Toppenish reports which are evidently spherical, plasma-like occurrences, although there are definite problems in scaling the laboratory work up to the size of phenomena in the field. More important, the laboratory luminosities are definitely not plasmas, as evidenced by the lack of microwave emissions[6]. This suggests that any field observation equipment designed to capture LP or EQL should include a broadband radio receiver as well as an optical spectrum analyzer.

Another potential mechanism involving the semiconductor properties of polymetallic ore bodies has been proposed by Demin et al.[9]. Their idea involves electrical discharges from cracks which are amplified by unusual occurrences of semiconducting minerals. These minerals would then become transistor amplifiers or thyristors in the bodies, with high-frequency stress waves producing piezoelectric polarized p and n junctions. This theory entails the generation of ultrasonic waves and electron emission, in addition to luminescence, and suggests that LP might be associated with polymetallic ore bodies near the surface.

Our present study suggests that EQL and LP are multi-mechanism, related phenomena, caused by changing stresses in the crust. If it can be shown that LP are caused by very small quakes, $M \leqslant 1.0$, then at least some LP would simply be EQL for very small earthquakes. However, considerable theoretical, experimental, and observational work remains to be done to elucidate these phenomena.

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Short Communications

Articular cartilage canals – a new pathogenetic mechanism in infectious arthritis

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Summary. In experimentally-induced erysipelas polyarthritis, preexisting cartilage canals in articular cartilage play a crucial role during the very onset of the disease. This observation might have some implications for the pathogenesis of other infectious arthritides in young animals or even rheumatoid arthritis in man.
Key words. Cartilage canals; erysipelas arthritis; rheumatoid arthritis.

When investigating the etiology and pathogenesis of any type of arthritis, one cannot avoid a basic study of the morphology and physiology of the joints. Generally, cartilage is considered to be free of nerves, blood vessels and lymphatics. The fact, however, that fetal mammalian cartilage is vascularized and that vascular channels, known as cartilage canals, course through the hyaline

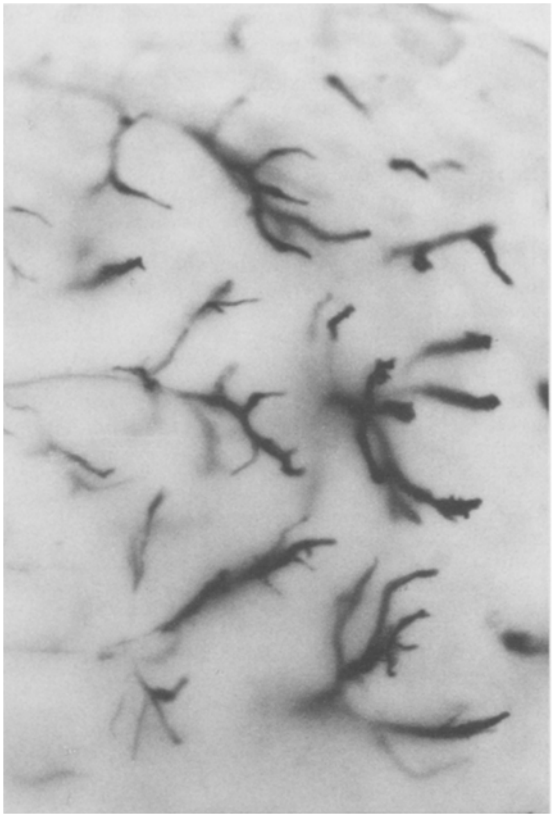

Figure 1. Demonstration of cartilage canals naturally filled with blood, in the distal femur condylus of a 6-week-old pig after treatment with cedar oil. They appear like tree branches with a blind end (× 25).

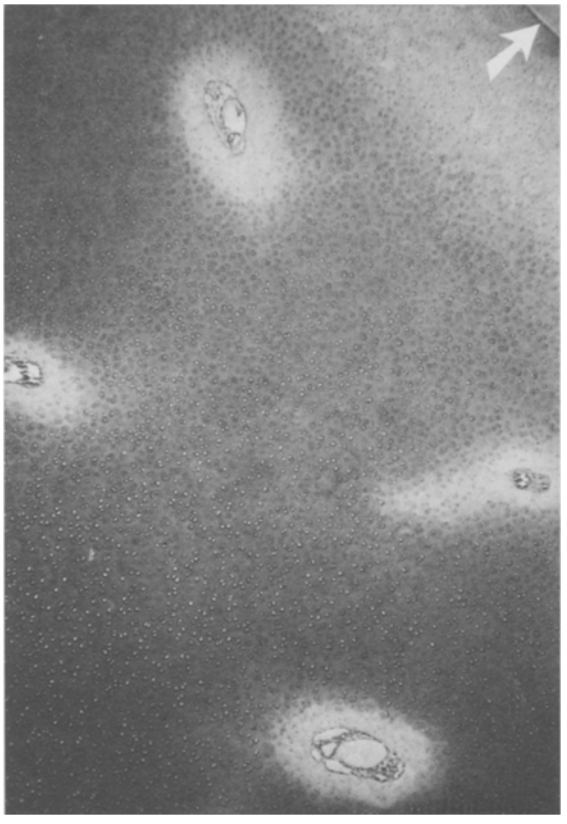

Figure 3. Four canals in the articular cartilage of the knee showing loss of metachromasia two days post infection. Arrow indicates cartilage surface (safranin 0, × 60).

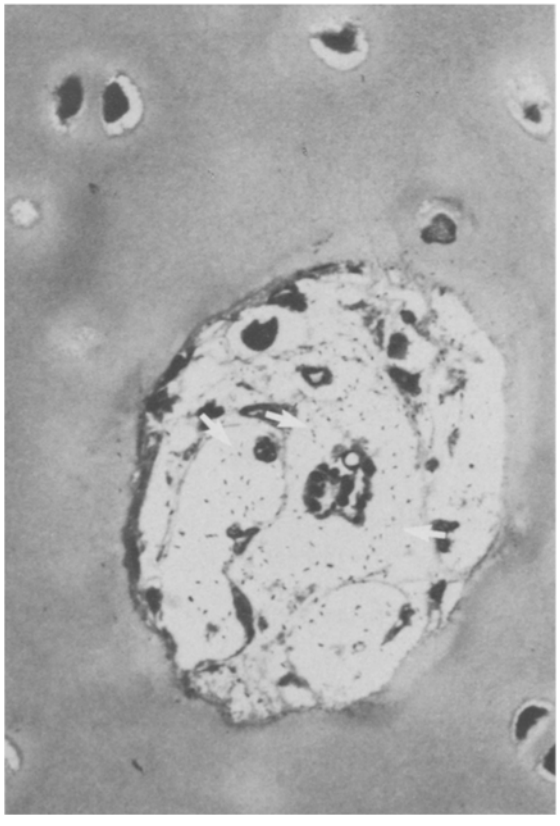

Figure 2. Cartilage canal with several vessels and loose connective tissue during bacteremia. Immunoperoxidase staining of bacteria (arrows) counterstained with hematoxylin (× 400).

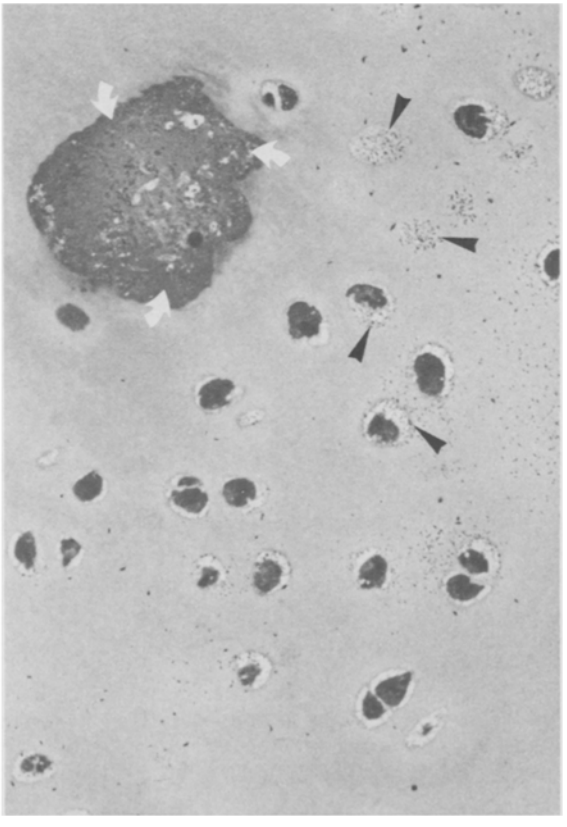

Figure 4. A completely thrombosed cartilage canal in knee articular cartilage four days after arthritis induction (white arrows). Bacteria have invaded the matrix and show tropism to chondrocytes (black arrows). Immunoperoxidase method (× 400).

cartilage of the epiphyses prior to ossification has long been known. For reasons yet to be determined, most observers have agreed that the canals do not at any time enter the region of the presumptive articular cartilage.[1,2] As a matter of fact, we and some other investigators[3–6] found abundant vascularized cartilage canals in articular cartilage of large domestic animals and man in early life, well knowing that immature joint cartilage shows no clear distinction between its permanent articular component and the underlying temporary cartilage of the epiphysis. Despite this conflict about the anatomical features, we would like to emphasize that the cartilage canals observed play a crucial role in the pathogenesis of *Erysipelothrix rhusiopathiae*-induced polyarthritis, which is a valuable animal model for rheumatoid arthritis.[7,8] The disease is characterized by chronic arthritic and deformed joints and shows rheumatoid-like histopathological features.

Materials and methods. Four pigs, aged 6 weeks, were infected intraarticularly with *Erysipelothrix rhusiopathiae*. The gnotobiotic animals were held and raised under specific-pathogen-free conditions. The pigs were killed two and four days post infection, respectively. From each individual the 12 major limb joints were removed and samples of articular cartilage were taken for histological evaluation. For the gross demonstration of cartilage canals, specimens of thick layers of articular cartilage were dried through graded alcohols and subsequently clarified by cedar oil. Due to the natural filling with blood the canals could be easily visualized (fig. 1) in the transparent articular cartilage matrix.

Results. Erysipelas polyarthritis in juvenile pigs was produced by a single intraarticular injection of viable bacteria. Two days post infection (p.i.) bacteria were found in the circulation and interstitially in almost all organs. The flooding and deposition of the microorganisms deep into articular cartilage appeared to be facilitated by means of the vascularized cartilage canals (fig. 2) which showed a marked perivascular loss of metachromasia (fig. 3). During bacteremia *E.rhus.* bacteria first stuck to the endothelium and then passed through the capillary walls into the cartilage matrix. This passage was presumably made possible by their enzymatic equipment (hyaluronidase, neuraminidase).[9,10] These initial mechanisms led to an activation of blood clotting factors and eventually resulted in a collapse of the hemostatic system with the formation of a disseminated intravascular coagulation. After four days p.i. bacteria could be demonstrated at some sites randomly distributed in chondrocytes and in the matrix of articular cartilage (fig. 4), especially in the infected knee joints.

Discussion. In previous studies on experimentally-induced erysipelas polyarthritis over a period of three years the following pathogenetic concept for chronicity has been suggested; an ineffective immune response leads to intracellular persistence of bacteria and bacterial antigens, respectively, in hypo- and avascular joint structures. No tissue damage occurs, at least for some time. The retained antigens play a continuing role in the maintenance of local immune complex- and cell-mediated hypersensitivity. Leakage of small amounts of immunologically active pathogens into the joint cavity in the course of a developing osteoarthritis provides the specific stimulus needed to elicit episodes of acute inflammation. The mechanisms of intracellular persistence of *E.rhusiopathiae* and the insufficient antigen clearance are topics for further investigation. In most of the chronically affected joints no organisms were found which could be cultured, but using immunohistological techniques *E.rhusiopathiae* antigens were detected in articular cartilage[11].

Despite intensive efforts, there is no firm evidence that rheumatoid arthritis results from microbial infections, but it remains a plausible explanation for most of the clinical and laboratory findings. On the basis of the observed pathogenetic mechanisms in our experimental animal model for rheumatoid arthritis, during the very onset of the disease, it is tempting to speculate that there is a causative agent in RA. It would show tropism for chondrocytes of articular cartilage, reaching them via the circulation by means of cartilage canals in fetal or early postnatal life; these extrinsic antigens do not induce death of chondrocytes or immune reactions. Later on, however, some yet unknown precipitating events might render the cartilage immunologically attractive, and the classical pannus in RA erodes into and around articular cartilage. This monocyte-rich granulation tissue is the only effective mechanism for achieving antigen elimination from the joint, at least as far as erysipelas arthritis is concerned. This might explain the failure to identify an infectious agent in the chronic, destructive phase of erysipelas and rheumatoid arthritis, respectively. Already in 1928 Hare[12] commented on this issue: 'I see little hope of advance from further research by post-mortem examinations of patients, bearing upon their bodies the sterile tombstones of rheumatic disease. Bacteriological investigation at the time of surgical intervention has been frequently tried, but offers little prospect of success, since the rheumatic patient does not submit himself to operation until crippled by reparative and metaplastic changes'.

In this context, it may be pertinent to offer two observations from our animal model of rheumatoid arthritis. Firstly, with the reservation that the structures undergoing repair are not infrequently the site of an acute exacerbation, there is considerable evidence for asserting that they offer no opportunity for the isolation of the rheumatic factor. Secondly, in attempting the isolation of the causative agent it should be borne in mind that RA could be the result of a multifactorial etiology and pathogenesis, but considering the animal model, it is likely that the responsible etiologic factor(s) are carried to the joint cartilage by the circulation in early postnatal life. Moreover, it is conceivable that an infectious agent transplacentally transmitted to the fetus selectively invades the cartilage from the highly vascularized cartilage canals.

There is increasing appreciation that e.g. viral infections during pregnancy may be injurious to the fetus. Though most of the effects of antenatal infections are recognizable shortly after birth, long term effects have also been demonstrated, and there is still much to be learnt about exposure to infection in utero. The observed cartilage canals in human articular cartilage in early life could potentially be of great significance in the pathogenesis and in the detection of the as yet unidentified cause of rheumatoid arthritis.

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