On Gelatinous Degeneration
With special reference to its Pathology.

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1879.
Gelatinous Degeneration
with special reference to its Pathology.

I was led to choose this peculiar articular
disease as subject for a Thesis, on account
of the number of well marked Cases of the
affection in the wards of the Royal Infirmary
Edinburgh, under my charge, as resident
Surgeon there last Summer.

All joints seem liable to be affected
by this disease, but the Knee is the artic-
ulation most commonly attacked.

Although it may occur at any age,
the majority of patients thus afflicted have
not arrived at the period of puberty.

Possibly the best introduction to a
study of this affection, will be to describe
or case, as a type of those seen in hos-
pital; the following then, may serve this
purpose.

A—B—act 10, admitted to the
Royal Infirmary, complaining of a swelling
of the right Knee joint, & inability to walk.
History.

11. Family: Father alive and healthy. Mother died three years ago of Phthisis. Has two brothers and one sister, all alive and seemingly healthy.

12. Personal: Has had comfortable home. Had Scarlet fever some time ago. No other ailment till the onset of the present illness. While playing with some of his companions, he was pushed and fell, giving his knee a slight wrench. Was able to walk home, and continued to go about as usual.

Soon afterwards, he began to feel a slight impediment in locomotion. This condition not improving, his father on examining his legs, found the right knee joint slightly Swollen; but as it was not painful to the touch, he did not consider it anything serious. However, as the boy became more and more unable to walk, and as the swelling had increased considerably, he brought him to the Infirmary for advice.
A few days afterwards, the lad was admitted to Ward —
Status on admission:

He seemed fairly nourished. Temperature and pulse normal. Systems normal, except a few enlarged glands in the neck.
The right knee joint presented a diffuse swelling of a semi-solid consistency. No fluctuation could be felt. The temperature was appreciably higher than in the left knee. On pressing the head of the tibia, slight pain was caused. The integument over the swelling was paler than usual, quite smooth. The contour of the articulation was completely lost.

Diagnosis: Relapsing degeneration of the knee joint.


Progress: A week afterwards, pain was better. Fomentations were discontinued. Patient seemed to improve for a time, but about a month after admission, pain returned to the head of the tibia.
Progress, continued.

A rigor ensued, and was followed by a marked elevation of temperature; at night it rose as high as 104°. Formulations were reapplied, and Quinine was given at bedtime, in 10 Grain doses. The pain increasing, & the temperature remaining high, it was thought necessary to cut down on the head of the Tibia, through the periosteum, with antisepsic precautions, in order to relieve tension.

Next day, the pain was much less, & the temperature nearly normal. Patient now continued to improve, and in six weeks, a Thomas' splint was ordered for him, & he was discharged much better.

About seven months afterwards, he was brought back to hospital; an abscess having formed at the inside of the articulation; this had been allowed to open spontaneously. On examination, it was found that the joint cavity was opened into, and
Progress, continued.—

Disintegration of the articulation had proceeded so far, that distinct grating was felt when the bones were forced together.

Patient's general health was so much impaired, that it was thought advisable, considering the state of disorganization in which the joint was, to amputate the limb.

This then may convey, in a general manner, an idea of the kind of case we met with in hospital practice.

Of course, many details are absent from the example just cited; but, in the subsequent consideration of this affection, these will be duly noted.

I propose to examine the phenomena of this disease, in the following order:

1. The Nomenclature.
2. Etiology.
3. Pathology.
4. General indications as to Treatment.
Nomenclature.

The name generally accepted in the Edinburgh School, is "Relatious degeneration," so called, from the jelly-like appearance of the transformed textures, to the naked eye.

"Pulpy degeneration," is a term more used in the London School.

"White Swelling," is another widely accepted name; the "Tumour Blanche," or "Tumor Albus," of the Continental writers. This title also is obviously given to express in a general way, the external appearance of the articulations affected by the disease.

Again from the association of this morbid process with the Strumous Diathesis, it has been called "Chronic Strumous Arthritic," "Scrofulous Disease of Joints."

"Fungous Arthritis," "Fungus Articuli," are names also adopted by some; while Rheinfleisch believing this disease to have its starting point in the Articular ends of bones, calls it "Caries fungosa."

From difficulties in determining and from the varieties of opinion as to the primary
seat of the morbid process, it is impossible to give a name anatomically and pathologically exact, but if we are contented with a Nonum
nature founded on the appearances presented by the altered textures then "Pathological Degenera-
tion" aptly enough describes it; though strictly speaking it is only the Synovial mem-
brane, ligaments & periarticular tissue which are transformed into this jelly-like matter,
the Cartilages & bones breaking down and disappearing either through invasion by this
morbid material or by some concomitant pathological process.

Etiology

Under this head at the very outset we are met with the query, Is this disease per se one of
the manifestations of the so-called Strumous Diathesis? That is to say, does this disease
arise spontaneously without any definite or determining Cause in the Strumous?

On looking over reports of these cases we find that in a very large majority the
family history reveals this constitutional fault.
A very great proportion of cases occur in early life. Patients may, up to the time of the appearance of this illness, have been quite healthy and may even then appear well nourished, their circumstances and surroundings may always have been quite comfortable; no history of anything which is supposed to have caused this ailment can be obtained. Yet, when they present themselves for treatment we find typical cases of this affection.

Having recognised the disease we direct certain general principles of treatment towards the local manifestations but we also proceed at once to combat the constitutional fault. In a great number of cases where the patient has come under notice at a comparatively early date and where the financial circumstances are such that every means of treatment are available, we effect a cure at all events of this phase of the constitutional weakness.

However, it is quite evident that we find a certain number of well marked examples of this disease where we cannot in the patient's family history discover the
Slightest trace of Constitutional Pains? What no sign of Struma or other weakness besides this presents itself. Only here we shall generally obtain a history of some fall blow or strain which the patient rightly or wrongly assigns as the initial element or cause of the illness.

Now then we have to do with a disease evidently of an inflammatory type which in every respect except in the absence of Constitutional Pains & the history of a fall or blow as a starting point resembles the previous instance the symptoms & progress being in both cases exactly alike.

Before going further we must make quite sure that besides the Clinical phenomena the pathological changes in the tissues involved bear out this resemblance. Examination of Specimens as we shall see afterwards shows that they cannot be distinguished.

How then is this to be explained? Are we to suppose that there has been a defect in our observation as to inherited Constitution? Surely not, for in many cases we
are in a position to convince ourselves fully on this score.

Are we to infer that, in consequence of, or superimposing upon, an accidental inflammatory affection of an articulation, e.g., the Knee, for the treatment of which we have been compelled to keep our patient at absolute rest, confining him to his room &c. &c., to some extent lowering his vital powers, we have brought about a state of body similar to that of our hereditarily strumous patient, &c, in virtue thereof, changed the type of disease from the purely inflammatory, to the purely Constitutional?

Such a hypothesis is too weak to be accepted, and in rejecting it, we admit that the disease may be of the inflammatory type.

If, then, we have to deal with an inflammation, the products are certainly in the first instance inflammatory, and not degenerative.

Here, then, it would appear, we arrive at the point where Constitutional defect or other depraved state of bodily health exerts a determining influence on the
Subsequent Course of the Affection.

Following out an ordinary inflammatory history, in a great many of our cases we expect to obtain resolution of the morbid products, & the restoration of the affected part, to a state not greatly removed from the normal condition.

Do we get this result here? In some cases we do: but in so few, that this most favourable ending may be ignored.

What then is it that prevents the organisation of the inflammatory products, and permits the retrogressive changes which follow? I believe it is at this point that the connection between this disease and Struma begins. Anything that will lower the natural reparative powers, by depressing in a general manner the vital activity, whether it be constitutional or acquired, may thus influence the progress of the pathological process.

But how are we to explain the fact, that it is amongst the members of Stramous families, we get such a large majority of our cases? We must admit that the
Great predisposing cause of this malady, is the strumous diathesis.

We cannot believe in the spontaneous or idiopathic development of this affection, but given the inflammatory attack, excited it may be by something so slight as to have escaped notice; this primary constitutional condition, or an acquired depraved state of body, may determine the peculiar type of disease we are now considering.

Thus then we resolve the etiology into:

(1) Some one or other of the ordinary exciting causes of inflammation. Together with,

(2) The strumous constitution, or it may be some other peculiar condition of body. As causes which determine the subsequent course of the affection.
Pathology.

The external appearance of articulations affected by this disease is striking enough, in so far as we have the normal configuration of the joint; the contour of the articular ends of the bones, the natural markings appreciable through the surrounding soft parts on inspection; if especially on palpation, replaced by a diffuse and general swelling, the integumentary covering of which, does not differ greatly from the normal, except perhaps in the obliteration of the natural folds of the skin; if the possible passage over its surface of a few distended veins. No inflammatory redness is present; on the contrary, the colour is, if anything, a little paler than natural; the skin being stretched and smooth. The general swelling is the more marked from the fact that the muscles in the neighbourhood have atrophied; in what manner we will afterwards examine.

The consistence of the swelling is the next point of importance; there is a semi-solid, doughy feeling about it which is remarkable, not the feeling of an edematous part.
Because in this stage of gelatinous degeneration there is an elasticity about the swelling which after digital pressure causes the parts to resume their original appearance.

Sometimes, through this thorough enlargement, unmistakable signs of fluctuation can be made out, but this is by no means an invariable condition.

As a rule, the pressure causes no pain to the patient, but there are, occasionally, painful spots; and in almost every case, if we examine the same patient at intervals over some extended time, we shall sometimes find the very slightest touch causes great pain; more especially, if we press over the articular extremity of one or all the bones forming the articulation.

In many cases, we find in the neighborhood of the articulation, the orifices of sinuses, which, on examination, may be found to end in the periarticular tissues, or may communicate with the joint cavity.

Again, we may find the actual state of matters which has occasioned the sinuses, namely, abscesses in the periarticular
Textures, which may or may not communicate
with the joint cavity.

According to the stage of the disease, we
find a modification of these appearances:
at the beginning, a slight diffuse painless
swelling of a soft elastic consistency, non-
fluctuating, with no discoloration of the
integument; this gradually increases, until
the contour of the joint cannot be recog-
nized; and afterwards, traces of local
inflammation in the swelling may be
superadded.

The position of the limb may also
be remarkable. If means have not been
taken to keep it in the straight position
(taking the knee as the example of the af-
fected joint) we find the leg becoming
gradually flexed upon the thigh, till they
may touch each other. This condition
is brought about by muscular action.

The position of greatest capacity of the
joint, or of greatest ease to the patient,
at certain stages of the disease,
by dislocations arising from destruction
of the articular textures.
Drawing from a knee joint opened into by a transverse cut below the Patella.
To expose the lower end of the Femur, the Patella & some skin were removed.

a. Portion of Shaf of Femur
b. Condylar end of Femur covered with Glistening material
c. Portion of Cartilage on trochlear surface uncovered
d. Folds of Skin thrown back
Let us now cut into the joint. In the articular cavity we find a certain amount of fluid, which may be more or less altered. Synovia, mucos-pus, or pus: there may be pieces of cartilage, & other debris floating about. If a peri-articular abscess has formed, & opened into the joint, the quantity of pus is much greater.

We notice that every texture forming the articulation is changed; the synovial membrane, capsule, ligaments & the surrounding cellular tissue are much thickened; the distinctive appearance of fibrous & fatty tissue is lost to a great extent, their place being taken by an almost homogeneous opaque jelly-like texture.

This tissue also covers the articular cartilage more or less completely. In the knee joint before we now (side drawing), the only portion of articular cartilage to be seen, is a piece less than a square inch in size, situated on the trochlear surface of the end of the Femur, between the Condyles; the rest of the cartilaginous investment of the end of the Femur, & the
Whole of the cartilage covering the articular surface of the head of the tibia are hidden by the deposition of this jelly-like tissue on their surfaces.

At this stage of the disease, I found in four knee joints I examined in the Edinburg Infirmary, a marked thickening of the investing cartilages, less in degree at the points where the cartilage of the one bone is opposed to that on the bone with which it articulates. At the periphery of the opposed surfaces, and at the sides, the thickening was very remarkable; in some instances, the cartilage was more than half an inch thick, as can be seen from the sections I made through these thickened portions.

This thickening of the cartilages, I do not find alluded to particularly by any writers on this subject, except Cornil & Ranvier, whose explanation of its occurrence will be given later on.

The articular ends of the bones are also palpably enlarged, very vascular, and much softened.
This seems to be the state of affairs in advanced cases, where as yet, there has not been much suppuration, or breaking down of textures.

Without septic influences being superadded, abscesses called "Sympathetic," form in the periarticular soft parts, as has been mentioned may open into the cavity of the joint, and accelerate the process of disorganization of the textures. But, let one or more of those abscesses open externally, and let the influence of septic organisms be added, and then disorganization proceeds rapidly.

This jelly-like new material, which as we shall see, is a kind of granulation tissue, breaks down into fine, in some cases previously undergoing a process of fatty degeneration (see Sections alluded to more particularly under Histology).

The cartilages now become gradually destroyed though apart from septic irritation there are other causes at work bringing about their destruction.

Now the surfaces of the bones are exposed.
And the osseous textures participate in the process of disorganisation.

Thus, according to the stage at which we get the disease, we can recognise and explain the condition of parts we find.

If we have an articulation, around which abscesses have opened externally, it also into the joint cavity, we may find the cartilage at some parts, still covered over with granulations; or it may be so thinned, thinned, or surrounded, or ulcerated, as to be hardly recognisable; at other places, it is quite destroyed; if the end of the bone is exposed, the bone generally, is soft, cheesy, and easily broken down. The muscular wasting alluded to before, has steadily progressed; on section, we find the muscular tissue much paler than normal.

Histology.

During early stages of the disease, in two cases, one a knee, the other an elbow joint, which we had in St. Duncan's wards last summer, it was deemed advisable to open into the joints antiseptically, in order to
relieve the pain arising from increasing tension. I had thus an opportunity of procuring some of the Granulation tissue into which the Synovial membrane was being transformed, while as yet the articular Cartilages were quite smooth, and uncovered by this texture.

The specimens so obtained, illustrate the appearances presented by the Gelatinous material, in the early stages of Aseptic Case.

In contrast to these, we had other two cases; this time a hip, and an Ankle joint; where operation was had recourse to early, as the strength of the patient was being rapidly exhausted. Abscesses having formed round about, & opened externally & into the articular cavity. The Gelatinous tissue was breaking down, & the Cartilages were thinned & eroded at points, from the rapid deorganisation ensuing after Septicity had been introduced.

The appearances noted here, may be taken as showing the changes in the structure of the Granulation tissue, produced
Glandular tissue from Elbow joint (Prep I)

Shows the stroma & the fibrous stroma, but especially the large cells of epithelial type mentioned as found only in this preparation.
Relativeness material from knee joint (Prep II)
Shews the ordinary structure of the gelatinous tissue during the early stages of the disease in acritic cases
by the small round cells in a fibrous stroma.
by its exposure to septic influences.

In the Elbow (Prep No I), the Synovial membrane had, from the deposit of inflammatory products, become much thickened; on microscopic examination, was seen to consist of, structurally, a very delicate fibrous stroma, studded all over with members of small round cells, exactly resembling leucocytes; here and there a number of large nucleated cells of the epithelial type.

This then, except in the presence of these large cells, represents a typical inflammatory exudation, with its migratory corpuscles and fibrillated lymph.

What the large cells are, unless they are the remains of the endothelial lining of the Synovial membrane, I am unable to say; as they do not occur in any of the other preparations.

The gelatinous texture from the Knee, shows the same fibrous stroma with its small round cells (Prep No II).
A cartilaginous material from a hip joint (Prep III)

Shows the changes in the structural elements subsequent to the introduction of sepsis, e.g., the loose stroma, granulocytic cells & granular debris resulting from the breaking down of the cells & fibres.
In both the Hip and Ankle cases, the structural elements are the same, but the Stroma is looser, the cells granular, and their shapes more irregular; besides this there is a quantity of granular material, the result doubtless of the breaking down of the cells and fibres. (Prep No III)

As to the Cartilage at this stage: in the Ankle joint case, where the foot was removed, several of the Tarsal joints were affected to a slight degree by this disease; so that they may be taken as examples of it in its early stages.

I examined the Calcaneus. Caleiform articulation.

There was no ulceration of the Cartilage; nor was it at any point covered over with granulations; though the soft parts around were gelatinous.

On examining a vertical section through the Cartilage, of a small piece of the bone (Os Calcin) (vide Preps. No IV); the Cartilage-cell proliferation of cells are seen to have proliferated, more especially near the attachment to the bone. There,
Cartilage to bone (Os Calcis) during early stage (Prep IV).

Shows the proliferation of cartilage cells within the primary capsule.
A capsule with its contained cells looks somewhat like a bunch of grapes. Some of the capsules containing 6 or 7 cells.

The structure of the cells is not altered, nor is the method of their arrangement, for as we pass from the bone to the surface, the disposition of the cells is in rows, 

They become flattened out parallel to the surface, as we get to the free border.

The bone here presents no morbid change. The bone except that in the cancelli there are numerous small round cells, mingled with the ordinary fat cells therein contained.

Later stages.

From what we have been able to make out in the early stages, it from the preparations before us, showing the histology of this disease in its advanced stages; the progress of the involved process is made quite evident.

The granulation tissue has gone on creeping over the Articular cartilage, it may form a complete covering, if the rapid disorganization set
We by the formation & opening of abscesses it does not ensue before its accomplishment. The tissue is now more highly organised; the original texture with its fine fibrous stroma, small round cells has become vascularised; & a much dense fibrous tissue has been formed. This then, more or less completely invests the cartilages & in a peculiar way appears to affect its nutrition.

We saw in the early stages, a certain amount of cell proliferation within the capsule, but as structural change; this proliferation has gone on in a remarkable manner (vide prep. XVI); the young cells have escaped from their capsule so that all attempts at making out their normal arrangement in lines like needles. Resulting from this, the cartilage is very much thickened.

Near the synovial surface, the cells, as they usually are in this part are flattened out, but this flattening here is much exaggerated; the cells in many instances being almost linear. In this situation, the matrix also is changed, it having become distinctly fibrocellular. This can be seen in Prep XVI.
Where there are cartilage cells apparently lying in the layers of the newly formed fibrous tissue; this latter being really the altered matrix.

The fibrous tissue, i.e., the gelatinous covering is much more dense near the cartilage than if it is as you pass from it.

At this stage, we note that the ligaments, tendons, and periarticular tissues, have been transformed into the gelatinous tissue.

So far then, there seems no reason why, if the inflammatory process were to cease, we should not have recovery with a limb fibrously enchylized. The cartilageous investments of the ends of the bone forming the articulation, are connected by this fibrous tissue, which seems highly enough organized.

In some cases possibly the health having improved, this does take place; if so in the best results we could hope for, if the disease has so far advanced.

We now come to the point where from failure of strength, exacerbations of inflammation, or other local nutritive or trophic
Cartilage & adipose fringe from Knee (Prep VI)

This represents somewhat diagrammatically:

1. Flattening of cartilage cells & fibillation of matrix
2. Passage into the cartilage of a process of the new tissue
3. Dense fibrous layer next the cartilage
4. Loose fibrous tissue as you pass from it with
   a transverse section of a vessel & a fatty patch
   in its midst.
Changes, degeneration of the transformed tissue begins to be noticed.

Here and there, in the newly formed tissue, patches are seen which are truly fatty. The dense fibrous layer next the cartilage still exists, but as we pass towards the surface we find a variety of appearances presented: scattered all over, are both transverse & longitudinal sections of vessels in numbers; round these occasionally nests of cells; strands of loose fibres with round cells in the meshes, also the little fatty portions, caused perhaps by the occlusion of some small vessels.

In preparation No VII made from a septic case, in an advanced stage of disorganisation; it may be seen that almost the whole of this new tissue has been transformed into fat.

The structure of the cartilage cells has now also changed: the protoplasm of the cell has shrivelled up; it presents a granular appearance; the cell wall
Cartilage from a Knee (Prep VIII)

Shows the fatty degeneration of the cartilage cells.
The substance of the cartilage has collapsed giving a very irregular appearance to what remains of the original cell. Preparation No VIII stained with eosin + picric acid shows that the granular appearance of the protoplasm is due to fatty degeneration. These changes exist throughout the whole substance of the cartilage.

No doubt then in this way to a certain extent the cartilage is destroyed, but this is not the only mode in which its integration takes place. If we examine one or two more preparations (Nos IX) we shall discover another and more easily demonstrable method whereby the principal destruction of the cartilage goes on. Looking along the line of junction of the cartilage and the newly formed gelatinous tissue we notice at various points processes of this latter passing into the substance of the cartilage. On examining the cartilage transverse sections through similar ripple-like processes can be seen in the neighborhood of these processes.
the breaking down of the Cartilage is to be noted.

Thus then we can understand how in a very short time the whole of the Cartilage will be destroyed & the bone exposed to the same disorganising influence.

What the exact pathological condition of the bone is I have not myself been able to determine but in considering the views of the pathologist, who have written on this disease I will give their statement.

The formation of granulation tissue similar to that into which the Synovial membrane is transformed in the articular ends of the bones is described by nearly all authorities & the invasion of the Cartilage at the bony surface as well as at the other by those processes before mentioned.

None of my preparations show this, though in some of them the line of attachment of the Cartilage to the bone is preserved.
The muscular atrophy is due to two causes: 
1. disease or diminished functional activity 
2. a fatty degeneration of the muscular tissue

It is in the parts of the muscle in the immediate vicinity of the articulation that this latter change has been noted.

To sum up then we have first a neoplasm consisting of small round cells in a fibrous stroma resulting from an inflammatory affection of the synovial membrane. This seems to spread in the manner described over the cartilages. The ligaments of periosteal fibrous tissue are affected like the synovial membrane. The new tissue becomes vascularised and a more highly organised fibrous tissue is the result.

From irritation probably the cartilage cells proliferate and the cartilage thickens.

Now something turns the balance from the side of organisation to that of degeneration. The fibrous texture begins to break down and becomes fatty, the cartilage cells undergo
fatty degeneration, processes of the newly formed tissue pass into the cartilage and rapidly destroy it; the bones possibly being similarly affected.

Let us now turn to the literature of the subject and see to what extent these views agree with the observations therein contained.

It is somewhat startling to find that the three authorities who have written most on the subject differ from each other concerning the starting point and progress of this disease.

Billroth asserts that the synovial membrane is the first part affected in the great majority of cases.

In all the cases I have had an opportunity of examining this has evidently been the case.

He says however "there are also other starting points for the disease, there may be a central or more rarely a peripheral Caries".
in the spongy epiphysis of a hollow bone or in one of the spongy bones of the wrist and ankle & this may perforate from within outwardly through the cartilage and thus excite the Synovial disease. Again there may be along with the fungous proliferation of the Synovial membrane an independent proliferation under the Cartilage in the boundary between it and the bone which subsequently unites with that from above so that the Cartilage lies partly movable between the two layers "vide Billroth, Surgical Pathology.

Cornil and Ranvier insist upon the Cartilage as being the seat of the initial lesion, "the diversity of opinions and the hesitation of pathologists show that they have not recognised the lesions of the "first period" (when clinically there is only slight impediment to motion & pain of feeble intensity) & therefore cannot understand the role of these lesions in the production of the accidents of the "Second Period." In the first period they consist in a destructive
fatty (granule graisseuse) transformation of the cartilage cells, and most frequently of the bone corpuscles of the epiphysis. In the second period the parts devitalised by the fatty transformation determine around them inflammatory affections of the bone. Synovial Membrane Translated from the Article on "Les Mécus Blanche" in the latest edition of Cornil & Ranvier's Pathologie.

Rindfleisch believes the bone to be the seat of the initial lesion and calls the disease "Caries fungosa." He says "On account of its intimate connection with joints, a connection which becomes more and more marked as the disease progresses, fungating Caries is viewed by most Surgeons as a chronic articular inflammation --- --- a fungating osteitis invades say, the lower epiphysis of the Femur and the upper epiphysis of the Tibia simultaneously. The hyperaemic medulla swells --- the growth of the medulla takes the direction of the articular surface from
the first -- the exudant granulations of the medulla protrude from them.
Soon form a continuous layer intercalated between the cartilage and the bone.
He then goes on to describe the permeation of the cartilage by processes from the granulation tissue, and its gradual destruction.

Further on he says: "While the cartilage is being permeated in this way from below, a precisely similar disorder has been extending in the opposite direction from above. It was not without intention that I laid stress on the implication of other periarticular elements besides the bones in the inflammatory process. Among them we have, first the synovial membrane, then the sub-synovial connective tissue, the ligaments, and finally all the connective tissue which is in direct continuity with that already mentioned as far as the skin."

Then is described the processes of granulation tissue passing from this
Surface into the cartilage making those from the bony surface. Coalescing of the cartilage being destroyed nothing but a layer of granulations separating the ends of the bones. He says "the matter may end here", again alluding to further disorganisation. He says "everything depends on the time, manner and locality of the suppulsive complication."

Vide Rundfleisch's Pathological Histology Col IV. Sydenham Society 1873.

It is obviously very difficult to obtain examples of this disease in all its stages, but the cases I have examined have been in every way typical cases. Some were in very advanced stages when they came under observation, others at early stages when I had every opportunity of watching the progress of the affection, until the time came when from intensity of disease or failure of health, operative interference, excision or amputation was had recourse to. I made microscopic investigation of these...
Cases in the early and in the advanced stages. The condition of things I demonstrated for myself agree in the main with Billroth's description of the Pathology regarding the Synovial membrane as the seat of the initial lesion. Looking to the clinical phenomena this is strongly suggested for in a majority of cases we have the disease pretty far advanced as shown by the swelling of the articulation and the impairment in locomotion while as yet there is no pain to speak of.

Now if dense tissue like Cartilage & bone were involved in the inflammatory process pain would at once arise. I think then until pain is added to the symptoms the inflammatory mischief is confined to the Synovial membrane & lax periarticular tissues.

I also agree with Billroth Concerning the structural changes in the Synovial membrane, the spreading of the new tissue over the Cartilage & the involvement of the ligaments & periarticular tissues; but Concerning the changes in the Cartilage my preparations have shown me
Phenomena not described by him. At an early stage the cell proliferation resulting in the thickening of the cartilage & caused by the irritative processes going on in the neighbourhood. Subsequently the changes in that part of the cartilage next the covering of gelatinous tissue, where the matrix becomes fibrillated and the cells structurally changed. Furthermore throughout the other parts of the cartilage the liberation of the newly formed cells from the primary capsule & their irregular disposition & then the fatty degeneration of the protoplasm & disappearance of the nucleus. All these facts seem to me clearly enough demonstrable in my preparations.

The invasion of the cartilage by the processes of granulation tissue, so well seen in preparations No. is described by Bilroth, and also in a modified manner by Rindfleisch, but is ignored by Cornil and Ranvier.

I will now state what Cornil and Ranvier
Concerning the changes to be noted in the synovial membrane and cartilage. These observations concerning the early stages are taken from two cases examined in what they call the "first period".

"During the "first period" in one of the two cases examined a concrete mucous exudation, greyish and gelatinous had formed on the surface of the synovial membrane and cartilage".

On the "second period", the synovial membrane becomes vascular and thickened, its adipose tissue disappears to give place to an embryonic tissue which buds out and produces pus which passes into the cavity of the articulation. When the disease is more advanced one sees coming from the extremities of the bones after the destruction of the cartilage, granulations which blend with those of the synovial membrane.

The cartilages (in the "first period") preserved their smoothness of surface; they present a slight opacity and have lost a little of their elasticity. On microscopic exam-
in the second period alluding to the increased thickness of the cartilage they say "the superficial layers of the cartilage having become inert by the destruction of its cells it results from this that the capsules of the deeper layers filled with secondary capsules and disposed in long narrow lines cannot open at the surface of the articulation and there empty their contents.

While this proliferation is taking place the matrix becomes transparent and segmented parallel to the axis of these lines this process determines the increased thickening of the cartilaginous investment which in places may become 7 millimeters thick.

When treating of the manner of destruction of the cartilages they say when the cartilaginous investment has undergone fatty degeneration of its cells in it while thickness it compacts itself like an inves
body. It softens in such a manner that under the influence of the movements of the articulation which still persist it detaches itself in shreds of varying size, which, retained by an end or completely free float in the articular cavity.

When the cartilaginous investment has not been affected in its whole extent, the deep layers which are generally respected present irritative lesions on their element. From these result partial thickenings, ulcerations with velvety surface, new formations of fibrous tissue.

If (the cartilage) disappears completely at a given time it is replaced by granulations or by embryonic fibrous tissue.

Translated from Article on "Tumeur blanche."

Here again there are points of agreement with what I have found, viz: in the thickening of the cartilage of in the fatty degeneration of its cells, which to some extent helps to destroy the cartilage.

But on the strength of the examination
of two cases in their "first period" they assert that the fatty degeneration of cartilage cells is the initial lesion: they could not find in the preparations I made from the specimens of the disease in its early stages. Besides, I did find this appearance subsequently when the disease was far advanced, at all events, when the newly formed gelatinous tissue had invaded the cartilage. It is then I think that the cells which have already proliferated & thickened the cartilage undergo fatty degeneration.

What appears strange to me however is the fact that they have overlooked the invasion of the cartilage by processes of this granulation tissue. This is so clearly demonstrable & obviously is the principal means whereby the destruction of cartilage goes on.

The views of Rundfleisch concerning the bone as the ordinary starting point of this disease cannot be admitted.
In none of the Cases I have examined did I find the bone sending out process of granulation tissue into the cartilage nor the layer of granulations between the bone & the cartilage.

I did not succeed in obtaining preparations showing the exact pathological condition of the bones themselves but from a naked eye examination of them I failed to detect the changes he describes as typical.

Billroth, Cornil & Ranvier acknowledge that in some cases the pathological process may start in the bone & this, of course, must be admitted.

Considering then the views already expressed as to the pathology of this disease & what my own preparations have shown me I would conclude that as a rule the starting point of this affection is in the Synovial membrane and that the first changes are inflammatory, the peculiar progress of the disease, where
During nearly the whole course the new products waves in a state between organization and degeneration being determined by the patient's state of general health. While accepting this as the ordinary rule we must in the face of the assertions of such eminent authorities acknowledge the occasional debut of this disease in the bones. I do not think there is evidence enough to incline us to adopt the view that the cartilages may also be the seat of its commencement.
Treatment

The great therapeutic agent on which we must rely in the treatment of this affection is rest.

Looking at the Pathology we see how we are encouraged to hope for a satisfactory result until very advanced stages.

According to the phase of evolution of the disease when it comes under observation so may the issue be determined; in early stages we may get complete resolution & preserve a movable articulation; in later stages we may get resolution with however degree of impairment of motion varying with the amount of fibrous adhesions left around it between the articular surfaces, sometimes complete fibrous ankylosis may result. In the latest stages we occasionally get true osseous Ankylosis.

We must depend upon absolute rest then to minimise the intensity of the inflammatory process, to prevent acute exacerbations of inflammation which
may bring about abscess formation, the most dangerous element that can be added to the train of symptoms.

Very special attention must be paid to the general health of our patients during the whole course of the illness. We must guard against the effects of close confinement, extending over a protracted period; the diet must be judiciously regulated and medicinal tonics carefully administered according to the indications presented.

When the disease has been present for some time, in order to promote absorption or favour organisation of the new products, we may have recourse to such measures as the induction of absorbent remedies or the application of pressure to the swollen articulation by means of medicated plasters — such expedients form useful adjuncts to the treatment by rest.

Pain, when added to the symptoms generally indicates (if we exclude the formation of sympathetic abscesses) the
affection of bone or cartilage.

For its relief, counter-irritation often proves useful especially the application of Corrigan's button round about the affected parts.

When the articular ends of the bones are implicated severe pain is set up owing to increasing tension, and this persists until the exacerbation of inflammation subsides. Frequently, under these circumstances, it is necessary to cut down on the end of the bone through the periosteum; this, if carried out aseptically, is a safe proceeding and is always followed by great relief not only from pain but also from the general febrile state accompanying the above condition.

The treatment of "sympathetic abscesses" is most important. They should never be allowed to open spontaneously, for the introduction of sepsis always induces rapid disorganization of the articulation.

Open them with aseptic precautions, then you may have no further mischief added to that already existing.

In cases where the knee is affected, the best method of putting the articulation at rest is by
the application of extension by weights & pulley, Sandbags being placed at the sides of the leg to steady it, but not so as to interfere with the extending power of the weights.
When our patients are sufficiently recovered to get out of bed, the splint invented by Thomas of Liverpool is very useful, as it enables them to walk about while the extension is still kept up.

In conclusion, I think if we are careful in our treatment of all slight syphilitic affections in children & if we thoroughly carry out the treatment by rest in our cases of Relapsing Degeneration during a sufficiently lengthened period, proper regard being paid to Complicating Symptoms which are ever prone to occur, we may do much to lessen the too large proportion of cases which otherwise may sooner or later necessitate excision of the joint or amputation of the limb.