disease of the joint is present, you will be able to detect it. You will also observe that there is no abduction or twisting of the leg outward, as shown to result when the joint has been long involved; but, on the contrary, the leg will be found flexed in a straight line with the femur, and has no outward rotation. The external openings of sinuses communicating with dead bone have such a characteristic appearance, described by the late Dr. Alexander Stevens as resembling the anus of the hen, as to be absolutely unmistakable. When this is present, therefore, you will at once use a flexible probe (*see* Figs. 144 and 145), which will follow the lead of any opening under the fascia or elsewhere, and finally conduct you to the dead bone, and then your diagnosis is positive.

In some cases, however, which have fallen under my observation, there were no openings until I had made one for the purpose of exploration. Such an incision can be made through the vastus externus muscle, when the bone is very readily reached without incurring any risk from hæmorrhage.

The incision will probably give free discharge to pus; and then, with your finger or probe, the exploration can be continued until the diagnosis is completed. In some cases, perhaps, the parts can be saved by making a free incision through the periosteum before death of the bone takes place. When diseased bone is found, proper measures can be resorted to for its removal. If you are not able to remove all the dead bone at the time of the first operation, draw a seton of oakum or an India-rubber tube through the wound, and leave Nature to remove the remaining portion.

An important point with regard to operations for the removal of dead bone in this region, as well as elsewhere, is to preserve the periosteum as much as possible.

The permanent deformity which commonly follows chronic disease of the knee-joint is anchylosis with distortion. The subject of anchylosis will be fully considered hereafter.

LECTURE XIX.

DISEASES OF THE JOINTS.—MORBUS COXARIUS.

Anatomy of the Hip-Joint.—Pathology of Hip-Disease.—Etiology.—Symptoms of First Stage.

GENTLEMEN: We shall next consider that malady which occupies the chief place among affections of the joints, namely, Morbus Coxarius, or hip-disease. But, before entering upon the consideration of the symptoms and morbid changes of structure in this disease, it will be necessary for me to give a brief description of the most important anatomical structures entering into the composition of the hip-joint, in order that you may fully comprehend the principles which I shall endeavor to establish as the proper basis for correct treatment.

ANATOMY OF THE HIP-JOINT.—The osseous structure of the hip-joint is made up of the *os innominatum* and head of the *os femoris*, the latter being received into a deep cavity of the former, the *acetabulum*, by a kind of articulation called *enarthrodial*, or ball-and-socket joint.

The head of the femur and the acetabulum are cancellous in structure; quite vascular, and subject to inflammation.

The acetabulum is lined with cartilage at all parts, except at a circular pit (fundus acetabuli), which occupies the lower part of the cavity near the notch, and is cushioned with fat. The head of the femur, which fits into and articulates with the acetabulum, is nearly two-thirds of the segment of a sphere, and is entirely covered with cartilage, except at the deep pit, which is for the insertion of the *ligamentum teres*, at its upper and inner face looking toward the cavity of the pelvis.

The proper ligaments of the hip-joint are the *capsular*, the *ileo-femoral*, the *ligamentum teres*, the *cotyloid*, and the *transverse*.

The *Capsular Ligament* (*A*, Fig. 146) is the largest and strongest capsule in the body. It is attached above to the outer border of the acetabulum and outer face of the cotyloid ligament; and below, to the anterior inter-trochanteric line, and neck of the femur, which latter it completely surrounds. It is thicker and

longer in front than behind, and it is more extensively attached at its upper part, where strength and security are required. The strength of the capsular ligament is further greatly increased by the *ileo-femoral ligament* (*B*, Fig. 146) which is accessory to it, and extends from the anterior inferior spinous process of the ilium to the anterior inter-trochanteric line. This ligament has been called the Y-ligament by Dr. Bigelow, of Boston.

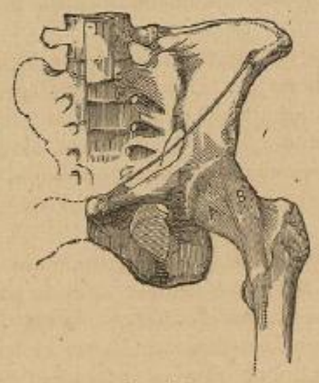

Fig. 146.

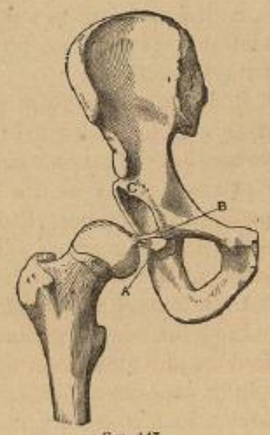

Fig. 147.

The *Cotyloid Ligament* (*C*, Fig. 147) is a thick prismatic ring of fibro-cartilage, mounting and attached to the brim of the acetabulum by which the cavity is deepened.

The *Ligamentum Teres* (*A*, *B*, Fig. 147) is attached by a round apex to a pit just below the middle of the head of the femur; it divides into two fasciculi, which are inserted into the corners of the notch of the acetabulum *A*, *B*, and the cotyloid ligament, and is covered by synovial membrane.

The *Transverse Ligament* is continuous with the cotyloid, extending from one point of the notch to the other, and completing the circle of the cotyloid ligament, thus converting the notch of the acetabulum into a foramen, through which the blood-vessels enter to supply the interior of the joint.

The synovial membrane is quite extensive, lining the capsular ligament, the free surface of the cotyloid and transverse ligaments and the ligamentum teres, as far as the head of the bone.

We are now ready to pass to the study of the pathology of this disease.

Pathology.—Under this head we shall describe the changes that take place in the tissues of the joint at the very beginning of the disease, leaving those which are present in the more advanced conditions to be considered in connection with the symptoms to which they give rise.

1. The disease may begin as a synovitis.

2. It may begin in a rupture, partial or complete, of the ligamentum teres; thereby interfering with the nutrition of the head of the femur.

3. It may begin from rupture of some of the minute blood-vessels which are situated in the bone just beneath the cartilage of incrustation. This may occur either upon the head of the femur or at some point in the acetabulum, and results from blows, jumping, or anything which may produce a sudden concussion of these articular surfaces. These three conditions require special consideration:

1. Of synovitis. Inflammation of the synovial membrane of the hip-joint may be produced in the same manner as it is produced in any other joint of the body, but it is almost always the result of exposure to sudden changes of temperature after violent exercise, such as skating, racing, jumping, playing at foot-ball and other movements that over-exercise the joint.

When the synovial membrane becomes inflamed, effusion of fluid into the cavity of the joint always takes place. The synovitis may be subacute in character, and attended by the effusion of only a small quantity of fluid, but not followed by disintegration of the tissues of the joint; or the same degree of inflammation, in some cases, may be followed by complete disintegration of the joint structures.

Again, the synovitis may be very violent, accompanied by intense pain and the effusion of a large quantity of fluid, and make rapid progress toward destructive changes within the joint. When the joint becomes distended with fluid there will be present a peculiar deformity, which we shall fully describe when we come to study the symptoms of the disease in its second stage. Of course the synovial membrane sooner or later becomes involved, as do the cartilages, ligaments, and bones, no matter how the disease begins; but that there are cases of hip-joint disease

which have their commencement in a synovitis I am fully convinced.

2. Any violent straining of the ligamentum teres, such as may be caused by forcibly stretching the legs apart, or by other violent exercise which gives motion to the joint to the extreme limits, may partially or completely separate it from any of its attachments to the bones. It is most likely, however, to be separated from its attachments to the head of the femur. When such an accident occurs the vessels which supply the head of the femur are destroyed, and necrosis follows as the result of interference with its nutrition. Secondary changes soon occur in the cartilages and the synovial membrane, and the case goes on, if not relieved, to the development of the disease in its worst form.

3. When the disease begins in the blood-vessels in the articular lamella, it first appears as an extravasation or "blood-blister" at some point. This is the nidus, or starting-point, and, if the damage done is detected at the time of the infliction of the injury, *rest, alone,* if continued for a sufficient length of time, will probably bring about a favorable termination in a great majority of instances. But the damage done not being detected, and in many instances not even suspected, the rest necessary is *not insisted* upon at the proper time; consequently the disease is slowly developed, and frequently is not distinctly pronounced until long after the accident that has caused the trifling damage to the blood-vessels, and given rise to so much trouble, has been entirely forgotten.

A pinch of the skin producing a "blood-blister," or slight extravasation of blood within the cellular tissue, is of common occurrence, and is of no great importance. If let alone, it will soon be absorbed; or at most, if you let the fluid out and do not irritate the wound, it will soon get well. But suppose, even in this most trifling injury, that, instead of giving it rest and time to heal, you constantly scratch it with a rusty nail; you will produce a sore that will last as long as the irritation is continued. This is a parallel case with a joint that is exercised after concussion, or a blow or wrench that has produced an extravasation of blood from the tufts of blood-vessels already referred to.

Now, while I believe that this disease begins in one of the three ways mentioned, I would have you understand that the disease does not progress very far, without involving all the structures entering into the composition of the joint.

For instance, when the disease begins as a synovitis, the cartilages, bones, and ligaments, sooner or later become involved. So, when the disease begins in destruction of the ligamentum teres, partial or complete, the same consequences ensue, and the same is true when the disease begins as an extravasation of blood in the manner described.

I do not believe, however, that the disease ever begins in the cartilages of the joint, for the reason that these structures contain neither blood-vessels nor nerves. Necrosis occurs *secondarily* in the cartilages on account of the loss of nervous and vascular supply to the tissues upon which they depend for nutrition.

This, according to my view, constitutes the pathology of this disease at its very beginning. There are other and very important pathological changes that occur as the disease progresses; but, inasmuch as certain symptoms, such as certain positions which the limb assumes, are directly dependent upon such pathological changes, I shall consider them in connection with the symptoms to which they give rise. We now pass to the subject of etiology.

Etiology.—Almost all surgical authorities agree that morbus coxarius is invariably the result of a contaminated constitution; in other words, that it is essentially strumous in its origin. This has been the universal opinion, and the doctrine has descended from teacher to student, and is still extant among the majority of surgical practitioners. It has been so often taught and enforced by frequent repetitions, that nobody considered it worth while to question its truth; but nearly all have taken it for granted that an assertion so positively made and universally accepted must be based upon mature investigation. When I first entered the profession I accepted this doctrine taught by our fathers, but must confess that I never was fully satisfied with regard to its correctness. Now, while I revere the labors of those great men in the advancement of scientific investigation, I must be permitted to question what is questionable, and to doubt what is doubtful.

Examination of the cases which have presented themselves to my notice since that time has convinced me that the cachectic condition so often seen is the *result* and not the *cause* of the disease; for very many of the patients in the earlier stages of the disease have possessed all the appearances of robust health, and, in all those cases in which the disease has been cured by Nature's method, the patient, subsequent to the cure, has been hale and

hearty. I do not suppose there is a person in this room who cannot call to mind some old fellow with a shortened hip, perfectly anchylosed, who yet has a ruddy face, a good healthy complexion, and is a vigorous, robust old man. If he had had scrofula in his system, it would have remained there, and when his hip had recovered the man would have been a miserable old fellow after all. The very fact of his becoming a vigorous, robust man after going through all the exhausting effects of hip-joint disease proves, in my judgment, that the disease is not of constitutional origin.

The additional fact that, in so many cases, the joint has been exsected when the patients have been, apparently, at the point of death, and after the removal of the dead bone have become vigorous, strong persons, is good evidence that the disease is not constitutional. Then there is the still stronger fact that, by treating the disease locally without reference to constitutional taint, we obtain perfect results, so much so that the patients recover with perfect motion and without the slightest deformity, which is the best proof in the world that the disease is essentially local in character.

Another fact worthy of consideration is that a very large proportion of cases of the disease occur in children, while the scrofulous condition is by no means so restricted.

I have unfortunately recorded only a small part of the cases which have fallen under my observation, but three hundred and sixty-five cases have been fully entered upon my record, and, of these, two hundred and twenty-one were under the age of fifteen years, and one hundred and twenty-one were under the age of five years. Similar results have been obtained by other gentlemen who have collected statistics upon this point.

Now, it is not necessary for me to prove that adults are nearly as liable to be affected with scrofulous diseases as are children, the less number of cases seen being due mainly to the fact that these sickly children are very liable to die before reaching adult life. If, therefore, we still adhere to the scrofulous theory, we are forced to conclude that the diathesis, which in childhood develops itself in joint-disease, manifests itself in some other way after puberty. This I cannot believe. Childhood is the age of restless activity, and, out of the hundreds of cases in which I have taken the trouble to trace their history, I have found that the

immense majority, I may safely say seventy-five per cent., have occurred in the most vigorous, robust, wild, harum-scarum children—those who take their chances of danger, who run races, climb over fences, jump out of apple-trees, kick their playmates down-stairs, ride down balusters, and are generally careless and reckless.

On the other hand, the adult does not place himself in the position in which he can receive so many blows or falls as the active child does, and furthermore he immediately notices the effects of his injury, and takes precaution against its development into serious trouble. The child, however, knows nothing of results, and, unless the pain from the injury is great, will probably fail to complain of it, and soon forget it altogether. This, I believe, is the true reason why so many more cases of joint-disease are seen in children than in adults.

I do not wish to be understood as saying that scrofula is a *preventive* of disease of the hip-joint, as has been asserted concerning my teaching. All things considered, a smaller amount of injury will produce the disease in one of these miserable, sickly children, than in a healthy, robust child. But the sickly, scrofulous child, who clings to his mother's apron, does not run the risk of getting hurt as do these active, restless children; consequently, the majority of cases occur among the active and robust.

From what has been said, you have probably already drawn the inference that I regard the disease as one almost invariably due to a *traumatic* cause, and *not* dependent upon some constitutional taint. To what has already been said upon this point, we may add the positive evidence of statistics.

Of the three hundred and sixty-five cases alluded to above, *traumatic* cause was assigned by the patient or the parent in two hundred and fifty-seven, while in one hundred and eight cases the cause was recorded as unknown.

In two hundred and seventy-eight cases, the previous general condition of the patient was good; in forty-two cases it was bad; and in forty-five cases it was unknown. These figures are taken from the notes of my own fully-recorded cases. Cases not fully recorded have been rejected in making these statistics.

Now, the cases in which the previous condition was bad, together with those in which it was unrecorded, make up less than twenty-four per cent. of the whole; and it is possible that very many of those had a traumatic origin that had been overlooked

or forgotten, owing to the insidious manner in which the changes had come on.

My own clinical observations with reference to this point stand by no means isolated. The same observations have been made by other surgeons, both in this country and Europe.

It generally requires a very close examination to find out the cause, since the disease does not usually immediately follow the injury, but often first manifests itself weeks, and even months, after the accident that has given rise to it has occurred; so that the patient and his friends naturally enough forget the accident and its connection with the disease, until especially reminded of it in the investigation.

So much, gentlemen, for the pathology and causation of hip-joint disease, and now we are ready to begin the study of its symptoms.

Symptoms.—These will vary according to the stage in which the disease presents itself.

Ordinarily three stages are described:

1. The stage of irritation or of limited motion, before the occurrence of effusion.

2. The stage of "apparent lengthening," or of effusion, the capsule of the joint remaining entire.

3. The stage of "shortening," or of ruptured capsule.

For the second and third stages, I prefer to use the terms *effusion* and *rupture*, rather than "apparent lengthening" and "shortening," as the latter describe only a single feature of the deformity present in each stage, while the former designate an essential pathological change which underlies a group of symptoms. What, then, are the symptoms of the *first stage?*

The symptoms of this stage are sometimes exceedingly obscure, particularly if the inflammation be of a low grade, or of the chronic character generally found in those of a strumous diathesis. The first thing that attracts the attention of the patient or his friends is generally a stiffness about the joint and a limping gait, for which, perhaps, they will be unable to assign a cause. The real cause (commonly traumatic) has been forgotten in consequence of the slow and insidious approach of the disease. This stiffness of the joint is commonly noticed first in the morning when the patient gets up. After he has been about for a while he becomes limbered up, and can travel without stiffness or appreciable limp. But, even then, when he stops walking or running he will, within a minute or two, invariably stand upon the sound leg, apparently for the purpose of relieving the affected one.

Now, even at this early stage of the disease, if the patient be taken to the surgeon, a careful examination will reveal the following condition of things:

It is to be noticed, however, that no deformity of which you are certain can be detected at this stage unless the patient is completely stripped of clothing from the waist down, and then placed in a proper position.

When the patient has been stripped, place him first in the standing position, and directly in front of you with his back toward you.

The light should fall directly upon his back, in order that you may not be deceived with regard to details of contour by any shadows. Your examination should not be hurried, for you wish to detect the disease in its very incipiency, in its most shadowy form. After watching the patient a short time you will notice that he makes a solid column of the sound leg for the purpose of receiving concussion and bearing the weight of the body, and also carefully avoids all concussion of the suspected limb. You will further notice that the suspected limb has a tendency to slight abduction and slight flexion at the knee and hip, but the feet stand parallel with each other. The natis upon the side of the lameness drops a trifle, is somewhat flattened, and the gluteo-femoral crease is lower and shallower than upon the healthy side. (*See* Fig. 148.)

This dropping of the natis is due to relaxation and gravitation of the gluteal muscles while the weight of the body is thrown upon the sound leg; for the same thing occurs if the knee-joint be affected, or a perfectly sound person throws his weight upon one leg.

This symptom, then, has a diagnostic value only so far as this —it indicates to us that from some cause the patient rests the weight of the body chiefly or entirely upon one limb. But from this peculiar favoring of the affected side we can often detect the incipient disease, even before a limp has been noticed. Next you will determine whether there is present any rigidity of the psoas magnus, iliacus internus, or adductor muscles of the thigh; for

rigidity of these muscles appears very early in the disease, and, if none of them give resistance to the full performance of their normal functions, it is fair to assume that the joint is not diseased.

To make an examination for this purpose it is necessary to lay the patient upon his back upon a firm, flat surface like a table

Fig. 148.

or floor. This examination *must* be made upon a *solid*, flat surface. A bed, or sofa, or lounge, therefore, will *not* answer; for the inequalities of either will adapt themselves to the curvatures of the spine, thereby preventing you from detecting the deformity of this early period of the disease.

Before proceeding further it is necessary to place the patient in such a position as will furnish a proper starting-point from which you may conduct your examination. Such a position is one in which the pelvis and trunk are at right angles with each other, and is obtained in the following manner: Lay the patient on his back upon a table, or some solid surface, covered only with a blanket, in such a manner that his entire spine will be brought upon the plane. This can be done by placing your arm under

the knees and lifting the thighs, or by lifting them in any other way, until the spinous processes of the vertebræ have touched the solid plane upon which the child is lying (*see* Fig. 149). Then draw a line from the centre of the sternum over the umbilicus to the centre of the pubis, and cross it at a right angle by a line drawn from one anterior superior spinous process of the ilium

Fig. 149.

to the other. When this is done, and the two lines above mentioned are at right angles, the spinal column is slightly straighter than normal, but it and the pelvis are at right angles with each other; and, if no disease exists within the hip-joint, the limb can be brought down, so that the popliteal space can be made to touch the plane, without disturbing the relation of the lines above described, or lifting the spinous processes from the plane. If you, therefore, hold the suspected limb in your hand in such a

Fig. 150.

manner as to keep the spinous processes on the table, while the other lines are at a right angle, you will observe that the well limb can be pressed down to the table so that the popliteal space will touch (*see* Fig. 150). The diseased one can be pressed down to nearly this position, but, before the popliteal space touches the plane, you will notice that the pelvis becomes tilted, making a curve in the lumbar vertebræ so that the hand can be passed between the child's back and the table (*see* Fig. 151).

This arching of the spine in many cases at this early period in the disease is so *slight* that it would be *entirely* overlooked were the examination made upon other than a *solid* flat surface.

Complete flexion at this period of the disease is also impossible. The well limb can be flexed so as to bring the knee in contact with the chest; but the diseased limb can probably be flexed only at a right angle or a little more than a right angle with the

FIG. 151.

body, before the pelvis will be raised. The moment the pelvis begins to rise, that moment you have reached the limit of flexion.

Adduction is very limited indeed. The diseased limb cannot be crossed over the opposite limb, and even by the time it has reached the median line the pelvis begins to move, showing that you have reached the extreme limit of adduction.

Abduction, particularly if the limb is slightly flexed and at the same time rotated outward, can be carried to an extent somewhat greater than adduction, but *not to full* abduction, before the pelvis will begin to move, showing that muscular rigidity is present.

Now, in whatever position the affected limb must be held in order to bring the pelvis and trunk into a normal relation with each other, that is, so that the two lines mentioned shall cross each other at right angles and the spine be upon the table or floor—such position indicates the *deformity* present at the time of making the examination, and the stage at which the disease has arrived.

In the first stage, therefore, as can be seen in these cases before you, the thigh is flexed very slightly upon the pelvis, and very slightly abducted; and, the pelvis being held *perfectly still*, very limited motion can be made at the joint, when slight extension is made upon the limb. Attempts to *extend* the limb beyond a certain point, as you now observe, tilt the pelvis; flexion beyond a certain point—in this case not quite to a right angle with the body, in other cases it may be to more than a right angle—tilts

the pelvis; whereas upon the well limb extension can be made complete, and flexion complete, so as to bring the knee against the trunk.

Abduction, adduction, and rotation, are also limited, as you observe, and when carried beyond a certain point the pelvis at once moves with the limb, giving the patient an appearance as if complete anchylosis had taken place at the hip-joint. But there is no real anchylosis present in this stage of the disease. There is anchylosis, perfect and complete to all *appearance*, but it is due simply to muscular rigidity. For, by placing the hand upon the pelvis, and making gentle extension upon the limb for a few seconds in the *line of the deformity*, motion can be made at the joint without causing pain; but the moment extension is removed limited motion causes pain, the muscles suddenly become rigid, and the child can be rolled around like a solid marble statue.

If the disease, however, has passed beyond the first stage, and effusion has taken place, then abduction is much more marked, and flexion is much stronger than in the first stage, but the *peculiar* feature of the deformity then is *eversion* or *rotation* of *the foot outward*. These symptoms will be more fully considered when we come to speak of the symptoms of the *second* stage.

Another symptom of the first stage that is too often overlooked is atrophy of the thigh or entire limb. Therefore, always compare the limbs by actual measurement, for the rapidity with which atrophy takes place in some cases is really surprising, and is due to the direct influence of immobility of the joint. The symptoms, as we have studied them thus far, all point to one thing, namely, fixation of the joint, restraining motion as much as possible. This will occur without the slightest recognition of pain on the part of the patient, and is due to what Mr. Barwell terms "joint-sense."

The symptoms of which the patient will complain are tenderness and pain. Tenderness is usually well marked, although sometimes it is necessary to make a thorough examination of the joint before its presence can be detected. The disease may be situated at any part of the joint-surface, and we ought, before denying the existence of tenderness, to make pressure upon every part of the head of the femur or acetabulum that could have been involved in the original injury.

This can be done by placing the thigh in all possible posi-

tions, and at the same time making pressure upon the head of the bone and the acetabulum by crowding the articular surfaces together.

In addition, pressure should be made upon the great trochanter in order to bring the head of the femur and acetabulum in contact from that direction.

Again, holding the knee with one hand and fixing the pelvis with the other, press the thigh-bone upward. This manœuvre generally causes pain, which can be detected in the patient's face, even when he denies he feels it. If the manœuvre *does* cause pain, then observe whether or not extension relieves it. To make your examination doubly sure, if tenderness has not already been detected, sweep with the thigh its largest possible circle, by which means the head of the bone cannot possibly escape being brought in contact with every part of the acetabulum.

Pain may or may not be experienced during the *first* stage, independent of motion or pressure upon the joint surfaces.

In those cases where the disease manifests itself immediately after the injury—which cases are probably either synovitis or periostitis of the great trochanter—the pain is also immediate and constant, and frequently excruciating.

In other cases, when probably the seat of the disease is in the articular lamella—either beneath the articular cartilage of the head of the bone or the acetabulum—pain is developed late in the first, or even not until the second stage.

This pain may be referred more or less definitely to the hip-joint and its surrounding tissues, or it may be so entirely located in the knee as sometimes to completely mislead the surgeon in his diagnosis. I have many times seen the knee blistered and treated for months, when there was no disease whatever at that joint, it being merely affected by the disease in the hip.

Mr. Barwell explains the knee-pain as follows: It is produced (1) by direct irritation of the nerves passing in close contiguity to the joint. These are the obturator nerves, the sciatic, the gluteal, and perhaps the anterior crural. It is produced (2) in consequence of an obscure sympathy between the two ends of the bone, or even direct propagation of the inflammation from one to the other; and (3) by spasm of certain muscles.

Such, gentlemen, are the symptoms by which you are to recognize hip-joint disease in the *first* stage.

No one of them is entirely diagnostic. The certainty of the diagnosis depends upon a careful consideration of *all* the symptoms described.

We have thus dwelt upon them at some length, because many of them differ from those of more advanced stages only in degree, consequently require only one description; but more especially because it is in this stage that the diagnosis is most difficult and important. In the later stages, it is almost impossible not to recognize the disease, but the patient has then endured great suffering, and perhaps irreparable mischief may have resulted, which might have been easily *prevented* had the true nature of the disease been early recognized and properly treated.

LECTURE XX.

DISEASES OF THE JOINTS.—MORBUS COXARIUS (CONTINUED).

Symptoms (continued).—Symptoms of the Second Stage and their Explanation.—Case.—Symptoms of the Third Stage.—Discussion of the Question of Dislocation in this Stage.

GENTLEMEN: To-day we will continue the history of hip-disease by first studying the symptoms of the *second stage*.

The symptoms described at our last lecture as belonging to the first stage—namely, pain, tenderness, swelling, atrophy, and limited motion—continue into the second stage of the disease, but are generally increased in severity.

The peculiar position of the limb gives to the second stage of the disease the name "apparent lengthening," but I prefer to designate it as the stage of effusion.

If you examine the patient while in the standing position, as in our previous examination (*see* Fig. 148), it will be noticed that the foot is now *everted*, and the leg is a little more flexed upon the thigh, the thigh is a little more flexed upon the trunk, the obliteration of the gluteo-femoral crease a little more marked, and the entire limb more markedly abducted.

The foot upon the affected side is somewhat in advance of the