It is almost 100 years since Dr Poncet attributed inflammatory polyarthritis to tuberculosis. The eponymous disease is remarkable in that it is rare (even where tuberculosis is common), curable and intensely controversial. It is never possible to explain with certainty what was described long ago but it is clear that the notion of a distinct form of aseptic arthritis caused by tuberculosis rests on observations made by several physicians, most notably Poncet [1, 2]. These physicians were undoubtedly thoroughly familiar with septic tuberculous arthritis. They observed, however, that some patients presenting with subacute or even chronic deforming polyarthritis—distinct from the usually monarticular or spinal septic tuberculous arthritis—also had tuberculosis elsewhere or familial exposure to the infection without clinical signs. Moreover, the wasting, debilitating features of chronic polyarthritis, sometimes associated with fever, resembled the constitutional features associated with tuberculosis. It seemed logical to suppose that this infection, so much associated with chronic disability, a relapsing course, fever and wasting may well give rise to a greater variety of joint disease than typical tuberculous arthritis. The more recent associations of RF production with tuberculosis might be seen as further supporting this case [3].

However the absence of conventional proof of an association between chronic arthritis and tuberculosis and the clearer delineation of the inflammatory arthritides, especially acute rheumatic fever and RA eventually caused that idea to be discounted [4]. Nevertheless the possibility that tuberculosis may cause a subacute or reactive oligo- or polyarthritis has remained, and irrespective of what he actually described ‘Poncet’s disease’ has come to denote a shadowy syndrome of aseptic polyarthritis or polyarthralgia associated with active tuberculosis elsewhere; unlike typical tuberculous arthritis several or many joints may be affected without apparent suppuration or caseation and cultures of joint material, in the few instances in which this has been attempted, are negative. But, like typical tuberculous arthritis, all observers agree that Poncet’s disease resolves with effective anti-tuberculous chemotherapy.

There are no substantial series of cases of Poncet’s disease in the literature and it is not described in some of the major textbooks of rheumatology, so it must be rare. In consequence experience of this condition has almost always been anecdotal with few opportunities for detailed investigation. Indeed several authors have asked the question: is Poncet’s disease a real entity at all? In reality this is a composite question which needs to be dissected carefully. At one level plainly the answer is yes. Polyarticular pain with synovitis without frank suppuration is well described even though the number of reported case histories [5–20] involved is small. Hameed and colleagues (in this issue p. 824) ask the far more poignant question: is this condition fundamentally different from tuberculous arthritis? And in so doing they broach some of the really big issues in rheumatology.

Tuberele bacilli and arthritis have been linked together in a long and complex relationship in both experimental animals and men. The capacity of mycobacteria to induce arthritis in rats following intradermal injection combined with mineral oil is well recognized [21]. Less well recognized is the development of granulomatous bone and joint lesions after immunization with the attenuated mycobacterium, *Bacillus Calmette-Guerin* (BCG) and after BCG therapy for bladder cancer. In the former, mono- or polyarthitis, often associated with osteomyelitis, reflects disseminated BCG infection [22–24]. In the latter condition, occurring in up to three per cent of individuals treated in this way [25–29], oligo- or polyarticular pain and swelling occur after intravesical instillation of BCG and symptoms may be exacerbated or allowed to recur with repeated treatments [26]. Although BCG infection may become established in the bladder and elsewhere, synovitis is non-specific and apparently sterile [30]. The resemblance to reactive arthritis is strengthened by the detection of HLA B27 in some patients [28, 29]. There is no doubt then that the introduction of mycobacteria leads to arthritis and osteitis, which in some instances appears sterile but in others is manifestly septic.

In spite of lack of enthusiasm for Poncet’s original theory, the possibility that mycobacteria may play a central role in chronic arthritis including rheumatoid disease is very much alive. T-lymphocytes within the rheumatoid joint specifically recognize mycobacterial antigens [31], including an epitope shared by both mycobacteria and human cartilage [32] and antibodies to mycobacterial proteins also recognize determinants within synovium from human RA and rat adjuvant arthritis [33, 34]. Moreover, in addition to the capacity of infections, including tuberculosis, to induce RF production similar changes in immunoglobulin glycosylation are seen in tuberculosis and RA [35]. Even antituberculous drug therapy appears to be helpful in some patients with rheumatoid disease [36]. In this respect, as in others, in the enthusiasm to discredit the doctrine of focal sepsis which occurred in the period between the world wars perhaps the role of mycobacterial infection in inflammatory arthritis was rejected too lightly. If so the restoration is well under way.

If Poncet’s disease is truly ‘reactive’ it must be sup-
posed that the joint lesion, although initiated by infection, is brought about and sustained by immune mechanisms without replicating micro-organisms being present in the joint. In theory 'reactive' and 'septic' are distinct and must involve different pathogenetic mechanisms. Recent findings in Lyme disease and in reactive arthritis call this view seriously into question. In both instances, although linked with infection, the joint lesions have appeared sterile using conventional culture techniques. However, the identification of viable *Borrelia burgdorferi* in synovium from patients with Lyme disease [37, 38] was followed by the detection of specific bacterial antigens [39-43], and in some cases DNA [44] and RNA [45, 46] in joint material from patients with reactive arthritis. In both Lyme disease and reactive arthritis associated with *Chlamydia trachomatis* infection, as in Poncet's disease, anti-biotic therapy influences the course of the arthritis [47, 48]. These findings seem to imply that these conditions are actually due to the presence of live replicating bacteria in synovium. In this context the distinction between reactive and septic lesions becomes distinctly blurred and in spite of important clinical and genetic differences between the two conditions it may be realistic to view them as occupying ends of a spectrum of bacteria-provoked joint disease. Throughout the spectrum inflammation may be driven by renewable bacterial antigens, our conventional classification of 'septic' or 'reactive' depending largely on the genetically determined response to those antigens rather than fundamentally different processes. As Hamed and colleagues point out, a search for living bacteria, even in conventionally unpromising situations, may be rewarded.

Polyarticular joint disease certainly occurs in association with active tuberculosis and resolves with treatment of the infection. To this extent therefore the entity of Poncet's disease exists. Hamed and colleagues are to be congratulated however for pointing out that at least in some cases with multiple joint involvement the joint disease, far from being 'reactive' is actually septic even though it does not conform to our preconceptions of tuberculous arthritis. It follows, therefore, that even low grade polyarthralgia or polyarthritis occurring in individuals with tuberculosis should not be assumed to be reactive simply because bacteria may be difficult to find within the joint. The experience of Lyme disease, reactive arthritis and now perhaps tuberculous arthritis is that infection in joints is harder to define than we thought. The terms used may not be appropriate and we may have to look for infectious agents in joints with new eyes. With the advent of the polymerase chain reaction, molecular hybridization techniques and monoclonal antibodies all the technology necessary for the job is available. Rheumatoid arthritis must be the ultimate target but Poncet's disease would be as good a place as any at which to start.

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**THE WAY FORWARD FOR HYDROTHERAPY**

Hydrotherapy is defined as 'a pool therapy programme specifically designed for an individual to improve neuromuscular skeletal-function conducted and supervised by appropriately qualified personnel, ideally in a purpose built hydrotherapy pool'. The use of hydrotherapy was first employed by Hippocrates (c. 450-375 B.C.) and is now commonly undertaken by physiotherapists world-wide including both the National Health Service and the private sector in the United Kingdom. Since the 1930s physiotherapists have employed neuromuscular theory and continually improved their techniques of pool exercise therapy with the 'Bad Ragaz', 'Halliwick' and 'stretching' methods. The Bad Ragaz ring method was developed at Bad Ragaz in Switzerland from the mid-1950s. It incorporates techniques of increasing progressive resistance whilst adopting the principles of proprioceptive neuromuscular facilitation [1]. The Halliwick Method was devised by James McMillan, M.B.E., in 1949 and is based on principles of hydrodynamics and body mechanics [2]. Whilst constantly refining their hydrotherapy techniques physiotherapists have done little to attempt to evaluate their work.

Green and his colleagues [3] provide good evidence to show that specific and properly graded exercises benefit patients with OA of the hip. Additional twice weekly hydrotherapy sessions for 6 weeks in one group of patients showed no additional benefit in any outcome indicator. The title of their paper is slightly misleading in suggesting home exercises are as effective as outpatient hydrotherapy for OA of the hip for the study design was not comparative. A greater number of patients would be required to establish any trend in further improvement from hydrotherapy on a patient