

# ACUTE HAEMATOGENOUS OSTEOMYELITIS IN INFANCY AND CHILDHOOD

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Chronic osteomyelitis is recognised as a very old disease, but acute osteomyelitis appears to have been understood only in the last 50 or so years. Until the latter part of the nineteenth century, acute osteomyelitis was only rarely recognised, except as a complication of open fractures or local sepsis.

According to Wilensky (1927) the term osteomyelitis was probably coined by Nelaton in 1844. Acute osteomyelitis has always been a serious disease because of the risk to the life of the patient as well as its tendency to chronicity and recurrence. Recent improvements in surgical care and chemotherapy have made it less formidable, but as Gilmour (1962) stated "few diseases remain unchanged over the years". The pertinent articles of 50 years ago (Starr 1922; Wilensky 1934; Crossan 1938) are now largely of only historical interest as a result of antibiotic therapy. The unpleasant complications of the past occur much less frequently, but are still seen, particularly when the disease has been underestimated or not recognised.

Attempts at understanding the disease began a century ago when Rodet reported to the Academy of Sciences in Paris in 1884 his experimental production of haematogenous osteomyelitis in animals by means of the intravenous injection of staphylococci. In a classical paper on experimental osteomyelitis, Lexer (1894) injected measured doses of cultured *Staphylococcus aureus* into the veins of young animals and found that it was possible, by traumatising a given bone shortly afterwards, to cause a focus of suppuration to appear at that site.

Many of the questions which still remain unanswered about this disease were asked by Hobo (1921) who provided a satisfactory explanation for the specific localisation of bacteria in the metaphyses of long bones. His descriptions of the vasculature of growing long-bones, although not the first, have been accepted and perpetuated in most subsequent reports of the pathogenesis of acute haematogenous osteomyelitis, and form the basis for Trueta's "three types of osteomyelitis" (1959). Indeed, Trueta reproduced Hobo's original diagram in this classic paper which related the osteomyelitic syndromes of infants, children and adults to the supposed

differences in the blood supply of metaphysis and epiphysis in the three age groups.

It was not until 1979 that Ogden provided definite histological support for an initial metaphyseal focus in human neonatal osteomyelitis. The blood supply of the metaphysis and growth plate has been clarified by Howlett (1979) and recent studies in our laboratory have documented clearly the histology and changes in circulatory pattern in the natural history of acute staphylococcal haematogenous osteomyelitis in the chicken (Emslie, Ozanne and Nade 1983; Emslie and Nade 1983).

Osteomyelitis that follows direct bone injury and acute haematogenous osteomyelitis are, for practical therapeutic purposes, dissimilar lesions. The principles laid down by Winnett Orr (Connolly 1982), used with great success in the treatment of open fractures, were much less successful in acute osteomyelitis of haematogenous origin. Before the introduction of sulphonamides and antibiotics, acute haematogenous osteomyelitis had a mortality of 20 per cent and a morbidity of 50 per cent (Trueta 1968). Kennedy (1944) found that the mortality dropped from 23 per cent to 3.5 per cent with the introduction of chemotherapeutic agents. During the last 40 years, in modern communities, that figure has dropped almost to zero.

Mitchell (1938) first reported the use of sulphonamides, and penicillin was first used by Trueta in 1941. Despite these remarkable advances, Bick, in 1941, warned that "in the treatment of haematogenous osteomyelitis, chemotherapy is not a substitute for surgical judgement, operative technique or meticulous after-care". It had then become apparent (Hoyt, Davis and Van Buren 1941) that some cases, particularly those diagnosed early and caused by organisms of low virulence, could be treated without operation. This marked the beginning of the era of controversy regarding the role of surgery in this disease.

In addition to the introduction of antibiotics, which have changed the natural history of the disease, there have been improvements in the techniques by which diagnosis might be confirmed, particularly in those of applying "nuclear medicine" to bone-imaging (Kirchner and Simon 1981); some statistical information regarding the outcome of primary management (Gillespie and Mayo 1981); and microbiological monitoring of causative organisms and their sensitivity to antibiotics (Nade 1977).

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