THE THREE TYPES OF ACUTE HAEMATOGENOUS OSTEOMYELITIS

A Clinical and Vascular Study

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It has been known for many years that acute haematogenous osteomyelitis varies in its clinical characteristics according to the age of the patient; thus osteomyelitis of the infant, child and adult constitute three separate clinical entities with few features in common apart from the generalised, or septicaemic, phase of the disease from which they all suffer.

Fraser (1924), Pasclau (1932) and Green and Shannon (1936) were among the first to isolate the infantile type from that of the child and to describe its main characteristics during early life, including its high mortality—up to 45 per cent in the series of Green and Shannon.

Since the inception of antibiotic treatment, Greengard (1946), Thomson and Lewis (1950) and Dennison (1955) have further contributed to the study of osteomyelitis in the newborn and have insisted on the existence of two forms, the severe and the mild. It must, nevertheless, be pointed out that the so-called mild type refers only to the mortality figures of this form of the disease, for even the milder form may cause severe and lasting damage to bone and joint in the infant.

The separate nature of acute osteomyelitis in the adult has been recognised for many years, but the first full description of the condition in the adult was given only twenty-one years ago (Zadek 1938).

In a systematic study of acute haematogenous osteomyelitis since 1944 we have treated 202 patients. This experience will be used here to summarise the main clinical features of these three types of osteomyelitis. I will begin by mentioning the main clinical features of the disease in childhood, which in osteomyelitis covers the span of life between one and sixteen years inclusively. This is followed by a summary of the disease in the infant and in the adult.

SEVERITY OF ACUTE OSTEOMYELITIS

This study of the severity of acute osteomyelitis is based on experience collected since the inception of antibiotics (Table I).

It is well recognised that acute haematogenous osteomyelitis is a disease in which the majority of contributing factors vary, including the nature and pathogenesis of the causal germ. Thus, whereas the prevalent bacteria in the older age groups—the child and the adult—is the coagulase-positive staphylococcus pyogenes aureus, the streptococcus pyogenes appear responsible for most acute bone infections in infants (63 per cent in the series of Green and Shannon; 53 per cent in this series). The almost general agreement existing at present on the severity and clinical characteristics of the three age types is not accompanied by an equal consensus of opinion regarding the causes responsible for the three different types.

In the present paper I shall attempt to offer an explanation for the diversity of clinical characteristics of acute osteomyelitis in the three ages in which they are grouped. I have so far been unable to find any similar explanation in the medical literature at my disposal.

As it is not my purpose here to study the clinical aspect of the three types of acute osteomyelitis, I have presented the table on the severity of the disease as a simple reminder of the three variants, without any aim at statistical accuracy.
LOCALISATION OF PATHOGENIC BACTERIA

Since the early experiments of Lexer (1896) it has been generally accepted that the nutrient artery is the main route for bacteria causing osteomyelitis, even if other bone vessels cannot be excluded as a route for the infecting organisms. From the experiments of Koch (1911) we know that an intravenous injection of bacteria localises in the metaphyseal veins in the bone only two hours after inoculation and that a focus of infection may develop there.

Hobo (1921) showed the part played by the vascular arrangement adjacent to the metaphyseal side of the growth plate in causing the localisation of the pathogenic bacteria in children. His diagram (Fig. 1) is based on observations of the normal structure of the vessels in that region.

| TABLE I | The Severity of Acute Haematogenous Osteomyelitis at Different Ages |
|-----------------|-------------------|-------------------|-------------------|
| | Children (per cent) | Infants (under one year) (per cent) | Adults (over sixteen years) (per cent) |
| Frequency of all patients | 80 | 7 | 13 |
| General severity before and during early treatment | | | |
| Very severe | 10 | 15 | 5 |
| Severe | 25 | 20 | 10 |
| Moderately severe | 65 | 65 | 85 |
| Local damage sustained before and during early treatment | | | |
| Permanent | 5 | 23 | 20 |
| Transient | 15 | 20 | 25 |
| None | 80 | 57 | 55 |
| Disability | | | |
| Very severe | — | 15 | 5 |
| Severe | — | 15 | 15 |
| Moderate | 6 | 12 | 18 |
| None | 94 | 58 | 62 |

In his studies of experimental infection, Starr (1922) showed that the organisms responsible for the bone infection were carried by the blood stream until they reached what is referred to as "the finer capillaries of the juxta-epiphysial region of a long bone," but he attributed the infection to the lowering of an undetermined "general resistance" of the patient. Wilensky (1934) pointed out the importance of what he called the fixation points of the disease and supported the views of Hobo on the vascular responsibility in the onset of infection. Finally, Leveuf (1947) denied that the disease in the child was initially localised in the metaphysis, as suggested by Lannelongue in 1879, and favoured the hypothesis of thrombosis of the main trunk of the nutrient artery from the onset of infection, as had been suggested by Hartmann as early as 1855.

I cannot trace that proper mention has been made by any author of the fact that the vascular pattern of the long bones occurring during the first year of life, during childhood,
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FIG. 1
Diagram of the course of the blood vessels in the marrow of a young rabbit. (After Hobo)

FIG. 2
Figure 2—Vascular loops under the growth plate. Figure 3—Large venous sinusoids where the venous limb of the vascular loops under the growth plate end.
and at puberty, is responsible for each of the three types of acute osteomyelitis. It is my purpose here to suggest that it is precisely the changing vascular arrangement at each age limit which explains the diverse clinical picture of acute osteomyelitis in every one of the age groups in which it presents itself.

**ACUTE OSTEOMYELITIS IN CHILDREN**

I begin by referring to this age group for it is the one which has been best studied from both its clinical and vascular aspects, apart from being considered by all authors as the acute osteomyelitis "par excellence."

![Image](image)

**Fig. 4**

The arterial supply of the epiphysis is largely disconnected from that of the metaphysis. Human specimen of eighteen months.

The vascular studies carried out in this centre have repeatedly shown the vascular arrangement underlying the growth plate, both by photography and microradiography (Trueta 1958a and b, Morgan 1959, Trueta and Morgan). We have been able to confirm that the capillaries adjacent to the growth plate in its metaphysial side are, apart from a narrow fringe at the periphery of the plate, the last ramifications of the nutrient artery; these, after turning down in acute loops (Fig. 2), reach a system of large sinusoidal veins responsible with others for the haemopoietic activity of the bone marrow (Fig. 3). It is here that blood flow slows down and that the pathogenic bacteria, particularly the coagulase-positive staphylococcus aureus, finds its ideal medium for development.

This system of blood lakes, beginning at the end of the capillary loop, spreads through the whole of the metaphysis in a pattern exactly corresponding to that of the distribution of bone sepsis in the early stages of osteomyelitis.

The peripheral branches of the nutrient artery, erroneously labelled end-arteries, are secondarily thrombosed by spreading infection from the venous side of the loops. Eventually the nutrient artery itself is thus occluded. Bone infection does not occur initially along the periosteal and metaphysial vessels because none has a system of vascular loops proximal to venous sinusoids like those distributed at the periphery of the growth cartilage.

In a study of the changes of the vascular pattern of the human upper femoral epiphysis during growth (Trueta 1957), it was found that the vascular barrier represented by the growth cartilage is first obvious at the age of eight months and is definitely established before the eighteenth month, except for some peripheral vascular connections between epiphysis and metaphysis (Fig. 4). Thus, from the point of view of the vascular anatomy, the infant becomes a "child" at the age of one year.

The extensive involvement of the metaphysial veins in acute osteomyelitis of the child causes early oedema. Transudates expand towards the surface of the bone across the cortex where it is thinnest, over the distal part of the metaphysis, and here the periosteum is raised from the cortex, disrupting all vascular connections between them. Soon pus follows the oedema and the periosteum lays down a new layer of bone—the involucrum—at some distance from the cortex, visible after a few days on a radiograph (Fig. 5). In another work we have
found evidence of the mechanism of involucrum formation (Fig. 6). The early deprivation of blood to the inner half of the cortex by the thrombosis of the nutrient artery, followed soon after by the interruption of the blood supply of the outer half of the cortex which accompanies the lifting of the periosteum, is responsible for the large cortical sequestra that are typical of osteomyelitis in the child (Fig. 7).

On the other hand, the isolation of the epiphysis from the metaphysis caused by the epiphysial plate provides protection both for the epiphysis itself and for the joint, and explains the rarity of joint infections and epiphysitis with growth inhibition in children, even if early treatment is defective (Fig. 8).

Summary—The aim of the surgeon in treating early acute haematogenous osteomyelitis in children should be to protect the blood supply to the outer side of the cortex to prevent the formation of an involucrum which would leave the cortex separated from its periosteum and cause sequestration.

In the child the disease tends to be more dangerous to life than to limb, for it may cause severe generalised toxaemia by massive absorption of toxins from the whole of the shaft. It seldom causes permanent damage to growth. On the contrary, in over 30 per cent of our cases in children growth was stimulated by the increased vascularity of the metaphysial side of the growth plate (Fig. 9).
ACUTE OSTEOMYELITIS IN THE INFANT

Table 1 shows that the important characteristic of acute osteomyelitis in the infant is the local severity of the disease, even in many cases considered "benign" in some classifications. As stated before, a particularly severe group occurs in the newborn, infected from the umbilicus.

It is my conviction that the more outstanding clinical features of the disease at this age should be attributed to the foetal vascular arrangement that persists in some bones up to the age of one year, with local variations corresponding to the time of full development of the epiphysial bone nucleus.

Some research, published in this Journal (Trueta 1957), and others in the course of publication, may help to clarify this point. From the time in the embryo when the ossification of the central part of the shaft of the long bones has started, the perichondral vessels progress towards the two ends of the cartilaginous "anlage" in a tortuous way, turning back when they reach the still unossified cartilaginous ends of the bone. From the last stages of intra-uterine life up to the first six months, in some epiphyses, when the growth cartilage is established but not yet limited by bone on the epiphysial side, vessels from the metaphysis penetrate the end of the "anlage," perforating the pre-existing growth plate (Fig. 10). At their ends those vessels expand, forming large venous lakes resembling metaphysial sinusoids (Fig. 11). They are situated close to the surface of the epiphysis. This explains the frequency of infections of the joint and of the epiphysial side of the preliminary growth cartilage in the infant.

In experimental work in this centre (Trueta 1958) it was shown that any severe damage to the cells at the epiphysial side of the growth plate is irreparable (Fig. 12); thus, both joint damage and arrest or disorganisation of growth are the consequences of the spread of bacteria to the ends of the nutrient artery in very early life (Fig. 13).
Another characteristic of osteomyelitis in the infant is the profuse involucrum formation, sometimes monstrously large (Fig. 14). But, contrary to the severity of the epiphysial lesion, the bulging new bone along the shaft represents only a transient alteration of which no trace will remain in later life (Fig. 15).

The extreme richness of blood flow through the periosteal vessels and the fertility of the cambium layer of the periosteum are responsible both for the early exuberant reactions and for the extraordinary remodelling that occurs in succeeding years.

**ACUTE OSTEOMYELITIS IN THE ADULT**

True acute haematogenous osteomyelitis of the long bones in adults is rare. On occasions acute haematogenous osteomyelitis occurs in the adult but is usually localised to the short bones, particularly the vertebrae, following infections of the pelvis (Trueta and Wiley 1959).

The main features of the condition in adults are the rapid spread along the whole length of the bone, the frequency of joint infections, the lack of large sequestration, and instead the irregular atrophy of the cortex, and the limited involucrum formation. All this leads to large extraperiosteal abscesses and chronic discharging sinuses when there has not been proper treatment from the early stages.

In the adult, as in the other types, the typical features of the disease may be attributed to the peculiarities of the vascular arrangement following the fusion of the growth cartilages. By the progressive penetration of the growth cartilage by metaphysial vessels, its height is reduced until finally vascular connections are established between the epiphysial and metaphysial system of vessels (Fig. 16). From then on, the blood in the nutrient artery reaches the surface of the epiphysis through large anastomoses; thus bacteria penetrating the nutrient artery may
Fig. 14—Enormous involucrum in an infant for which the elasticity of the periosteum and its osteogenic power are responsible. Figure 15—The large involucrum has completely disappeared four and a half years afterwards but the permanent epiphyseal damage is interfering severely with growth.

be brought to the vascular loops under the articular cartilage (Trueta and Harrison 1953) and spread the infection into the joint.

The fibrosis of the periosteum in the adult and its adhesion to the cortex make its detachment by pus more difficult; this prevents the formation of subperiosteal abscesses and thus preserves the blood supply to the outer half of the cortex. Consequently, large sequestra are not formed (Fig. 17). Instead, the rapid and progressive cortical absorption may allow a fracture to occur if no protection is used. The tendency to chronic infection in the marrow, from phlebitis within the bone, and joint infection are the two main factors responsible for the crippling severity of the condition in the adult (Fig. 18). The lack of reparative capacity apparent after the fusion of the epiphysis makes chronic infection the most frequent sequel of acute osteomyelitis in the adult.

**DISCUSSION**

It is beyond my purpose in this paper to enlarge on therapeutic considerations, but it may not be out of place to suggest some lines of treatment which are supported as much by anatomical vascular research—the object of this paper—as by fifteen years of clinical study of acute osteomyelitis.

Specific antibiotic treatment instituted as early and as radically as possible must be the main aim of any treatment of acute haematogenous osteomyelitis in any of its three age forms. If started soon enough, it may control the infection before severe vascular damage has been caused 1) in the epiphyseal "anlage" and joint in the infant, 2) in the cortex of the shaft in...
the child, and 3) in the joint and bone marrow of the adult, these being the most commonly and severely affected regions in the three age types of bone infection.

When some delay in the antibiotic treatment occurs, frequently because the appropriate antibiotic is not available or known, the main object of the treatment must be to reduce vascular damage to the utmost. Early, effective, radical aspiration, or preferably incision and lavage of the affected joint in the infants and adults, and splitting of the periosteum, are the most conservative procedures for the preservation of what still may remain of the blood flow in the affected bone and joint. One thing must never be forgotten, and this is that no antibiotic will ever reach the foci of infection without the preservation of some local blood flow.

The vascular anatomy may also explain the predominance of the streptococcal infection in infancy and the staphylococcal in childhood. In early life bone infection has been rightly compared to a cellulitis and a germ such as the streptococcus haemolyticus, with its lytic activity, easily invades the joint and nearby epiphysis. On the other hand, the coagulase-positive staphylococcus aureus, the common aggressor of the bone in childhood, needs for its development a stagnant or moderately active circulation such as that in the venous sinusoids under the growth plate. It may well be that the rarity of streptococcal bone infections in children is not so much due to a lack of a streptococcal bacteraemia at that age as to the incapacity of this germ to localise in metaphysical sinusoids; this is, as yet, mere conjecture, but I consider it highly possible.

**CONCLUSIONS AND SUMMARY**

1. The three age types of acute haematogenous osteomyelitis are conditioned in their respective clinical features by the differing nature of their vascular bone pattern.
2. In the infant the condition causes severe and often permanent epiphysial damage and joint infection, a large involucrum but only transient damage to the shaft and metaphysis.
3. In the child the condition is responsible for extensive cortical damage with involucrum formation, but, except for some stimulation of growth, permanent damage to the growth cartilage and to joints is exceptional. Chronicity of the disease is rare if treatment has been effective.
4. In the adult acute osteomyelitis of the long bones is rare. It causes very frequent joint infection; the cortex is absorbed instead of sequestrating. The whole of the bone is invaded and frequently leaves chronic infection in the bone marrow.
5. The vascular characteristics of the bones in each age group and their relation to the onset of infection are described.
6. Some general directives for management based on these facts are suggested.
REFERENCES