INFECTIVE OSTEOMYELITIS IN CHILDHOOD.

BY

H. A. T. FAIRBANK, D.S.O., M.S. LOND.,
F.R.C.S. ENG.,

SERNIOR SURGEON, HOSPITAL FOR SICK CHILDREN; SENIOR
ORTHOPEDIC SURGEON, KING'S COLLEGE HOSPITAL.

By this title I mean an osteomyelitis, acute, subacute, or chronic, due to one of the pyogenic organisms, excluding those due to tubercle and syphilis. From the pathological point of view osteomyelitis may result in rarefaction, in sclerosis or necrosis. The particular result that we see depends on the virulence of the affecting organism. An acute infection is more likely to lead to some rarefaction, and particularly to necrosis; a chronic one to rarefaction or sclerosis. These cases of infection of bone used to be classified into myelitis, osteitis, and periostitis, but, speaking generally, every inflammation of bone which has lasted for more than a day or two involves all these structures, periosteum, bone and marrow, wherever it begins. The trouble usually starts in the soft bone at the end of the diaphysis as an osteitis, affecting the cancellous tissue at the end of the bone, and very soon getting into the medulla and causing a myelitis. Periostitis does occur as the essential part of an inflammatory lesion, but only exceptionally. The periosteum is only affected secondarily in the ordinary cases which we see and which we are considering to-day—i.e., the ordinary acute inflammation of bone in children. We occasionally see a lesion where the infection is very definitely limited to one side of the bone, and, therefore, looks rather as if it began as a periostitis, but the possibility of acute periostitis occurring by infection through the blood stream can be disregarded. The causative organisms are various, the commonest undoubtedly being the Staphylococcus aureus. Fraser, of Edinburgh, some time ago noted the infective organisms in 200 cases: in 61 per cent. he found Staphylococcus aureus, and 29 per cent. pneumococcus, so that these two groups represented 90 per cent. of the cases. Six per cent. grew streptococci, and 3 per cent. the Staphylococcus albus. Occasionally unexpected organisms occur as the cause of this condition. For instance, Dr. Nabarro tells me there have recently been two cases in this hospital where Bacillus paratyphosus B had caused an osteomyelitis without any antecedent or present infection of the bowel; the bone lesion seems to have been the only lesion where the organism was found. One of these cases proved fatal. Typhoid osteomyelitis has, of course, been recognised for many years, though it is not seen much nowadays since typhoid fever has become so much less common.
Mode of Infection.

The usual point of entry is through the throat and mouth—septic and carious teeth and infected tonsils and adenoids being responsible. Boils and septic sores of the skin also sometimes form a mode of entry for the organism. The infection is always blood borne; it is carried round in the circulation until it happens to lodge in a particular bone and causes an acute inflammation. Occasionally we find that a joint becomes infected through the blood stream, and that the bone is infected secondarily, but this is usually merely an erosion of the ends of the bones following the acute arthritis, and is quite distinct from the type we are discussing. We also exclude those cases due to compound fractures, so many of which were seen during the war and which rarely occur in children. In the ordinary type, with no wound at all, the infection is always blood borne. In early infancy the umbilicus is a possible mode of entry of the infection, but personally, I am of the opinion that it is more common to get a streptococcal arthritis from this source than an ordinary osteomyelitis. In some of these cases the arthritis is primary, and in some it is secondary to infection of an epiphysis. Nowadays we consider that a blood infection of a mild type is a common occurrence, but, fortunately, the organisms do not get much chance of multiplying—they get killed as soon as they get into the blood stream, while some are excreted in the urine, and so on. Repeated and mild septicemia in children is common. Only in a certain proportion of the cases do the organisms settle in some portion of the body—e.g., the end of a bone—and set up an acute inflammatory lesion.

Predisposing Causes.

Anything which lowers the resistance may be a predisposing cause of osteomyelitis. The exanthematous fevers, for example, accompanied as they often are by sore throat, and particularly scarlet fever, obviously favour the entrance of pyogenic organisms into the blood stream. With the exception of typhoid fever the bone infections complicating the ordinary acute fevers of childhood are similar to those which occur apart from such. I do not believe there is any particular form of osteomyelitis associated with measles, scarlet fever, &c., but that such infections are due to the lowered resistance of the patient, aided by an easy mode of entry for the common pyogenic organisms. I may mention, at the same time, that in my opinion this holds good for the forms of arthritis which sometimes complicate these fevers. With the exception, perhaps, of scarlet fever, in which a type of rheumatic arthritis occurs, I think these bone and joint infections are the same as those arising from other sources, and without any antecedent exanthematous fever. In cases of pneumococcal osteomyelitis a certain number, though not all, have a history of antecedent pneumonia or bronchitis. As to the particular age at which acute infections of bone occur, some people say that 6 is the commonest age; at any rate, it is under 12 years. The condition occurs much less commonly in adolescents.

I might here say that the older the child the more likely is the infection to be a severe one which the typical text-book description will suit. The younger the child the more likely is the case to run a subacute course. Older children are much more likely to get a virulent infection with severe toxemia, high temperature, and, perhaps, rigors, &c. I do not see any reason for the infection attacking children of 6 years or so in particular, except possibly that at this age they are more likely to suffer from throat troubles of various sorts, whereas a great many of the older children have had their tonsils removed. Growth is certainly not more brisk, I consider, at this age than at other times during the growing period. Surely at puberty growth is much more brisk; a boy of 16 may grow several inches in a few months, and a girl may do the same at a rather younger age. Another feature which is drawn into the etiology is trauma; this is rather a difficult question to decide upon. A history of trauma is very often absent. Blows are said to produce damage which lowers the local resistance and favours the incidence of osteomyelitis at the injured spot. If this were so we should surely expect the peristomeum, or subperiosteal bone to be first infected, which is certainly not the case. Juxta-epiphysial strain must be the lesion if trauma is playing a part, and yet in cases where clinically we know that an epiphysis is loosened, we certainly do not get an osteomyelitis; At the same time, I am not prepared to exclude trauma altogether. As regards sex, boys are much more commonly affected than girls, the difference according to some authorities being as much as 6 to 1. We must admit that trauma is more common in boys. As regards the bones affected, the order of frequency is as follows: Tibia, femur, humerus, ulna, radius. The scapula, spine, fibula, and ilium are occasionally affected, but very rarely. The tibia is certainly by far the most common site.

Pathology.

What are the pathological changes which take place in acute osteomyelitis? As we have seen, the micro-organisms may be entering the blood stream constantly from the source of infection, whatever this may be. Some of these organisms become lodged in a particular bone. The disease always begins in the metaphysis—i.e., at the end of a long bone near the epiphyseal line, and not necessarily the main epiphyseal line; we find it beginning, for instance, near the great trochanter, and even near the lesser trochanter, though in the femur it is much more common at the lower end.

This curious fact of the commencement of the infection being localised to the part of the bone mentioned above has not been explained. The terminal vessel arrangement has been suggested as an explanation, for if one of these little vessels becomes blocked by organisms, a tiny portion of
the growing bone is entirely deprived of its blood supply. This seems to me a more probable explanation than the suggestion that it is due to the great vascularity of the part, to which some writers attribute it. There is, of course, a close network of vessels in these actively growing ends of the bones, but I do not quite see why this should favour the micro-organisms. In my opinion the greater vascularity of a part would favour the tissues and not the organisms. I think some further explanation is necessary. The fact that each portion of soft tissue in the end of a bone is surrounded by a solid shell, and, therefore, cannot swell, must be a very important factor in deciding that the germ shall win the fight.

Having begun in the cancellous bone the inflammation rapidly spreads to the periosteum on the one hand and the medulla on the other. What usually happens is this: there is an exudation of fluid under the periosteum which is clear to start with, and this lifts up the periosteum from the bone; in the ordinary staphylococcal infection this fluid very soon becomes pus, so we have pus spreading up under the periosteum and pus spreading up the medulla, and pus spreading in the bone itself. The result is necrosis of a portion of the bone. On the outer side, where the periosteum is lifted up, the bone beneath is entirely deprived of an important portion of its blood-supply. In the bone itself, where the swollen tissues are surrounded by unyielding walls, the pressure becomes great at once, and the circulation very soon ceases, while in addition the organisms themselves are producing virulent toxins; all these things combine to lead to the death of a varying portion of the bone. This necrosis will extend until the tension in the tissues is relieved; in other words, until either the pus finds its way to the surface or the case is operated upon.

It is, therefore, obvious that the earlier a case is operated upon the better for the patient from every point of view. Supposing it is not operated upon, the pus will find its way through the periosteum and an abscess will form in the muscles. This relieves the tension to some extent, but only imperfectly. Nowadays, medical men are alive to the possible diagnosis at once, and realise the need for immediate intervention, though at one time it was common enough to have cases coming to hospital which had been watched for so long that the whole shaft of the tibia, for instance, had become infected and necrosed. Separation of the epiphysis in such cases used to be common, but this is not so now; it is seen nowadays most often at the upper end of the femur. Occasionally one sees both epiphyses separated, with the shaft of the affected bone entirely loose and necrosed. Occasionally the infection is of a less virulent type, and instead of acute necrosis occurring, the condition is more or less chronic from the first, and a collection of pus forms in the bone, while new bone is laid down beneath the periosteum; this is one form of Brodie’s abscess. Radiograms show the abscess cavity as a more or less translucent area with condensation and enlargement of the bone around. This condition can also occur as a late result of the acute type of osteomyelitis, when it is not uncommon to find a residual abscess at one end of the shaft months, or even years, after the acute infection has been efficiently dealt with at the other.

This reminds us that one particular infective organism can, under varying conditions, be responsible for inflammation of varying degrees of severity. The same organism can, in the same patient, produce a chronic or subacute inflammation either before or following a very acute condition. I have seen a dry arthritis of the hip-joint with a suppurative arthritis of the knee in the same leg; a non-suppurative arthritis of the hip with an acute osteomyelitis of the opposite femur. A boy (radiograms were shown) came in with a very acute osteomyelitis of the left tibia, from which he nearly died. This began two years ago; only after several operations were we able to get the leg to heal. Recently he came to hospital with a chronic osteomyelitis of the ulna. I have not the least doubt that both lesions were caused by the same organism.

Now, what happens when the bone is opened up and drained? Two processes invariably occur: first, there is a laying down of new bone to take the place of that which is dead; and, secondly, a separation of the dead bone from the living. To take the first one. Under the periosteum, which has been stripped up, but which usually retains its vitality, a thick layer of new bone is laid down, the so-called involucrum, in which cloaca occur, through which the pus is discharged. This involucrum is separated from the old bone where this is necrotic, but is fused to the adjacent bone, which has just managed to retain its vitality. The separation of the sequestrum takes place as a result of the growth of round cells and osteoclasts which eat through the bone until they have separated entirely the dead from the living.

Theoretically, this separation should occur at the expense of the living bone, just like an area of moist gangrene is freed at the expense of the living tissue. At the same time there is probably a certain power of destruction of dead bone, because there is reason to believe that small sequestra can be eaten up entirely, and bigger ones can be broken up. After the sequestrum has been removed, the cavity left gradually gets filled up with granulation tissue, scar tissue, and eventually bone. The thickened bone gradually approximates to its normal size, though it rarely becomes quite normal. Unfortunately this is not always the case, even in children, and “bone cavities” remain, as can be seen in this radiogram, which shows a hole left by the removal of a piece of dead bone. This cavity cannot by any possibility heal, because its solid walls cannot shrink. If the cavity is widely open on one aspect the tissues may be drawn into it so that the cavity is obliterated, but if the edges of the cavity are at all abrupt, or there is only a small opening in the bony wall, this cannot occur. It may, however, heal up for a week, a month, or for
many months, and then one day the latent abscess cavity flares up and the wound breaks down again. Several such cavities may be present in the shaft of a long bone, one or other flaring up at varying intervals for many years. In many such a case is never obtained except by amputation. A mild infection—e.g., by a pneumococcus or Staphylococcus albus—may give rise to a "serous osteomyelitis"—i.e., the stage of serous effusion beneath the periosteum is never passed, the trouble subsiding without the formation of pus. Some surgeons prefer to wait and see what will happen in cases of a mild type before operating, particularly where there is a history of pneumonia.

As regards joint infection secondary to osteomyelitis, this should not be common, except in certain situations. The knee-joint is not usually affected from the femur, though it can be. It is common to get a non-purulent effusion into the knee-joint, but this subsides when the osteomyelitis has been dealt with. Osteomyelitis of the upper end of the femur, on the other hand, is much more likely to lead to suppurative arthritis, for obvious anatomical reasons. The hip, elbow, and knee are the most likely joints to become infected secondarily.

With regard to acute arthritis of infants, this often begins as an acute epiphysitis. This is a very fatal type of acute arthritis, and in some of these the infection begins in an epiphysis, and the resulting abscess bursts into the joint through a small hole in the cartilage. These holes have often been seen at operation. The order of frequency of the joints affected is as follows: hip, knee, shoulder, ankle, elbow, and wrist. Patients in this group may be as young as two months; in these early cases the umbilicus is a probable source of infection.

**Symptoms.**

There is a stage of preliminary malaise, and then sudden acute pain at the end of one of the long bones; this is very severe and is sudden. The patient shows signs of toxæmia—scanty urine, very high temperature, sometimes rigors in older children, glandular enlargement, and leucocytosis. There is usually tenderness of the affected bone before there is any obvious swelling. The patient resents any approach to the limb or any movement of it. It is excessively tender, and in the younger children the case seems almost like scurvy at this stage. After a day or two there will be a little swelling and redness, most marked on one aspect of the limb and situated towards the end of a long bone rather than at the adjacent joint. All the earlier symptoms are by this time increased. There may, however, be a little easing off of the symptoms—the temperature being rather lower and the pain less intense—which may be misleading. This is explained by the fact that the pus has reached the surface of the bone and is spreading under the periosteum, and the tension is thus relieved a little. Later an abscess forms in the soft tissues, and fluctuation may be felt. Operation should be performed, however, long before this is possible.

**Differential Diagnosis.**

What conditions have we to consider in the diagnosis of this affection? With acute cellulitis of the tissues over the bone it is very difficult to lay down definite rules; if the swelling is towards the end of the bone, near to, but not of, a joint, it is suggestive of a bone lesion, while the general symptoms would in the latter case be much more severe than if the superficial tissues only were involved. Acute arthritis is not, as a rule, difficult to differentiate, except in the case of the hip-joint. It is sometimes extremely difficult to be sure, with an obvious lesion at the upper end of the femur, whether the hip-joint in addition is involved or not. The same difficulty may arise in the course of an operation when the trouble has begun in the upper end of the femur near one of the trochanters. In less acute cases osteomyelitis has to be diagnosed from fracture, but the greatest difficulty of all is met with in the younger cases, where scurvy is a possibility. In the latter condition an examination of the urine for red blood-cells, and an inspection of the gums may be of great help; a blood count should be done. With regard to new growths, radiograms furnish the most useful evidence. In chronic cases it is sometimes necessary to exclude tubercle, but in this condition there is rarely any periosteal thickening, while there is a tendency to general rarefaction of the bones of the affected limb, such as is never seen in osteomyelitis. In osteomyelitis the periosteal thickening of the bone is the most important sign. In tubercular dactylitis, however, in contra-distinction to other bones, periosteal thickening is present.

**Prognosis and Treatment.**

The prognosis in acute cases is always grave, especially when the larger bones are involved, when the lesion is near the trunk, and when the case is very acute, with marked signs of toxæmia. Laridaceous disease sometimes occurs in these cases, and I have seen a child die from this disease in two months from the onset of the osteomyelitis. Adults have been known to die from laridaceous disease within one month of the onset of acute osteomyelitis following a gun-shot wound.

**Treatment.**—In my opinion these cases should be operated upon as soon as a diagnosis has been made, and in doubtful cases to establish the diagnosis. The only except to this rule is in a chronic case (not subacute), where there is no very definite abscess and general symptoms are entirely absent. In these cases operation need not be rushed at. In the ordinary acute or subacute case operation should be performed without any delay, the medulla being invariably opened where there is any elevation of the periosteum, and to the full extent of such elevation. In addition, if pus is found in the bone, the radical operation suggested some years ago by Mr. Waugh should be done, all infected bone being removed, even to two-thirds of the circumference of the shaft. Cases treated in this way can heal up in the course
of a few weeks with no sequestrum whatsoever. At the very least a good opening must be made in the medulla. There is no sense in using a sharp spoon in the medulla—it does no good. What is necessary is a free opening for the evacuation of the pus. Bier’s treatment is useless in this, as in all other conditions. Vaccines seldom have any effect. Antistreptococcal serum is of use in streptococcal cases. Plenty of fluid must be given—by mouth, per rectum, or subcutaneously; this is absolutely essential.

Blood transfusions are occasionally necessary in desperate cases. One more point with regard to the operation. I do not consider that early total removal of the whole of a diaphysis is a sound procedure. It may be wise in chronic cases where there is a good deal of new bone formation, but not as a primary operation in an acute case, unless the shaft is totally loose, but such a condition should never be allowed to occur. In removing sequestra in the chronic stages of the disease it is best to wait until these are loose, and then to leave a cavity of a shape that favours healing—i.e., one into which the soft tissues can fall with ease.

ANTENATAL CARE
AS IT AFFECTS THE CHILD IN UTERO.*

BY
THOMAS WATTS EDEN, M.D. EDIN.,
F.R.C.P. LOND., F.R.C.S. EDIN.,
CONSULTING OBSTETRIC PHYSICIAN, CHARING CROSS HOSPITAL, LONDON.

(Concluded from p. 39.)

THE FETAL PERIOD.

Effect of Maternal Diet.—A question related to that which we have just considered, but in reality distinct from it, is whether specific deficiencies in the mother’s diet can influence the development of the foetus. It is obvious that this may be so, although general insufficiency of food may be without effect. In its clinical aspect this question has hardly been explored at all, so far as I am able to find, but there is a certain amount of experimental work which I think deserves notice. In 1921 Reynolds and Macomer published a series of observations on the manner in which the fertility of rats could be influenced by feeding them on specifically deficient diets. In his experiments he took a certain strain of albino rats which had been inbred for 34 generations, and whose fertility-rate was well determined. The rate was somewhat lower than that of rats of a heterogenetic strain.

For the purpose of his experiments the rats were fed upon four different kinds of deficiency diet; the deficient elements were not absent from the diet, but were present in quantity somewhat less than the standard quantities of the stock diet with which they are compared. The four varieties were:

1. Low in fat-soluble vitamin.
2. Low in protein.
3. Low in calcium.
4. Double deficiency—i.e., low in protein and fat-soluble vitamins, high in carbohydrates.

The test consisted of determining the number of matings which proved fertile, and resulted in litters, under each form of diet, and the results were as follows:

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In addition to diminishing the number of fertile matings it was found that by continuing the deficiency diet through several generations complete infertility could be produced. These infertile rats were apparently well-developed animals, with the exception of those bred under a double-deficiency diet, which were feeble and undersized.

The authors made a further very interesting observation—viz., that abortion with a macerated foetus occurred in eight instances out of a comparatively small number of rats on a double-deficiency diet. After an extensive search through the literature of rat-breeding, they had failed to discover an instance of this occurrence under other conditions. The authors further state that Steinbock has also reported the birth of macerated foetuses in cattle and swine fed on similar deficiency diets.

It cannot be questioned that these observations of Reynolds and Macomer are very suggestive. It is difficult to make tests of sufficient numbers of cases in clinical work, but occasional opportunities may arise of seeking the explanation of failure to conceive, or, after conception, of failure to carry the child to term, in deficiency of some important dietetic element. We have all seen those rare but puzzling cases in which a woman has on several occasions carried her child almost to term, and then for some quite obscure reason the foetus has died in utero before labour has set in. Such cases, of course, are generally syphilitic, but failing this explanation, it would be worth while to explore the possibility of a dietetic deficiency being the operative cause. Occasional instances of what used to be called “habitual abortion” may also possibly arise from some dietetic fault.

The calcium content of the diet of an expectant mother is especially deserving of consideration. Blair Bell has estimated that the mother parts with 800 g. of calcium during the nine months of pregnancy; unless this element is freely renewed in the diet, it is quite possible that the foetus may be calcium-starved with disastrous effects to its vitality. Milk and green vegetables are the principal dietetic sources of calcium, and this element can, of course, be readily supplemented medicinally.

Effects of Alcohol on the Fetus.—I do not wish to raise the vexed question as to whether
Infective Osteomyelitis in Childhood

H. A. T. Fairbank

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