On the treatment of so-called cases of "PERIOSTITIS" in growing bone by trephining.

BY

In April (1892) I was acting as House Surgeon to the Macclesfield General Infirmary. One evening, I was called out to see a little girl L---- B----. I found her suffering from acute pain over a spot at the outer side of the head of the left tibia. The temperature was 104.2 F. No redness, but slight swelling over the painful spot. I considered the case to be one of "Periostitis", and put my knife into the swelling, making an incision of about 1 1/2 inches right down to the bone. No pus followed this, and when seen the next day there was no abatement of the symptoms. I took her into the children's ward of the Infirmary, and enlarged the opening. The following morning there was a copious discharge of pus, but the knee joint was swollen and red. The febrile symptoms had not gone down. I at once laid the knee open, and flushed and drained the joint. Two days later there was great pain and tenderness at the outer side of the lower end of the femur, on the same side. There was also slight oedematous swelling. This was incised, but no pus discovered. The next day there was
a copious discharge of matter. The knee seemed to be quite disorganised, and the child was going down hill rapidly. Amputation was decided on, and Mr. Somerville removed the limb. The child recovered perfectly, the flaps uniting by first intention.

This fearful calamity set me thinking. How had I been to blame? Our late Professor of Surgery had hammered into us what to do in these cases. "Gentlemen", he would reiterate, again and again, "It is a case of Periostitis, put your knife down to the bone". Periostitis, Periostitis, omnium est Periostitis. I had followed his instruction to the letter, yet I did no good. There was no pus until the second day, and by that time the knee was involved. Of course thinking over it, one knows that it was a case of Ostitis, but Ostitis had been described to me as a rare disease, Periostitis as a very common one.

The above will furnish us with a text for our thesis, now let us look at the actual cases.
Case 1.

L---- B---- aet. 13. female.

Seen first, at home, April 14th, complains of pain at a point, on the innerside of the left tibia about two inches below the knee. No redness, but a slight oedematous swelling. Temperature 104.2 in the axilla. Patient will hardly allow any examination of the part from the intense pain. She is of the fair strumous type. The family history is bad, two sisters having died of what appears to be tubercular disease. Considering the case to be one of Periostitis I incised the swelling, through the tissues right on to the bone. No pus followed the incision, only a slight amount of glairy fluid.

April 15th. No change in general condition. Pulse weak, thin, & rapid. Tongue dried. Patient restless & inclined to ramble. Temperature 104. A slight amount of pus oozing from the incision. Patient ordered to be removed into the Infirmary, where she was taken the same day.

April 16th.

Temperature (on admission) 103.6. Evening 104.
This morning 104.2. Pulse 140, small. Patient very restless, slight delirium. Chloroformed and incision enlarged. There is very little stripping of the Periosteum.

April 17th.

General condition shows no improvement. Knee joint shows signs of inflammation, is hot, painful, & swollen.

April 18th.

Patient chloroformed. Knee joint incised, well flushed and drained. There was only a sero-purulent fluid in the joint.

April 19th.

Joint suppurating freely, washed out & dressed. No improvement in the hectic conditions.

April 20th.

There is a point of tenderness on the outside of the left femur, with slight swelling. (Temp. 104.4 morning 105. evening.)

April 21st.

Chloroformed, & free incision made down to the bone, only a drop or two of pus escaping.

April 30th.

The patient is going from bad to worse, so amputation
Centre of Infection of Femur

Epiphyseal

Internal Semilunar Cartilage

Centre of Infection of Tibia

Recumbent Shaft of Tibia
was considered necessary. Chloroformed and Mr. Somerville F.R.C.S. the Surgeon in charge, removing the leg at the junction of the middle & lower third, of the thigh.

June 27th.

Dismissed to the Southport Sanatorium well. The flaps healed by first intention.

Examination of limb removed.

The leg is very much emaciated. On making a section into the joint, the whole cavity is found to be completely disorganised. The internal semilunar cartilage of the tibia is loose and eroded, while below it a probe can be passed through a small opening into the head of the bone below.

On section of the bones, a patch of localised softening & necrosis of the bone is found, occupying the upper end of the tibia, the upper third of the shaft being necrosed. The centre of greatest destruction is a point about half an inch below the Epiphysial cartilage. Here there is

(Note: The cartilage-junction of the Epiphysis and the Diaphysis we shall in future call the Juxta Epiphysial line.)
a small cavity about \( \frac{1}{4} \) of an inch in diameter, from which a probe can be passed to the surface of the bone, under the periosteum, and upwards into the joint, the Epiphysis being eroded.

The periosteum is thickened, and the bone exposed at the surface for about 2\( \frac{1}{2} \) inches in length.

The focus of inflammation of the end of the femur is a point about \( \frac{1}{4} \) inch above the juxta Epiphyseal line, on the outer side. The destruction of bone is not so great as in the Tibia. The Epiphysis is slightly affected, but only to a depth of about \( \frac{1}{4} \) inch. There is an opening communicating with the surface of the bone beneath the periosteum.

A portion of the femur was decalcified, and examined under the microscope. Unfortunately the destruction was so great that little could be made out as to the minute pathology. The loops of blood vessels in the cartilage were blocked up with round cells and debris. The layers of the cartilage were also invaded, the round cells pushing aside the cartilage cells, and so causing an appearance in striking contrast with that of normal bone-forming cartilage. The cartilage cells themselves
were affected, some swollen up and half broken, while others shewed signs of vacuolisation. Here and there were groups of bodies presumably micro-organisms.
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Case 2.

Edith F---. aet. 12. Admitted into the Infirmary May 15th, under care of Mr. Bland M.R.C.S. Patient is a strumous looking child with recent cicatrices in the neck from glandular suppuration.

Ten days ago she began to complain of pain above the left ankle joint. She was very hot and dry. No doctor was called in for two days. In the interval the parents had poulticed the limb. Mr. M--- saw her on the 7th of May & ordered the poultices to be continued. On the following day he made a free incision on to the bone, letting out a quantity of matter. She did not improve much, and on the 14th. pain started in the right ankle. Mr. M--- advised her removal to the Infirmary.

May 15th. On admission there is found to be an incision about 1 inch long over the left internal malleolus, which is discharging freely. A probe could be passed in an upward direction under the periosteum for about three inches, encountering bare bone the whole way. The joint does not seem to be involved.

There is great pain over the right internal malleolus, where there is a reddish oedematous swelling about 1\frac{1}{2} inches in diameter. Temperature 103. 6. Tongue parch-
ed, and coated with a brown fur. My junior house surgeon administered chloroform, and made an incision over the swelling right on to the bone. About two drams of glairy, reddish, semipurulent fluid escaped from under the periosteum. No opening could be discovered leading into the bone, so a gouge was used, and the bone opened a little above the juxta-epiphyseal line. The tissues within were found to be sodden with pus, and quite soft. A Volkman's spoon was used, and the debris cleared out. The cavity was packed with Iodoform and made as aseptic as possible.

May 16th. Patient much improved. Temp. 102.2 (last evening) & 101 this morning. Tongue moister, and patient has asked for her milk once or twice.

May 17th. Child has improved up to a certain point. The temperature goes up each evening to 99 or 100 presumably from the left tibia, which is freely suppurating. The wound in the right tibia shows signs of filling up very fast. There are one or two picules of bone to be felt which are evidently necrosed.

July 21st. The wound on the right ankle has quite healed, that on the left is still discharging freely.
A large sequestrum is forming, but is not loose yet. Mr. Bland declines to remove it at present. General condition good. There is still a slight evening rise of temperature.

(As I left the Infirmary at the end of May to start practice in the town, I have been unable to trace this case further. She was dismissed from the Infirmary, in the condition of the last note, (supplied by my successor) to return in a short time for operation. Probably she drifted off to Manchester and was there operated on.)
The three following cases I have been able to note through the kindness of the present House Surgeon Dr. Allardice M.B.C.M. Univ. Glasg.

Case 3.

Ada S---. aet. 10. Admitted March 4th.

Patient had been seen late on March 3rd., and at once ordered into the Infirmary. On admission patient complained of intense pain on the right shoulder, at a spot about 1\(\frac{1}{2}\) inches above the insertion of the deltoid. There was no redness, and only very slight swelling. The pain on pressure was extreme. Temperature on admission (12 noon) 102.4 at 4 P.M. 103.6. The child was chloroformed, and a free incision made on to the bone. On opening the periosteum a slight amount of red, glairy, fluid escaped. No opening or roughness was found on the surface, but acting on my suggestion Dr. Allardice made a free opening into the bone with a gouge, and then with a scoop cleared away all the softish, pus-sodden material within, leaving a cavity large enough to admit the point of my little finger. The wound was plugged with Iodoform & treated aseptically.
March 5th.

Temp. Last night 102.4. this morning 100. No pain. Slight discharge from the cavity.

March 14th.

Child up and running about the ward (with arm on splint). Temperature normal. The cavity is filling up quickly. There have been one or two spicules of bone which have been discharged. No separation of the periosteum can now be made out.

April 15th.

Dismissed almost well, to return as an out patient.
Case 4.

Alice S----. Aet. 8.

Patient was first seen by the House Surgeon on Jan. 13th. at her own home, suffering from all the classical symptoms of Periostitis, at the junction of the upper and middle third of the right tibia. The leg was incised, and a small quantity of pus escaped. The wound was dressed each day by the district nurse, and healed over, but when seen on Feb. 2nd. there was evidently an accumulation of pus under the cicatrix.

Feb. 3rd.

Patient was admitted into the Infirmary.

Feb. 4th.

Patient chloroformed and the cicatrix re-incised. There was very little stripping of the periosteum. About 2 drams of pus were evacuated, and one or two small 6picules of bone removed.

Feb. 10th.

Patient progressing well.

Feb. 27th.

The cavity is almost healed up, only a small sinus being left. There is slight thickening of the bone around
the wound.

March 14th.

Patient dismissed well.

Case 5.

A baby 6 weeks old was to-day (April 7-94) brought to the out-door dispensary. There is extreme tenderness and pain over the lower end of the right fibula, with a distinct amount of swelling and marked redness. Fluctuation is present. No history of trauma can be obtained. An incision was made over the swelling and carried down to the bone. The knife was then thrust straight in-to the soft bone, and a narrow wedge-shaped piece removed.

The child was after a day or two taken into the children's ward, and dismissed in three weeks time perfectly cured.
Now "doctors differ" on the subject of acute periostitis. In this country the teaching is that it does occur as a primary inflammation, and not as secondary to ostitis or osteo-myelitis. On the other hand the French and German Schools incline to the opinion that it never occurs as a primary disease but always secondary to osteo-myelitis. Holmes in his "System of Surgery" (3rd. Ed. Vol. 2.) is "Confident that cases of acute periostitis do occur, in which the whole thickness of the shaft (and sometimes the entire shaft,) perishes, without any implication of the medullary membrane." Probably Mr. Holmes means "without any," primary "implication of the medullary membrane", otherwise, as Tubby in his paper on Periostitis justly observes, it reads very like nonsense.

(Note: It will be noticed that I do not here refer to chronic Periostitis, a subject I do not propose to consider as it almost always depends on Tubercle or Secondary Syphilis and so cannot be considered apart from those subjects.)
Professor Jones of Manchester in his work on "Diseases of Bone" seems to consider, that while Inflammation sometimes does start in the periosteum, it more often starts in the medulla, and very rarely in the bone itself.

A.H.Tubby takes a very strong stand on the continental view, (probably imbibed with his foreign study) and denies the possibility of primary periostitis, refering the starting point of inflammation to the Juxta-Epiphysseal line.

A growing long bone, histologically may be considered to consist of Cartilage, Cartilage-formed bone, Periosteum-formed bone, and the endosteum, composed of marrow and a fibrous membrane which supports the blood-vessels, the whole of these tissues being covered with an outer envelope --- the periosteum.

Further, the long bones may be considered to consist of the Diaphysis and the Epiphyses. The Diaphysis receives its vessels from the periosteum and from its own special nutrient artery. The blood supply of the Epiphyses is separate from that of the Diaphysis, and the
same vessels which supply the Epiphyses also supply the synovial membrane.

The periosteum consists of two layers, an outer fibrous one, in which the vessels are supported and an inner, osteogenetic layer. It will serve no useful purpose to go into any minute account of the growth of bone, but generally, we may state that -- Cartilage is replaced by Enchondrial bone, and the Enchondrial bone is in turn replaced by the Periosteal-bone. In the embryo the amount of Periosteal-bone is almost nil. In the adult there is little or no enchondrial bone remaining, and during growth where periosteal-bone is thickest (i.e. the middle of the shaft) there we have least enchondrial bone, while at the growing ends we have little or no periosteal activity:-- in fact "the amount of periosteal bone is in an inverse ratio to that of the enchondrial bone." (Klein)

The periosteum, then, has its final work spread over 19 to 20 years. The whole amount of bone produced at any given spot having, barely, one quarter to three quarters of an inch of thickness. Compare this with the activity of the enchondrial bone, with perhaps a shaft of 12 to 18 inches to produce. Inches, in fact, to be
produced to the fractions of inches of periosteal bone.

out a doubt the centres of activity of a growing bone

the junctions of the enchondrial bone of the diaphy-

with the cartilage. Here we have the tissues in

ve change. The blood supply free and active, and

whole tissue in a state of unstable equilibrium.

it is a law without exception that where tissue

ges are most active, there we find inflammatory

ges are most frequent. Hence we develop the first

in our evidence that periostitis is less frequent

inflammation of the bone tissues.

In the past the Periosteum has had a practical

poly of the surgeon's attention, for instance Holmes

is "System" says -- "In truth Periostitis seldom

rs uncomplicated, unless in the course of Secondary

ilis," and then goes on to devote pages to its des-
tion contenting himself with a page-and-a-half des-
tion of Ostitis. Erichsen (Vol.2. 9th.ed. P.268)

considers that "acute infective osteo-myelitis is rarely

met with in this country" yet Makins in two years can
collect 41 fatal cases in one hospital.
According to Pepper (Surgical Pathology) the inflammation of the periosteum starts in layer, and not in the deeper cellular layer. In the early stage of inflammation of the periosteum, according to Holmes, (op.cit.ant.) if that structure be examined it is sometimes found to be pink and injected, "Sometimes the periosteum does not (at least it very often does not) display any distinct signs of inflammation, either in change of colour or of thickness." This is a statement very difficult to reconcile with the idea of an acute inflammation of the periosteum, yet it is one that must have come within the range of observation of all who have had to lay open the periosteum. Time after time you lay it freely open, and wonder how such slight inflammation of a fibrous structure, could have been attended with such serious results.

This fibrous nature of the periosteum is one of the reasons why stripping of the periosteum is so extensive, when pus forms between it and the bone. If it were a tissue liable to acute inflammation, we should expect to find it easily ulcerated through at the point of inflammation, especially as it is, according to Pepper.
the outer fibrous covering that is first affected.

Now, the re-formation of the bone depends on the periosteum, "repair is from any of the original periosteum that may remain." (Pepper. op. cit. ant.) If the periosteum be destroyed there is no new formation of bone. Acute inflammation of the periosteum then, if it existed as a primary state, would kill the periosteum, through the excessive inflammation far more frequently than it does. Can we for a moment imagine that an inflammation that kills the diaphysis, perhaps the epiphyses, which may destroy joints, and kill the patient by long suppur- ation or sharp Pyaemia, will yet spare the tissue it started in, or, at most, destroy an inch or two of it. Frequently we do find some destruction of the periosteum, leaving an opening into the shaft through which nature provides an escape for the necrosed shaft within. No! No! nature is too careful, and never leaves the process of repair to a tissue which is very liable to destructive inflammation.

After amputation through a long bone we have all the tissues, which we are discussing, liable to the ac- tion of the same inflammatory agents. Yet what do we
find? The bone and marrow are very liable to inflammation when the stump becomes septic. The fungous protrusion from the end of the sawn bone, is a condition well known to all of us, In it we have the bone and marrow involved, but as a rule the periosteum escapes, with but slight inflammation, and we find it forming a ring of new bone at the extremity of the stump.

But, the friends of the Periostitis theory will say, See how often trauma is associated with Periostitis, and surely of all the tissues the periosteum is the most liable to violence? But is this so? Let us look for a moment? How about the weight of the person at each step, the jars when he falls, the tension in lifting, are not all these received by the bones and communicated to the epiphyseal cartilages? Surely the cumulative action of these little blows are far greater in their effect on this delicate growing texture than a single blow? How often do we find, in children, twists of the cartilage, not indeed amounting to actual separation of the epiphysis, yet quite enough to prove to be the starting-point of inflammatory changes.
On the opposite page is a photograph of a case in which the inflammation led to the formation of an abscess in the lower end of the tibia. The photograph is taken from a specimen in the Infirmary Museum. Amputation was performed for some reason or another, which is not made clear in the notes. However it will serve our purpose in the consideration of the seat of inflammation. I suppose that it cannot be denied that abscesses in bones, are the result of inflammatory agents identical with those that produce acute osteo-myelitis, the only difference being in the amount of mischief done. The site then, of these bone abscesses should give us a very fair indication of the origin of inflammation. We know that much the most common site for such abscesses is in the ends of the long bones. The site in the one shewn is typical of all.

The inflammation may be represented by a diagram as that on the opposite page, which is taken from the photographed specimen.

Let us suppose that A is the centre of infection. Then the spread of the inflammation in any given plane may be represented by the two diverging lines AB, AC.
The length of these lines depends on the acuteness of the inflammation, they may involve the whole shaft or they may be short. From B and C our lines of inflammation may be supposed to converge to E as the inflammation dies away. But the inflammation will form a similar figure in an upward direction. This will be smaller from the increased resistance to its onspread of the Epiphysis, and may be represented by A.F.D.G.

Such then is our theoretical figure, and it must be admitted that the actual figure is very similar.

Another point depends on this. Every one must have noticed how seldom the pus "points" at a point near the juxta-epiphyseal line. Well we see at once the explanation. The point of exit will depend on the points B and C. These we have seen are variable, but are always below (or above) the juxta-epiphyseal line. Indeed the point of exit of the pus should be a guide to our estimate of the severity of the disease, so that the nearer it is to the middle of the bone, the graver should be our prognosis. Certain it is that, where the whole of the diaphysis is involved, the pus mostly forms at the centre of the shaft.
Again, how seldom do we find a sequestrum cut off flat at the Epiphysis "as a rule some portion of the shaft remains attached to one or other of the Epiphyses"! (Bryant's Surgery Vol.2) A glance at our diagram explains the reason of this, in a way that no other explanation will.

The periosteum is continuous with the synovial membrane, which may indeed be considered the periosteum of the epiphysis. How then does periostitis affect the synovial membrane. Time after time we meet with total destruction of the diaphysis with preservation of the epiphyses and the joints. In those cases in which the epiphyses are affected, it is found that the extension has been through the epiphysis itself, and not by way of the synovial membrane. Only one museum specimen of the latter state of matters could be discovered. (Tubby.)

The photograph shewn gives us a very good idea how the epiphysis is attacked. That the epiphysis should escape so often must be a matter of great importance, and is but another instance of the providence of nature in providing a separate blood supply to the synovial membrane and epiphysis, from that of the diaphysis. In the
diaphysis once the process of inflammation starts the Haversion canals are rapidly blocked and the spread of the inflammation is rapid. There is much more resistance to its spread in the epiphysis, with its more ample blood supply, but when once it is attacked its destruction is almost as rapid as that of the diaphysis.

But what of the following class of cases which are by no means uncommon? A child gets a blow on the shin. We have the usual signs of inflammation. Pain, swelling, heat, and redness, which seem to be quite superficial. A poultice is put on the part, and an incision lets out a small quantity of pus, while later we get exfoliation of a lamina of bone. Is not this like a case of periostitis? Well might it not just as much be a case of Ostitis? What structure is most liable to injury? The periosteum is crushed on to the bone, but the bone itself is also crushed, and the vessels in their hard canals, are far more liable to be blocked up than those in the fibrous envelope of the periosteum. A superficial lamina of bone would be killed, and pus would be formed under the periosteum there being in fact no inflammation of the periosteum.
Paget in his Clinical Lectures on Typhoid mentions a case, (as a complication of enteric fever) in which there was a sort of recurrent periostitis with no death of the bone. Also a case in which there was disorganisation of the fibrous layer of the periosteeum without death of the bone.

As far as experimental work goes, the French school with Lannelongue at its head has been very active. Tubby made some experiments while in Vienna. Rabbits were the subjects, and the Staphylococcus Aureus, was injected in various ways. His experiments prove conclusively that the juxta-epiphyseal line is the point where the microbes "most do love to congregate." And that even when they are planted right under the periosteum, the bone was attacked before that structure was affected.

Some observers have laid stress on the fact, that it is necessary to have some slight injury to the bone before the micro-organisms will lodge, but as we have already shewn, almost every child would present such a condition, from the continual jars on the juxta-epiphyseal cartilage.
But when all the theory of the subject is exhausted, the result of treatment must be our chief proof. Until within recent years no one ever thought of laying open the bone in cases of periostitis. But now we have changed all that. Surgeons since the day when Ollier (Traite des Résections et des operations) taught us that there was no danger in opening the medullary cavity, have treated this tissue with but scant respect, and in no branch of surgery have the results repaid them better. In all the cases I have been able to collect, when trephining has been early, the result has been most satisfactory. Page, in his very interesting lecture in the Clinical Journal (Vol.3.No.13) brings this out very well. Even while I am writing this thesis out, I notice how Mr. Edmund Owen, in a lecture on Septic Osteitis in children (Lancet. May 26th. 1894), strongly insists on the benefit of trephining.

So far we have considered the part the bone tissues, as distinguished from the periosteum, play in inflammation, but what is the function of the several tissues, which make up the bone i.e. of the bone tissue proper, and the medulla and the growing cartilage. The bone tissue itself may be dismissed from consideration
with its fibrous matrix, and earthy deposit it is not a tissue that lends itself easily to inflammation. But when we come to consider the other two factors we are at a loss. Here we have two tissues in active growth. It is not, as if the marrow were confined to the medullary cavity and large spaces, but it follows the vessels up to their minutest ramifications. Now the cause of Osteo-myelitis is found in the presence of certain microorganisms, the difficulty has always been to account for their choice of the bones as a resting place. Tubby shewed that these organisms if injected into the system still settled on the ends of the bones to proliferate. The explanation has so far not been forthcoming. All are agreed that they are carried in the blood, but there, as far as I can discover, their observations have ended. I would suggest that it is the well known function of blood elaboration, and destruction of useless blood corpuscles, by the marrow, that is the cause of the micro-organisms resting in the bone. We have an unhealthy strumous child, whose vitality has been lowered by exposure and cold, the micro-organisms are inspired and obtain admission into the blood. In health they would
be soon destroyed by the action of the white corpuscles, which would envelope them, and in time eliminate them. But when the patient is in weak health, the white corpuscles will be destroyed in the ordinary blood elaboration process of the marrow, the micro-organisms are set free, and collect, as we may suppose, in the fine loops of vessels in the growing cartilage. Here they have a tissue which is only too ready to flare up into inflammation, and we get all the elements which are necessary to osteo-myelitis. In such a way would I, then, distribute the blame to marrow, and growing cartilage.

From the intimate connection of all the tissues which form the bone it follows, that if one is affected, the others sympathise very quickly with it. There are one or two cases in which we have had acute necrosis of the diaphysis, and yet the periosteum has quite escaped. It is in such cases that sub-periosteal resection is of such benefit.

So much then for the consideration of the seat of acute inflammation. The cause has been shewn by Lannelongue and Achard to be the Staphylococcus citreus and albus, in cases of osteo-myelitis, and Tubby and
others have shewn that the same micro-organisms are present in so-called cases of diffuse Periostitis. As to the effect of trauma in these cases, in about half of the cases, we find that it was traced, but in very few was the history at all conclusive. I suppose that it would be difficult to find any child who has not fallen, and hurt itself within the last day or two.

Sex seems to play a certain part, there being about two males to one female attacked. It is curious that the five cases I have reported were all in the female sex.

Age has, as we might have expected a most decided influence. Thus from Makins (Makins & Abbott. St. Thos. Hosp. Rep. 1889-90-91 new series XIX 193-217) we get

Under the age of 2 years -- 8
Between ,, ,, ,, 2 & 5 ,,-- 2
,, ,, ,, ,, 5 & 10 ,,-- 12
,, ,, ,, ,, 10 & 15 ,,-- 12
,, ,, ,, ,, 15 & 20 ,,-- 6
Over ,, ,, ,, 20 ,,-- \( \frac{1}{41} \)

The proportion between 5 & 15 is very marked, and is exactly what we should expect from our consideration
as to the seat of the inflammation.

The case above 20 (aet. 38) is one of a small class that do exist, and are true cases of periostitis accentuating, if it were possible to do so, the fact that true periostitis, when it does occur, is an incident of adult life.

Before proceeding to the final point of treatment of this disease, we may perhaps with advantage discuss the individual cases which have come under our notice. Fortunately they are a fairly typical lot, so that we shall be able to consider most of the points in connection with this disease.

The first case that of the little girl L---- B--- might serve as an object lesson as to the effects of wrong diagnosis or rather right diagnosis which did not extend far enough. Yet it is a case which is typical of thousands more. There was no difficulty with the symptoms, they might have been extracted from a classical description of this disease. The treatment followed was such as is recommended in the best text books. The fact that there was so little pus under the periosteum, should have made us look deeper for the true seat of the inflam-
-mation. But it was another case of having eyes and not being able to see. The next incident to notice was the involvement of the knee joint, by extension through the Epiphysis, and then the fresh start in the end of the femur. This was a fresh focus of infection, and not an extension from the joint. Even now our eyes (for now the case was under the care of one of our best surgeons, a man who has been connected with the Infirmary for 20 years) were not opened and the destruction was left to take its course in the femur. At last the patient's condition became so hopeless that amputation was resolved on, which came as salvation to the patient. Nothing in Surgery is more wonderful than the way these cases heal up after amputation. I can recall, in my small experience, at least three cases, where healing after amputation has been almost by first intention. Would trephining at an early stage have saved this limb? I think so. If we had opened the head of the tibia we should have allowed free exit to the matter. The tension would have been relieved, and the vessels would have recovered themselves, the perforation of the epiphysis would certainly have been avoided and the joint saved. The infection
of the femur might have occurred in spite of everything, but at least the bone could have been at once opened, and the infected parts removed.

Case 2 was one to which I owe a great deal. In some respects it was a most fortunate one for me. When it was admitted into the Infirmary the condition of the left ankle, left but little room for doubt as to the condition starting in the right one. It was in fact a typical case of infective ostitis. The first case had left such an impression on me, that I did not hesitate to open the bone, and the splendid result only reassured me in the plan of treatment. This case illustrates the difficulty of the differential diagnosis from acute rheumatism. This is really the most important point in the diagnosis. Owen in the paper above cited gives very clear directions. The difficulties of the situation he puts extremely well. "A boy," he says "who happens to have a "rheumatic" father is playing out in the wet days of a cheerless November. He may be out of condition, and he may have had a blow or fall upon the knee some days before and have forgotten all about the hurt. One evening he comes home crying and saying that his knee
"hurts" him and is stiff. His mother takes off his wet boots and trousers, and puts him to bed, where he passes a restless night. In the morning the mother looks at the leg, and finds the part against the knee flushed, hot and tender. She knows nothing about diaphyses, epiphyses, or synovial membranes, but she has some practical acquaintance with rheumatism; and knowing nothing else she may consider herself a specialist in that department of practice. So she applies a fomentation, and she calls in a medical man, telling him that her boy has "rheumatism at the knee" and unfortunately, he accepts her diagnosis.

The above word-photograph gives the whole scene, and was exactly what happened in this case, except that the mother continued her treatment for two days, and then the fluctuant swelling below the joint, at once disclosed the nature of the case.

The chief points to be noticed in the differential diagnosis are (1) The pain, as a rule the first thing complained of, is at a point below the joint and not in the joint itself. The articular surfaces may with care be pressed together without causing pain. The fingers
on being pressed over the head of the bone cause extreme pain. (2) Movements of the joint, conducted with care are free from pain.

The fact that two or more joints are sometimes affected by osteitis adds to the difficulty of diagnosis, though sometimes, as in the present case, from the suppuration having started in the one before the symptoms begin in the other limb, it is a help to diagnosis. The diagnosis by the results of treatment by Salicylic acid are inapplicable to the present cases, as the mischief has been done and the pus shewn itself long before the drug would have time to act.

With regard to the sequestrum in the left tibia I consider that a subperiosteal resection should have been done. It was quite useless waiting for the loosening of the sequestrum, which was only wasting valuable time, and diminishing the patient's chance of recovery.

In case 3 the only question that we need discuss was as to propriety of laying open the bone. On opening the periosteum a small quantity of pus escaped. Well I did not consider that it was enough to have caused all the high febrile condition. The bone was quite smooth
and hard, indeed it was difficult to get the gouge through the outer shell, but when once through, the welling of the pus out, shewed that we had taken the right course. One other point I may notice. The incision was carried right through the fibres of the deltoid muscle, although in this case it did not make any difference, yet I do not think that I should do it again, but rather work in from the outer side of the muscle.

Case 4, that of Alice S---- presents more points of discussion. Now here is what looks like a typical case of periostitis of the upper third of the right tibia. The periosteum being early incised heals up covering in a sequestrum, which at length in its efforts to get free produces an abscess below the cicatrix. When the abscess cavity is opened only a small sequestrum is removed which certainly does not extend as far as the juxta epiphyseal line. It is just such a case as this that must give pause to the theory that they are all cases of Juxta-epiphysitis. Yet there are points to be considered in favour of it being a case of ostitis. For instance, there is very little destruction of the bone at the surface, due perhaps to the early opening of the periosteum,
while it is more extensive towards the centre.

This case, moreover, shews us of what value gouging or trephining would have been even if the disease had not proceeded from Juxta Epiphysitis, for undoubtedly the sequestrum could have been removed, at the time when the periosteum was opened.

The case of the baby, 6 weeks old, illustrates the early age at which ostitis may occur. The case might perhaps be best compared, with those cases of strumous dactylitis, so common in young children, and should lead us in these cases to make an effort, by an early incision, and freely laying open the bone, to save the bone affected.

The final point for our consideration, is the all important one of treatment.

In the past the surgeon has been too apt to wait for the formation of pus, trying to get rid of the inflammation by means of poultices, &c, with results which are a blot on the profession of surgery, viz:- Pyaemia, necrosis, with its long continued suppuration, stiff-joints, excisions, and amputations. Has the fault lain with the surgeon, or are such results inevitable? If it be
granted, as I think it must be, that the thesis that, "inflammation in the growing long bones begins, as a rule, in the bone itself, and only as an exception in the periosteum", be true, then we are to blame. The teaching of the schools has all been for simply making an incision on to the bone, dividing the periosteum freely. We have been far too chary of touching the bone. There is very little danger in opening the medullary cavity of a bone under proper precautions as to asepsis and drainage. The question is whether we should always open the bone and to this the reply must be, "Certainly." Even when on reaching the bone, this is found to be seemingly unaffected, we should open it. We may take it as a fact that if the periosteum be raised from the bone, the bone underneath is in a state of necrosis, with vessels congested and engorged. The mere fact of opening the bone relieves this congestion, and it is wonderful to mark the rapid way in which the bone to a great extent recovers itself. The rapid way in which the cavities heal up shew that little harm will be done even if, which is most unlikely, no good is.
It follows from what we have seen, that the point of selection for opening the bone should be as near the juxta epiphyseal line as possible. If the periosteum has been raised from the bone at a point a good deal below this line, I do not see that much is to be gained by making an opening higher up, but would rather open the bone where it is exposed, knowing that the very fact of the pus coming to the surface there, shews that the pus within the bone, will be found very close to the surface in that situation. When the bone is opened a long spoon will clear out most of the necrosed tissue. In children the bone does not need a trephine, although it will in young adults, the gouge being quite capable of opening the medullary cavity, and having made the opening a Volkmann's spoon is the best instrument to remove the pus-sodden marrow and any sequestra. If the interference has come too late, and a large portion of the diaphysis forms a sequestrum then sub-periosteal sequestrotomy should be performed. Free drainage must be provided and the limb placed on a suitable splint.

Then let your constitutional treatment help to build up your patient, your Iodides, Cod liver oil,
Hypophosphites or what not.

When filling up of the cavity is completed a change of scene, perchance the invigorating breezes of the hill side, or the warmer zephyrs of the sea shore, will complete the cure of the patient, restoring him to health and increasing his belief and trust in your skill as Physician and Surgeon.

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