire to subject the spinal cord to a microscopical examination, my thought being that in some of these old cases, where caries sicca had existed for years, some change might have occurred in the cord as a result of the prolonged reflex movement. The cases of spinal arthropathy which I have seen, differ in many important respects from the lesion I have attempted to describe, where, as I believe, the neural symptoms are purely reflex. Hereafter I shall speak more particularly upon this extremely interesting subject, and after studying M. Valtat's monograph, "*De l'Atrophie Musculaire Consecutive aux Maladies des Articulations*," a recent work, which I have just received from the author.

---

\* Am. Jour. Med. Sci., April, 1876.



