Tubercular Disease of Bones & Joints; Clinical and Pathological Studies.

by

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The study of tubercular disease is one of great interest and importance both scientifically and practically. Of late, especially since the discovery by Koch of the Tubercle Bacillus, our knowledge of the pathology of the tubercular process has been considerably extended and modified. The practical result of this has been that certain local affections, whose pathology was hitherto indeterminate, have been relegated to the tubercular group; their treatment founded on more scientific principles.

Let us at the outset briefly review the various phases through which our knowledge of tubercular disease has passed from the time of Hippocrates till the present.

Hippocrates and other ancient physicians recognised that Phthisis developed more or less directly after certain surgical accidents or diseases but the nature of this relationship was beyond their knowledge. It was only after Laennec's discovery of "grey granulations" and their connection with tubercular processes that any definite ideas were formed.
Michel & Nelaton were among the first to point out that in certain forms of Chronic Osteitis these grey granulations were present & to infer a similarity in these diseases to the Tubercular lesions in the lungs.

But views so heterodox as these were not allowed to pass unchallenged, & they were strenuously opposed by such authorities as Nimeyer, Vinten & Rintfleisch, who asserted that the caseous masses described by Lacasse & Nelaton were produced by a "serpuloid" degeneration of the products of a non-specific inflammation. (What is meant by the word "serpuloid" I shall consider later.)

However, in more recent times, the view held by Nelaton received ample proof by the work of Villermine & many others, who proved definitely the identity between tubercul granulations & the granulations found in fungi. Here's the point. Moreover, they also proved the insusceptibility of the caseous products. Still later, Kerner & Paulit showed that certain chronic alterations in bone described as "Streptoid Caries", were really tubercular in nature.
important step was the discovery by Koch of the bacillus which he con-
siders pathognomonic of all tubercular affections.

Such is a short resume of the various advances in our knowledge of Tuberculosis which we have now come to regard as tubercular. Nearly, if not quite all these chronic affections of bones & joints which were formerly described as "Strumous" or "Sercfulous."

It would be well now to consider what meaning is conveyed by the words "Strumous" & "Sercfulous."

As a student I was taught that a person of "Strumous constitution" was one in whom slight injury, or even no appreciable injury, was apt to set up certain chronic diseases tending more to degeneration than to repair, that he was liable especially to pulmonary glandular & joint affections e.g. Phthisis. Caseating glands in

the neck - Glatenia's disease of joints. "Sercfulous" to my mind conveyed much the same meaning but was more frequently applied to the glandular lesions - "Strumous" being used to describe the joints. A prominent
Characteristic of Strumous diseases was their tendency to recur, to appear in other situations than those originally affected. Heredity was also pointed out as a prominent feature.

Sherris, in his work on "Scrofula and its gland affections," gives the following definition of the "Scrofulous ulcer"—"A tendency in the individual to inflammations of a peculiar type, the distinctive feature of which are—they are usually chronic, apt to be induced by very slight irritation, to persist after the irritation has been removed. The ulcereations are remarkable for their cellular character, amongst the products being giant cells; if a certain stage be reached, tubercles; for their resistance to absorption; for their non-vascularity; for their tendency to degeneration and caseation."

In another chapter, after stating that inoculation of scrophulous matter can produce general tuberculosis, he draws the following conclusions as to the relation between Scrofula and Tuberculosis:

The manifestations of Scrofula are commonly associated with the appearance of Tuberculosis.
2. The form of tuberculosis met with in scrofulous diseases is usually of an elementary & often of an immature character (This is not the case as my pretense shew).

3. Scrofula, therefore, indicates a milder form or stage of tuberculosis & all the two processes are simply separated from each other by degree. But I am inclined to go further to state that the two processes are identical & I maintain that all the so-called Strumus & scrofulous affections are really tubercular for the following reasons:

1. Their anatomical characters are exactly alike—Sections from scrofulous tissues showing the most perfectly formed tubercle foci.

2. Inoculation of scrofulous matter produces typical tubercular lesions.

3. The tubercle bacillus has been found in all forms of scrofulous disease.

I would, therefore, discard the words "Strumus" & "Scrofulous" as redundant & confusing, & substitute the term "tubercular" as more correct & definite. In defence of my opinion I will
It is impossible to distinguish clinically external localised tubercular lesions occurring in phthisical patients from similar ones which used to be termed Scrophulae, occurring in individuals healthy in appearance, without phthisical trouble, the whole clinical history being the same.

I shall now consider the onset of tubercular disease in bones and joints. There are three necessary conditions to be insisted on:

I. A predisposition to tubercular disease hereditary or acquired.
II. An exciting cause.
III. A specific virus.

In the great majority of cases, one can establish a tubercular history— not necessarily a phthisical one, but may be only what would formerly have been described as a history of Scrophula. These, or others have pointed out that "Scrophulous" parents may have phthisical children, "phthisical" parents, "scrophulous" children. Every
one must have met with parents obviously "strumous" looking but who repudiate indignantly any consumptive taint in their family. Still even then there are cases where you can assign no parental cause. But very often in these cases you find that the surroundings, the unsuitable or scanty food, the contaminated atmosphere & want of proper physical training, engender a condition of low tissue-vitality which may fairly be described as an "acquired tubercular cleanner". Even now there remain a few cases in which you can not justly infer either an acquired or an inherited predisposition, & without doubting the correctness of the information received, I can offer no explanation of their occurrence.

In the 32 cases I have collected there is an unequivocal tubercular history in 14 - over 42 percent.

The exciting cause is most frequently an injury more or less severe. As regards this I would point out that apparently very slight & trivial injuries,
appear to be sufficient to commence the mischief — injuries to which the majority of people would give notice. Another exciting cause seems to be excessive strain e.g. heavy shovelling — a long clay washing in washerwoman's loss of nerve energy in a paralyzed joint seems to have been the exciting cause in a case reported by Dr. Howes. For Rheumatism, key tweaking threatening the joint, is to be regarded as an exciting cause I cannot say, but in more than one case it has preceded the tubercular disease. In my 32 cases there is a history of injury in 16 — of overstrain during work in 2 — of preceding disease in 2. Allowance must be made for slight injuries having been overlooked. The first two conditions would probably be conceded by the majority of surgeons, but in regard to the specific virus no such unanimity exists.

That the bacilli can be demonstrated in the bones & soft part of a tubercular joint, no one can deny — but how did they get there, whence did they come, & what is their actual significance? These are questions on which great
divergence of opinion exists.

In a primary tubercular bone or joint affection, without breach of surface, the bacilli can only come from the blood. This implies their existence in the blood prior to the onset of the disease. Now I am not aware that it has been proved that tuberculous bacilli, as such, exist in the blood of those predisposed to tubercular disease, nor do I consider it at all likely, because, if such were the case, any slight injury of or injury to an organ would form a suitable nidus for their growth and development, whereas we know that such individuals may recover perfectly from an inducing attack of bronchitis, pharyngitis or enteritis, etc.

Still, for the sake of argument, we must back our granted that the tubercular bacillus does exist in the blood in some form.

How does it act?

Liegler, in his work on Pathology, says: "Clinical experience would seem to indicate that the Bacillus Tuberculosis is not our ordinary Bacterium such as may enter and affect any organism without distinction. It would rather
soon as if infection occurred only when a definite predisposition existed or when a considerable quantity of virus is introduced. This predisposition may be general or local. We may imagine the course of infection to be this— the bacilli settle in a tissue accessible from without & pass thence into deeper structures & ultimately into the blood. If, without any local settlement, they may be taken up by the circulating juices directly & carried to various parts.

Koch (quoted by Ziegler) says— "The bacilli grow slowly, & after inoculation, proceed to develop & multiply only when they reach a spot where they are not subject to much mechanical disturbance. This may explain why many persons, although exposed to infection again & again, yet remain uninfected. It is moreover conceivable that individuals, in whose tissues inflammatory changes have already occurred, are those most disposed to tubercular infection."

Kieners (vide Truax, op. cit. p20) has shown that injections of tubercular matter into a live joint will produce a white pustule of that joint.
Schüller injected tubercular matter into the breasta (without wound), then covered the prints & found that a while scurrying followed.

Thrace (19th Jan. 1887) mentions that if rabbits be allowed to breathe an atmosphere of tubercular spores & then have their prints covered, these contractions become tubercular even when the lungs are unaffected. These experiments of Schüller & Thrace are specially important & show that the bacilli can enter the blood & go to the lungs without any disease of these organs.

A consideration of all these facts explains satisfactorily the occurrence of many local tubercular lesions—a person recovers an injury to, or has a simple inflammation of, a bone or joint, which devalizes the tissues for a time being, so that the bacilli entering the blood from without or already existing therein find a suitable soil for their growth & development. But why does this apparently simple sequence not follow in every case—why, under similar conditions, does the same cause not produce the same effect?
There are undoubtedly degrees of receptibility in different individuals and these would appear to be also, in the same individual, periods either when these tissues are more susceptible or when the virus is more active. Periods when existing lesions progress more rapidly, others appear accompanied by more or less pyrexia. This condition has been noted and described by Dr. John Duncan as "Strumous Fever".

To H. Standfield, in a communication to the Lancet (Vol. 51, p. 37) referring to the fact that the disposition to contract Phthisis may vary at different periods of a person's life, remarked that it is often of a localized character and that the frequency of local tuberculous lesions of joints "would point to the supposition that all individuals do not afford an equally suitable soil for the growth of the tubercle bacilli nor does the same individual at all times.

This may be explained partially by the fact that, apart from their peculiarities, the general health of tuberculous individuals must vary.
in the same way as that of other individuals. Therefore if the general health is below par there is less resistance offered to the onset of tubercular disease as well as other forms of disease. This is also corroborated by the results of treatment in man. The general health is fostered the greater the likelihood of the cure of any chronic tubercular affection.

Although the foregoing experiments show how, under certain circumstances, the tubercle bacilli can enter the blood through healthy lungs, still individuals predisposed to tubercle do not as a rule “live in an atmosphere of tubercular spores.” Neither do they have “tubercular matter injected into their washbasin.” Therefore it would seem necessary to rule for granted, at least in some cases, the presence in the blood of tubercle bacilli with all their specific properties—a fact not yet established.

But I ask if this supposition is really necessary? Could it not be imagined that the bacilli exist in the blood in an innocuous form—
specific form that when opportunities occur they acquire their peculiar and virulent character? Such a theory seems at least very applicable to the cases under discussion. A person predisposed to tubercular disease receives an injury to a joint, which sets up a simple inflammation (a condition which in all probability proceeds all tuberculosis). If he is in good general health at the time the vitality of his tissues is sufficient to resist the development of the bacilli; the joint as a rule recovers. But, on the contrary, if from his health has been impaired by bodily or mental suffering or other cause, the result is different. Instead of a healthy inflammation resulting from the injury one has a low asthenic type which is commonly described as Phthisis or Scrofulous and which soon becomes tubercular because here is the soil in which the bacilli can grow, develop, and acquire all its virulent properties, so as to be capable of multiplication and reproducing the same decrease by
uterulation - This process being aided by the fact that the chronic inflammatory action is kept up by a state of unrest on the part of the individual until the disease is fairly established.

This theory is strengthened by the fact that certain plants are known to display a similar peculiarity viz. that they acquire properties varying with the soil in which they are planted. It also avoids the disagreeable alternative of having for granted the presence in the blood of tubercle bacilli as such.

I will now cite cases to illustrate the foregoing remarks.

I. S. H, male, 7 years a very fair pale weak looking lad. One brother died of some chest affection - mother at present suffering from Phtisis. Was admitted into the Birmingham General Hospital (attestation B. S. H.) suffering from Gouty degeneration of the ankle joint with abscess on the lower third of tibia. The disease was of 5 months duration & began after a Piti fracture of same leg.
After amputation there was found well marked tubercular changes of the ankle joint.
This is case is important for the rapid progress of the disease and for the strong hereditary predisposition.
II J.H male aged 18 - fairly well nourished but of the fair complexioned, red-haired type of "Sirena". There is a history of Phthisis on his mother's side. Treated in the Edinburgh Royal Infirmary for osteo-muscular degeneration of the left ankle joint. The disease had existed for one year & dated from a fall from a bicycle for which he was treated by a local surgeon. An attempt was made because the foot by a partial excision but unfortunately the extensive pneumonic supervinced which progressed to Phthisis - the Spatium showing tubercle bacilli. Amputation was eventually performed & he made a good recovery.

III J.W. Age 12, strong well nourished healthy looking woman with no phthisical history. Treated in the B.H.H. for osteo-muscular degeneration of left knee joint. Duration of disease 5 yrs - no history of any injury. No other tubercular affection.
IV. G.G. male age 34. Fairly well nourished but not a healthy looking man. Family history good - mother Rheumatic.

Treated in 13.9.15 for Hip joint disease - Tuberculous testis & discharge of pus from urethra. Had had pain in the hip joint for 4 yrs but testis and began 6 months prior to admission. No history of injury nor of any assignable cause.

In cases IV-V we have to deal with perfectly typical cases in every respect – a hereditary predisposition & an exciting injury ending in Tuberculous disease. The duration of the disease in these two cases - 6 months in IV & 1 yr in V - is of some interest. I am of the opinion that the more pronounced the hereditary tendency the more rapid is the progress of the disease & the less the resistance to the spread of the virus.

But compare these cases with IV-V.

In case V there is apparently no cause whatever for the onset of the disease in the knee - one can only conjecture. Althou she could remember no injury then is still room to imagine that
Then might have been some slight injury to which a woman in her position would pay no heed, and after a lapse of 6 yrs might easily forget. There is also a possibility that her parents, tho' not consumptive as she would understand the term, might have been abrumy, saturnine or tubercular - call it what you will - that she inherited this constitution, afterwards modified by healthy surroundings. The duration of this case points to a considerable amount of tissue resistance which was undoubtedly aided by careful treatment. Case iv presents the same difficulties and in this case there are two distinctly distinct lesions to deal with. I should refer again to this.

These two cases are typical of a considerable number. I seem to be beyond any aetiologic explanation.

Can the tubercular process spread by direct extension from bone? There can be no doubt that it does spread from joint to joint e. g. in the wrist to the foot. But here there is a certain amount of continuity between
The different points may mean by the synovial membrane, so that it is merely the natural progress of the disease. This is not exactly what I mean.

Kernig in his work on "Tuberculosis of bone I joints" (1884) says that he has seen a direct extension of the tubercular process to the peritoneum in cases of hip joint disease. He advances the theory that the disease travels along the lymphatics. As a proof of this, he urges the fact that in cases of disease affecting the joint, he has found tubercles in the lymphatic glands above the lesion. That is a very interesting observation but one I have had no opportunity of verifying. Still I have frequently observed the same process in glands at the root of the lung in phthisical cases.

If this were the way by which a general, as well as a local, infection might take place.

As to the infection of the peritoneum in hip cases, in the very limited number of post mortem examinations I have seen, I have never observed it.

The only case at all like it, which
I have been able to find, in one given by Macamara in his "Diseases of Bone and Joint" (1887) - a case of his joint disease in which, at the post mortem examination, there was found a cancerous mass under the pectoralis fascia - but he gives no details.

There was no other authority whose works I have read, makes any remarks on the subject.

The following interesting case came under my own observation.

I. C, male, aged 28 yrs. treated in B. G. 111 for Glauber's disease of Knee joint. Leg was amputated & patient recovered. During convalescence I discovered that he had a Pott curvature in the dorsal region of his spine. It had occasioned no trouble & he was unaware of its existence. One day he went out of the hospital for a few hours, intoxicated. The following day he looked flushed & complained of headache & pain in the back which I attributed to his previous delirium. In the evening his temperature rose & he was slightly delirious. Gradually he became comatose, with jerking of the muscles of face & limbs - then strabismus developed & finally he died.
At the post-mortem examination there was found special & basical meningial hemorrhages in character & starting from the Cervical Vertebrae.

Now I think there can be no doubt that in this case, from some cause or other, either alcoholic brain, exposure to cold, or injury, fresh inflammation was set up in the diseased Vertebrae that the inflammation spread to the spinal membrane & thence to the base of the brain. Moreover, I think there is equally little room for doubt that the tubercular nature of the inflammation was determined by the similar nature of the special lesion. The disease spreading by the lymphatics there was a focus of infective germs ready to break mischief. If mischief of a specific kind whenever an opportunity occurred.

In Gonorrhoea & Syphilis the secondary glandular effects are not of a simple character but of a nature determined by the original disease & arguing by analogy why should not a tubercular lesion determine subsequent tubercular inflammation.

It may be said that these secondary
Tubercular affections are quite accidental. They occur so rarely as to have no connection with the original disease. How many cases of hip disease develop peritonitis? How many cases of spinal caries develop meningitis? But it is not a question of frequency of occurrence, what I wish to establish is whether such infection ever does occur. It whether we ought to bear such an event in mind as a possibility. In many diseases, rare complications are recognized, e.g., how few cases of tonsillitis develop cystitis by direct extension; other examples might easily be added.

One must also bear in mind how the tubercular process is modified by treatment. How such patients are usually placed under circumstances which tend greatly to diminish the chances of any further extension of the disease. How then is hardly any possibility of any abnormal congestion or irritation to assist the situation.

The specific nature of the disease is in itself, to my mind, a strong argument in favour of the possibility of local infection. Körnig's observations, taken
in conjunction with the case I have just related, confirm me in my belief that such a sequence ought always to be kept in mind and guarded against as far as possible.

What is the relationship between these affections of bones & joints & Pulmonary Tuberculosis? Can the system generally be infected from them?

The frequency of pulmonary tuberculosis in pulmonary subjects has been noted by many observers. 

Hegel (op. cit.) quotes the following: 

"The natural death of the pernicious is by consumption." 

Richter believes that pernicious is one of the chief predisposing causes of phthisis. 

"Great care was paid of phthisis as "pernicious" of the lung."

Hamilton says that at least 9 out of 10 who die of consumption are pernicious subjects.

Riutsholz holds that tuberculosis (including phthisis) rarely ever occurs except in pernicious individuals.

On the other hand, Hensel observed that in 87 cases of pernicious bone joints aseptic red but joint affection only 6 died with enteritis in the lungs.
Villermé affirms that a considerable number of the tuberculous (including phthisical) show no visible or known signs of spondylosis.

It was observed that in 54 cases of phthisis only 7 showed any present or past manifestations of spondylosis. It adds, "I would acknowledge no relation between spondylosis and phthisis, other than that of identity & the actual panniculus of the two diseases. It would entertain no such alliance between them as that by cause-effect and I would regard or primary & secondary disease." It also maintains that phthisis is by no means a common complication of spondylosis. Either immediate or remote. Adding as an explanation the fact that "if a patient has one form or even well-marked manifestation of spondylosis, he is not likely to develop another at the same time." This fact also explains why phthisis is not more frequent in hipped cases who lie horizontal for months, in cases of angular curvature of the spine with deformed chest.
Since the actual lesion has been demonstrated in all forms of tubercular disease the question is, Can the infection spread from the lung to a joint? Conversely, from a joint to the lung, becomes all the more interesting.

As far as I can judge it cannot be proved that distant organs can, perhaps, be infected the one from the other.

But given a patient with a tubercular joint, who subsequently develops a pulmonary lesion, I think there is a much greater chance of the lung becoming tubercular than if he had not had the joint lesion. Of course there is always the answer that the lesion was pre-existing & his lung put into a condition suitable to afford a lodgement for the bacilli independent of any pre-existing disease.

But does not the tendency of the disease to affect one part after another point to some infective agency at work, or are we to regard the process merely as the result of similar causes acting at the same or different times in different parts? It must be granted that, in a tubercular joint, the bacilli—supposing them to be the active cause—
are carried with the blood stream.

There is impossible communication
with the external air. That being so,
it is not natural to suppose that a
pre-existing lesion would increase the
supply of bacilli to render further
infection more likely.

I may here quote cases in illustration
of my meaning.

The case of J H already mentioned-
tubercular disease of ankle. Subsequent
Pneumonia going on to fibrin.

J  G  W  Male  Age  34  Treated  in  the  B.E.H.
for tubercular disease of the ankle. There
was slight consolidation at the
apex of the left lung of which he
was unaware, although "he did cough
a little now and again". The foot was
amputated & prepared concurrently for
a day or so, but when the posterior flap
alveoplasty I left the greater part of the
end of the fibula exposed. Soon after this
there was a marked increase in his
pulmonary symptoms, followed later
by anemia & abdominal distension
ending in death shortly after the
operation. At the post mortem exami-
nation both lungs were found infiltr-
ted with tubercle & there was also
prove without doubt that the pre-
existence of Pulmonary tuberculosis is an important predisposing cause of joint disease. This point is actually the case. I firmly believe although it is almost impossible to prove it clinically. But it stands to reason that apart from any infective agency, the debilitating effect of Mithini would certainly predispose to the onset of tubercular disease elsewhere. Add to this the possible (in my opinion certain) chance of clerest infection from the lungs when an opportunity arises, then everyone must admit that all local injuries to tissues of joints in Mithini patients ought to be treated with the greatest care & thoroughness.

Regarding these statements that the occurrence, in the same patient, of Mithini & local "Serfulous" disease is rare, I that the presence of one well-marked Serfulous lesion is a safeguard against the occurrence of the other. I can only point out that in 32 cases of tubercular disease of bone & joints, 9 cases had lesions in the lungs, 2 in the chest, 2 in the brain, 2 in the membrane, 1 in the intestines, 1 in glands & 1 hepatic.
Two of these cases presented 3 distinct lessons each.

This brings me now to the consideration of general infection from a local lesion. The preceding arguments to a large extent hold good. Judging from a paper in the Lancet (June 14, 1887) on a case of General Peritonitis, it may have modified his views on this subject. He says "In a patient predisposed to a simple injury may include to localize the first manifestation of the trouble (Peritonitis) from which the specific affection may slowly spread, assuming the mode of progress of a very chronic pyaemia". There would therefore seem to be no doubt of the principle of "Cause to Effect" in this particular case. The interesting point in this case was the fact that tubercular disease appeared in the knee joint of a paralysed leg, which had received no injury but was probably predisposed by loss of nerve influence.

While resident in the Northern Hospital, Liverpool, I had, through the kindness of Mr Rusey, the opportunity of seeing the following case: - CC male, aged 28??
was admitted for tubercular disease of the knee caused by injury 6 months previously. Twelve months before, he had one testis removed for tubercular infiltration, but there was still a mass left leading to the stump of the cord. Two months previous to admission he developed phthisical symptoms. With a view to checking the progress of infection by removing the other, the leg was amputated and he gradually passed into a condition resembling pyaemia & chlor.

On post-mortem examination there were found extensive tubercles in the lungs but in no other organ. In this case it is clear that in previous histological processes progressed as an infective disease. In my opinion the testis was the original focus in cell protein. The cord was cleared beyond the point of secession from this source. The injured knee was infected, followed subsequently by the lungs. Hering (op cit) makes rather an extraordinary suggestion in this connection. He says that tubercular disease is part of infection that the occurrence of general infection from a local lesion.
is clear. Thus he says that several cases occurred just after operation "as if the affection was directly conveyed through the act of operation."

The case of G.W. might be regarded as an example of this, but I am more inclined to think that the affection took place previous to the operation. Unless there was disease in the tissue above the line of incisionation, which certainty was not visible to the naked eye, I do not see how infection could possibly occur. Moreover by the operation the source of infection was removed.

One would rather expect incrustation to take place in excisions and partial operations when diseased tissue is left in the hope that it may necrose or be absorbed.

Even after these considerations one must dismiss the fact that the infecting germs must exist in the blood before you can get an initial lesion. Therefore the system must be considered already inoculated. The essential question then seems to be - does the primary lesion increase the supply of bacteria, or supply them of greater virulence or germinating power?
Since writing my Thesis I have met with a very interesting case. It is the case of a child aged 5 yrs. who was operated on by a practitioner in this Island. The child pined in good health except for Tubercular disease of the right knee joint. This was se-<br>closed last week. The following day the child was very well & cheerful. At 6 o'clock p.m. the was agitated with convulsions & frequent vomiting. At 9 she died at 11.30 the same evening. The cause of death was obviously Bacal meningitis & almost certainly of Tubercular nature.<br>This case is interesting from the point of infection following operation, & I thought it worthy of mention.
The Histology of Tubercular Osteitis

In studying this subject one naturally wishes to compare it with simple osteitis to notice the points of similarity and of difference.

This I have attempted to do, limiting myself to the process as seen in the cancellated tissue, to which tubercular disease is almost entirely limited.

I shall begin with a brief description of the normal structure of cancellated bone, my description being taken mainly from Lucien "Anatomy" and Macnamara, "Diseases of Bones & Joints."

Normal cancellated bone.

A thin layer of solid bone surrounds the cancellated tissue at the ends of the long bones. It also forms a shell round the small bones of the tarsus and carpus. From this, bony processes or trabeculae extend inwards forming the framework which supports the medulla.

This latter consists of a reticulum of adipose tissue, the spaces in which are, for the most part, filled with fat cells. Besides these there is a varying number of other cell elements — small round cells resembling white
blood corpuscles.

2. Larger nucleated cells which give of

fusion & thus form smaller cells (blood

corpuscles?) - hence called "blood cells.

3. Larger multinucleated cells of varying

shapes - osteoclasts or myelocytes.

4. Fine masses of protoplasm.

Amongst these cells are found arteries,

veins & capillaries - the two first having

periarterial lymphatic sheaths.

The trabeculae, which have the usual

structure of bone, are lined by a layer

of osteoblasts, especially in young bone.

These osteoblasts play an important

part in the developmental & patholo-

gical changes in bones.

Such is a brief description of the ste-

ment entering into the structure of

the cancellated tissue of bone.

II

I shall now describe the changes

seen in Simple Rarifying Osteitis.

By this I mean that form of inflam-

mation by which a bone is opened out

varied or modified changed accord-

ing to the duration & severity of the disea-

se. Such a process may be seen at the

cut end of a bone after amputation,

at the point of separation of a gan-
guinnes longer or less, be added conditions. I believe also that it is E.

Envenom pathologically to the "Caries" of every writer except Corvol & Ramier, who distinguish a variety which they call "True Caries."

In obturit as in the inflammations

Then an. Vera Stage.

1 Congestion of the vessels
2 Effusion of lymph & leukocytes, followed by the formation of granulation tissue.
3 Absorption - Organisation or degeneration of the inflammatory products.

The first stage shows nothing microscopically but dilatation of the blood vessels in the medullary & sub-periosteal biexes.

If the inflammation continues then follows an exudation of lymph & leukocytes around these dilated vessels. These leukocytes gradually find their way between the fat cells of the medulla.

(See diagram on next page)

Then they proliferate, aided by increased effusion & the proliferation of the connective tissue cells of the medulla, form a more or less compact granulatoin tissue containing numerous blood vessels. This tissue gradually con-

...
Compressed fat finally obliterates the fat cells, completely fills the medullary spaces.

In some cases these spaces are filled by a much less organized tissue—a lympho-fibrous matrix with a few fibrous t-cells, which condition may be due either to a slighter degree of inflammation or to its ashenic type, lying amongst the granulation cells, and due to their larger nucleated cells, notably descendants of the blood cells, or also large multinucleated phagocytes.

The foci framework is at first unchanged but as the process advances the trabeculae become necrotic and form new marrow in their sockets (analogous to involutional accretion). These are either an osteoblast...
or a bed of granulation tissue.

The number of osteoclasts varies greatly
in some places. They are very numerous,
but still appear to be the sole agents in
causing absorption of the bone, while
in others they are few or far between.

Either by their agency or by the com-
pression produced by the growing
granulation tissue, the bone trabeculae
are gradually split and consumed. As
seen in solid bone, this process of
absorption goes on in the star-shaped
cavities, resulting in the opening out
of these cavities, destruction of the
depth between them as that adjacent
cavities communicate. (See diagram next page)
So this way the bone is gradually
obliterated until, in advanced cases,
Then only remain little speckles of bone here and there in the midst of the granulation tissue. Generally these speckles are surrounded by osteoblasts until they completely disappear, especially in the bones of young subjects.

These osteoblasts either are destroyed or which is more probable, become merged with the cells filling the medullary space. What part does the bone corpuscle t its contained cell play?

This is a very debatable point. Bellard in his "Surgical Pathology" says, "The bone corpuscles are unchanged, no destruction starts from them. Occasionally we see them half destroyed at the edge of a piece of bone. The bone cells themselves may escape and in the
increase of the young cells (inflammatory) or probably they die.
Cornil & Rauvin say—"On the edge of the notch, the bone corpuscles, open to permit the cells they contain to escape, which cells become distinguishable from the embryonic cells first mentioned (granulation cells)." "There is never any fatty changes in the bone corpuscles."
So far the Authorities agreed with my opinion, founded on the pictures I have examined quite in accordance with these views.
But Cornil & Rauvin then describe what they term "true Caries." The distinguishing feature of this form is that its initial lesion is a fatty degeneration of the bone cells prior to any inflammatory action.
The only other mention of such a condition is made by Macnamara (1875) who says "Caries or ulceration of bone has, by some authorities been assigned to fatty degeneration of the bone corpuscles. Elements of the osseous tissue."
He does not give the names of his "Authorities" but states a case reported by Dr. Clark to the Pathological Society in 1881 - a case of "Caries" of 11 yrs duration.
in which there was extensive fatty degeneration of the bone. But there is no further description of the nature of the "case" nor proof given of the priority of the fatty degeneration. Rather do Cornil & Ravicz describe the class of case in which this change occurs.

Maenamaro seems to think that in case of this point, the disease & consequent atrophy might lead to fatty degeneration of the bone cells. I think this particular would act as for. S good brother & all up inflammation. He completely upsets his theory by giving, as examples, cases of white swelling which are not cases of Simple Carcin but of Tubercular Carcin. In which there all are agreed fatty degeneration in a very secondary process.

Excluding, therefore, cases of "true Carcin," (which I have never seen, I don't know where to look for them,) there is no extensive fatty degeneration of the bone cells in Simple Ratabing Sticks. When it does occur, I believe with Peiper (Elements of Surgical Pathology) that it takes place after it not before.
The death of tracheoles which is really due to the destructive inflammation. The bone cells, however, in my opinion play a merely passive part in these death processes. The necroptotic degeneration becomes merged with the indistinguishable from the surrounding granulation cells. These latter, together with the osteoclast, I consider to be developed from the inflammatory leucocytes that not from the existing bone cells, as Reimannischen would have us believe.

Such being the process in simple Ranvier's osteitis, how does the disease differ from it? My opinion is that the two processes are essentially the same, modified in the latter variety by the peculiar osteitis condition which leads to the death of osteites. The destructive process goes on in exactly the same way. The starting point is a simple inflammation caused by injury to the bone, and in my sections of tuberculous bone, all the appearances already described may be seen - in bone speedy well because they are not already infiltrated with tubercle or only slightly so.
In the dilatation of the blood vessels, formation of granulation tissue, disappearance of the muscular fibers, gradual erosion of the trabeculae. This is followed by the deposit of fibroblasts. The formation of a fibrous, non-cellular tissue from the ordinary inflammatory tissue, which still further destroys the trabeculae until they get broken into minute fragments and finally disappear, unless the process is arrested.

My opinion as to the identity of the two processes is also held by Woodhead, who, in his "Practical Pathology," defines fibrous tissue as "Rafying tissue plus the deposit of albumen." Coriuil & Maurier would appear to hold...
a somewhat different view. They divide tuberculosis into two forms: 1. Confluent - in my opinion a hair-splitting division. Prior the latter results from the fusion of the follicles in the former - i.e., directing the discrete variety, mention a zone of irritation surrounding the tubercular focus. "The zone of irritation often extends to the anastomoses of the spongy tissue, in which the discrete tubercular car are found notched as in States. These phenomena are also found at points far removed, so that it is logical to suppose that formative (?) irritation precedes the formation of the granulations - which is saying that "States precede tubercles" - a fact which some is perfectly apparent from the clinical history of the cases from the appearance seen in sections of the bone.

But they proceed to describe the confluent variety by how Cataractus by the tubercles. Sufficient because, leading to Cataractus in the surrounding areas from vascular obstruction. "But these areas which Cataract from vascular obliteration differ from those which Cataract by breaking down of the tubercle,
mucous as the former retain their adipose cells whereas in the latter the adipose cells have disappeared in the Ostitis which preceded the Tubercul.

Such an appearance I have never seen in any of my sections, I would point out that the preceding Ostitis is not as limited as the foregoing statement would lead one to infer, but extends to 'areas far removed from the tubercular focus.

Pepper adopts a somewhat similar view and considers that the tunicular spaces of the bone canals are obliterated by the tubercular growth which is extravascular. "This takes place before the keratous has time to absorb the case of typical bone forming embedded in the tuberelc, do not appear notched as in Rarefying Ostitis."
Such a statement is disproved by the preceding diagram, drawn from one of my sections, which depicts an appearance to be seen in any one of the sections; but I can only wonder how any one could advance such an opinion if he had previously examined any specimens.

The part occupied by the bone cells is similar to that in cartilaginous tissue; the opinion brought out by Bossier and Rieussec, who thus mark a distinction from "true Caries".

Macnamara, on the contrary, makes the following assertion, in regard to tubercular arthritis: "If sections of the bone be examined we find that the connective tissue element, long for the greater number of the muscles has disappeared, and that their place is occupied by fat cells. The vacuoles also contain fat cells to a greater or less extent."

Again I wonder how such a statement could be advanced, because my sections are almost all taken from cases of Tubercular Arthritis, after examining them. The utter fallacy of his opinion is apparent.
In many sections, tubercle follicles are found in all stages of development, from a simple aggregation of small round cells, through the intermediate forms with epithelioid cells up to the complete granuloma formation. The younger forms are the more interesting inasmuch as some observers have endeavored to show that such collections of small round cells are not characteristic of tubercle but are to be found in any chronic inflammatory tissue. Such may be the case although I doubt it, but here we have multiple big sets of typical follicles as that their identity cannot be doubted. From the study of these sections alone one might almost be certain in diagnosing such a collection of cells as tubercular.

Finally, what about the tubercle bacillus? I have not been able to demonstrate its existence partly from imperfect methods but at least partially from the difficulty in manipulating sections of tissue. There seems, however, to be no doubt of its existence in these cases.
Macnamara says he has found it in the later stages of the disease but not in the earlier. In his book The Zucker Schober 1 Kreuzer who affirms that "the presence of the tubercle bacillus has been established in forty cases of intervertebral of bone and joints." We have determined with certainty that in all cases of surgical intervertebral it is as easy to observe the bacillus in bone, with the microscope, as in the sputa of the intervertebral.

Volkman found the bacillus in 176 out of 200 tested cases.

Koenig states that the bacilli are usually found in some undoubted cases absent.

Thomson regarding the presence of the bacillus in intervertebral disease of bone and joints, there seems to be a unanimity of opinion.
Report of Cases.

1. A.H. age 18 yrs, treated in the Edinburgh Royal Infirmary for Subarachnoid Arthritis of the left ankle joint. Patient was fair and slender, not pale, well nourished. Father died of Pneumonia. The onset of the disease was attributed to a fall from a bicycle two years previous to admission. A partial excision was done but during the progress of the case he developed acute Pneumonia which passed into Pneumothorax. Hemoptysis, purulent sputum, chest radiographs showed extensive bilateral involvement. The foot was then amputated, but afterwards progressed rapidly.

2. G.W. age 34, a delicate looking man with a family history of Tuberculosis. Admitted into the B.S.I. for Tuberculous Arthritis of the ankle joint, but he had also slight consolidation of the apex of left lung. Foot was amputated, but died 6 weeks afterwards from Tuberculous Endarteritis of Left Lung.

3. M.R. age 38, a very pale looking man, treated in the B.S.I. for Tuberculous disease of hip joint of 9 months duration. No history of injury or of Tuberculosis in his family.
4. A.S. age 42. Suffered from disease of the hip joint of 6yr duration - also from a "cold abscess" over the scar of the foot. Family history apparently 100% free from history of injury. Died pateently. At post-mortem examination the left lung was found collapsed of the pleura extensively thinned & collapsed - a condition, considered by the Honorary Physician, due to tubercle.

5. E.H. age 7yrs. Suffered from disease of ankle joint caused by a PHT fracture sustained 5 months previously. He was a weak & delicate child but with no other disease. Mother suffers from Pthriris at present time.

6. G.S. age 12. Suffered from disease of the ankle & feet due to injury 3 years before. Previously has a partial operation performed. He was a well-nourished & fairly healthy looking lad. Mother very pale & delicate but affirms that there is no consumption present in the family.

7. E.W. age 24, a fair fat, healthy looking man, with disease of knee joint of 5yr duration. No history of injury or of Pthriris.
9 G.H. age 28 years. Suffered from disease of Achilles tendon, attributed to a sprain 1 year ago which he left untreated. As far as he knew his family were healthy. He had no other disease.

9 J.C. age 16 suffered from disease of knee joint of 3½ yrs duration - attributed to a fall. Father's brother died of phthisis. Father delicate but healthy so far. Boy himself otherwise healthy.

10 R.W. age 9 yrs. Suffered from disease of Achilles of 8 months duration. History of sprain. Mother & 1 brother phthisical. He himself accused of "strumous" looking.

11 J.C. age 28. Suffered from disease of knee joint — forma of 2 yrs duration, latter indefinite. The knee was first injured by a fall but he went about for 2 or 3 months until the knee began to swell & get painful. He was then treated in a hospital for 4 months & the knee got as well that he resumed his work, with the result that his knee soon became worse than first. He was incapacitated in the B.G.H. He was a fairly healthy looking man but could tell nothing about his family. He subsequently died from intercurrent meningitis as previously related.
12 TP age 36. Suffered from disease of knee attributed to his kneecap falling into it 6 yrs ago. His father died of Malignant Peritonitis. Other relatives healthy. He himself had inflammation of lungs when 21 yrs old. He appears to fit quite well but for the past 6 yrs he has had a cough t Spit. Had Haemoptysis 3 yrs ago, also 18 months, 6 months ago - the left lung being essentially diseased. Further progress unknown.

13 J.H. Age 18. Disease of knee joint. He sprained his knee 3 yrs ago - was treated & appearing fit well. Worked about until 6 months ago when the knee began to swell & get painful. He feels healthy. His family history is good.

14 J.K. Disease of elbow caused by falling down stairs 16 months previously. Father died of consumption. Last patient has always had poor health.

15 A.L. Age 11. Disease of ankle caused by injury 5 months previously. Father previously by brother & sister died young. He himself delicate & thin.

16 G.G. Age 34. Disease of hip of dysplasia had also a tubercular lesion & a discharge of pus from urethra. Family history good. He history of injury to the joint but when first noticed it was neglected.
17 W.J. Age 14 yrs. Healthy boy in bed with disease of left knee of 2 months duration began without any definite cause. Father dead but distant known cause of death New relations healthy.

18 A.H. Age 11 yrs. Disease of knee of 2 yrs. duration. History of injury 8 yrs ago of Rheumatic fever about 2 yrs ago. One brother dead of 'a collapse'. Father and mother alive.

19 E.B. Age 14. Disease of hip of 1 yrs duration. No history of injury or of Phthisis in the family.

20 J.G. Age 29 yrs. Disease of elbow of 2 yrs duration which he attributes to 'a hard days shovelling'. He was long used to manual labour. General health good up to 1 year ago when he began to have slight cough. Now the cough is severely depressed. Sputum showing tubercle bacilli. There is a history of consumption in a cousin.

21 S.I. Age 69. Disease of breast of 14 yrs duration. Father had 'a little Asthma' but died old. No other history of lung disease. No history of injury but one of hard manual labour by Rheumatism which especially affected the hands.

22 L.P. Age 50. Located in the Liverpool Northern Hospital for Phthisis of several years duration.
He also had spinal caries. Had increased pain in the back for about 12 months but the projection only occurred about 2 months ago - he being in bed almost entirely for the past year. His family history of phthisis nor of injury to back.

23. CE aged 30 treated in the Liverpool Northern Hospital for disease of Knee & Phthisis. Suffered from 17 months ago but noticed nothing wrong until 6 months ago when he again injured it, after which it became swollen & painful. 13 months ago he had one abscess formed for tubercular ulcer & a sinus still remains. The phthisical symptoms are only of 2 months duration. Family history unknown.

24. Mr. G. Aged 32. Disease of Elbow & Phthisis. Phthisis has existed for several years. Elbow disease for only 8 months. His career since but her occupation was that of a washer woman. Her sputum always negative. Spite.

25. H. Age 12, a delicate looking child with disease of knee joint. Injured it 18 months ago, has been treated ever since & is in a fair way to recovery. Mother died of Phthisis & her sister is under very Brother's treatment at the present time for that disease. (The following 2 cases are by Meenanmore)
26 I W age 8 - Disease of hip joint. No family history of rheumatism. No cause assigned. He finally died at the first medical examination. There were pains between the Picatta and Pincus, being inflammatory to a mass of scar tissue under the pia mater.

27 G B age 10 - Disease of the bones of the right foot of 2 years duration. Father died of consumption. The patient was delicate and nervous. He after went deaf from dipsy of the ears.

28 I G age 16 - Disease of ankles due to a sprain received while running. He was very delicate and had chronic strumus nodding. He himself was fair-haired and had enlarged cervical glands.

29 R W age 12 - Disease of hip joint. He was fair-haired and delicate nodding. No history of injury.

30 R B age 6 - Disease of right knee caused by a fall. Delicate and strumus nodding but with no history of tuberculosis.


32 E W age 6 - Disease of hip joint. Originated by a fall. Father treated for rheumatism at the Barnes Jewish Hospital.